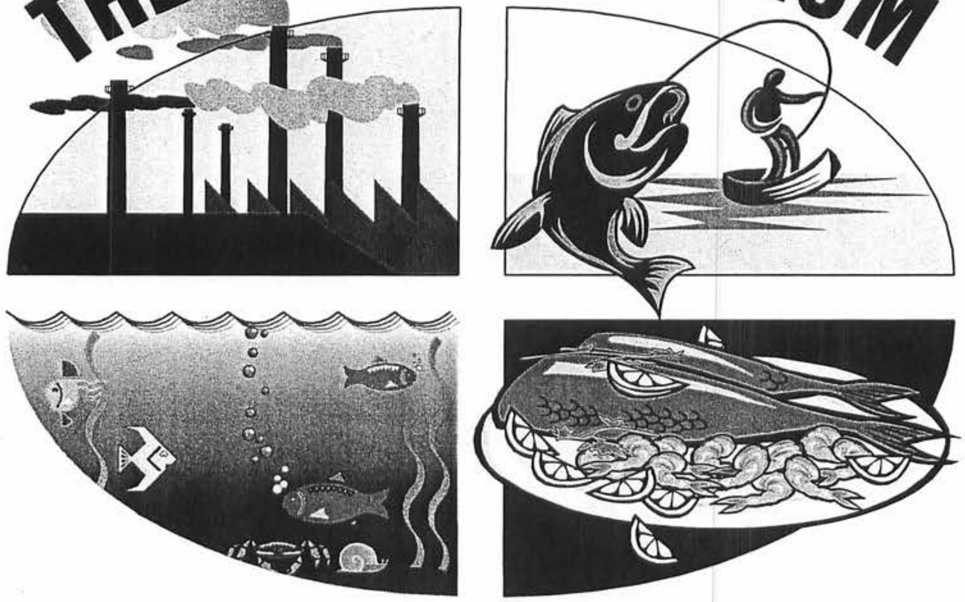
MASGC-W-02-001

THE MERCURY FORUM



Assessing Methylmercury in the Northern Gulf Environment

May 20-21, 2002
Mobile Convention Center
Mobile, AL

Sponsored By:

Mobile Bay National Estuary Program

Sea Grant Mississippi-Alabama

THE FORUM Industry Partners in Environmental Progress

MOBILE BAY WATCH MOBILE BAYKEEPER

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Welcome

LaDon Swann
Interim Director
Mississippi-Alabama Sea Grant Consortium
Auburn University, Department of Fisheries and Allied Aquacultures

On behalf of the Mobile Bay National Estuary Program; the FORUM, *Industry Partners in Environmental Progress*; Mobile Bay Watch; and the Mississippi-Alabama Sea Grant Consortium we thank you for participating in the Mercury Forum. Since January the four sponsors have devoted countless hours toward developing a balanced program with the sole objective of providing you with the best available information on the issue of mercury in the Gulf of Mexico.

What lead to this meeting? The mercury debate has been ongoing for more than 30 years. However, a series of articles on mercury appeared in the Mobile Register beginning in July 2001. Not long afterwards it was evident that our Gulf needed a venue to provide answers to the questions regarding mercury in the Northern Gulf of Mexico. This public forum is a step toward a better understanding of the effects of mercury on humans and the environment, and how we communicate any risks to our coastal stakeholders through an organized approach of open dialogue and collaborative partnerships among the public, scientists, industry, environmentalists, regulatory agencies, and risk communication specialists.

As a participant you need to know we have an excellent cross section of every group with a stake in resolving the mercury issue. We have over 200 participants from 13 states and the District of Columbia. The program in which you will participate is intentional broad and crowded because of the scope of issue areas we want to present to you. Still, there will be ample time for each of you to talk openly during the question and answer periods, over breaks, during lunch, and on the river cruise this evening.

This forum is not the first meeting sponsored to address mercury, nor will it be the last. What makes this meeting unique is the quality of speakers who have taken time out of their schedule to help us tell a story. As this story, grounded in science, unfolds think about what we need to do at the local, regional, national and international level to address the concerns people have about mercury in our Gulf.

We hope you enjoy your stay in Mobile and take time to enjoy our beautiful "Emerald Coast".

The Mercury Forum Schedule

May 20, Monday

7 a.m.-3 p.m.	**Registration**
8:00 a.m.	**Welcome** Dr. LaDon Swann Mississippi-Alabama Sea Grant Consortium Honorable Jeff Sessions United States Senator for Alabama
8:30	**Introduction and Purpose of Meeting** Mr. David Yeager, Mobile Bay National Estuary Program

Mercury in Humans
Moderator: LaDon Swann

8:45	**Health Risk Assessment** Dr. John Risher Agency for Toxic Substances and Disease Registry
9:10	**Seychelles Study** Drs. Philip Davidson and Gary Myers University of Rochester School of Medicine and Dentistry
10:10	**Break**
10:30	**Faroe Islands Studies** Drs. Richard W. Clapp and Philippe Grandjean Boston University School of Public Health
11:30	**Question and Answer Session**
Noon	**Lunch**
1:00 p.m.	**EPA Fish Consumption Advisories** Mr. Joel Hansel U.S. Environmental Protection Agency - Region 4
1:20	**Development of Methylmercury Reference Dose** Dr. Kathryn Mahaffey U.S. Environmental Protection Agency
1:40	**Development of Consistent Mercury Advisories in the Gulf of Mexico** Dr. Frederick Kopfler EPA - Gulf of Mexico Program
2:00	**Panel Question and Answer Session**

2:45 **Break**

Mercury in the Environment

Moderator: Mr. Steve Perry, THE FORUM, Industry Partners in Environmental Progress

3:00	**Historical Background of Mercury in the Environment** Mr. Charles Moore South Carolina Department of Natural Resources
3:20	**Chemistry of Mercury to Methylmercury** Dr. Gary Gill Texas A&M University - Galveston
3:40	**Atmospheric Deposition of Mercury** Dr. Jane Guentzel Coastal Carolina University
4:00	**Offshore Oil and Gas Sources** Dr. Jerry Neff Battelle – American Petroleum Institute
4:20	**Panel Question and Answer Session**
5:00	**Adjourn**

6:00 p.m. **Evening cruise and reception aboard the Cotton Blossom**

May 21, Tuesday

8:00-noon **Registration**

Current and Proposed Mercury Science and Education Projects
Moderator: Mr. David Yeager, Mobile Bay National Estuary Program

8:30 a.m. **Fish Advisories in Alabama**
Dr. Neil Sass
Alabama Department of Public Health

8:45 **Fish Monitoring Programs in Alabama**
Mr. Fred Leslie
Alabama Department of Environmental Management

9:00 **Survey of the Occurrence of Mercury in Fishery Resources of the Gulf of Mexico**
Dr. Frederick Kopfler
EPA - Gulf of Mexico Program

9:15 **Methylmercury in Marine Fish: A Gulf-Wide Initiative**
Mr. Ron Lukens
Gulf States Marine Fisheries Commission

9:30 **Gulf-Wide Fish Monitoring Program**
Dr. Spencer Garrett
National Marine Fisheries Service

9:45 **Panel Question and Answer Session**

10:15 **Break**

Selected Mercury Related Research
Moderator: Dr. Richard Wallace, Auburn University Marine Extension and Research Center
and Mississippi-Alabama Sea Grant Extension

10:45 **Current Research into Mercury Control from Coal-Fired Power Plants**
Dr. Larry Monroe
Southern Company Services, Inc.

11:05 **Distribution of Mercury in the Mobile River Basin in Relation to Land Use**
Dr. Kimberly Warner
University of Alabama, Department of Biological Sciences

11:25 **Social Impact Assessment of Mercury Contamination in Mobile River Basin**
Dr. Hobson Bryan
University of Alabama, Department of Geography

11:45 **Panel Question and Answer Session**

Noon.	**Lunch**

Economic Realities of Mercury in the Environment
Moderator: Ms. Casi Callaway, Mobile Bay Watch

1:00	**Seafood Industry Perspective** Mr. Bob Collette National Fisheries Institute
1:20	**Recreational Fishing Perspective** Dr. Bob Shipp University of South Alabama
1:40	**Environmental Perspective** Ms. Felice Stadler National Wildlife Federation
2:00	**Minamata Plus 50: Where Are We?** Dr. Leonard Levin Electric Power Research Institute
2:20	**Panel Question and Answer Session**
3:00	**Break**

Where Should "WE" Go from Here

3:30	**Needs Analysis**
5:00 p.m.	**Adjourn**

Mercury in Humans

Health Risk Assessment
Dr. John Risher
Agency for Toxic Substances and Disease Registry

The Agency for Toxic Substances and Disease Registry (ATSDR) is a component of the U.S. Public Health Service, under the Department of Health and Human Services. Among its legislative mandates, it is charged with determining levels of significant human exposure for environmental substances identified at hazardous waste sites. These chemical-specific human exposure levels, known as Minimal Risk Levels (MRLs), are derived for different routes and durations of exposure, and are based upon scientific studies of laboratory animals, controlled human clinical studies, and/or epidemiological data from human populations. The process used for deriving MRLs is essentially the same as that used by the U.S. Environmental Protection Agency (EPA) for calculating oral Reference Doses (RfDs) and inhalation Reference Concentrations (RfCs).

The first step in this process is a critical examination of the overall database for the chemical or substance under investigation. From that analysis, a single study or group of similar studies that represent(s) the single effect that is believed to be the most sensitive toxic endpoint is selected as the "critical study." Typically, a no-observed-adverse-effect-level (NOAEL), representing the highest dosage at which no observable adverse effects were seen in the critical study, or a lowest-observed-adverse-effect-level (LOAEL) then serves as a starting point for MRL or RfD/RfC derivation. After appropriate adjustment for duration of exposure and/or dosimetric airway adjustments (for RfCs), a number of uncertainty factors are applied to account for various areas of uncertainty in the MRL or RfD/RfC calculation.

Whether the traditional NOAEL/LOAEL approach or the benchmark dose approach is employed, the NOAEL, LOAEL, or benchmark dose is divided by a composite uncertainty factor to arrive at the MRL or RfC. Such health guidance values may then be employed by public health officials to make decisions deemed necessary for the protection of the public health.

John F. Risher, Ph.D.
Senior Health Advisor, Toxicology Information Branch
ATSDR's Division of Toxicology, Atlanta, GA

Education
B.A. Zoology, Miami University, Oxford, OH
M.S. Physiology, Miami University, Oxford, OH
Ph.D. Environmental Health (Toxicology), University of Cincinnati, College of Medicine,
Department of Environmental Health

Work Experience

- Twenty-three years experience in the environmental health area with the Agency for Toxic Substances and Disease Registry (ATSDR) and the U.S. Environmental Protection Agency (EPA).

- Currently assigned as the Senior Health Advisor in the Toxicology Information Branch of ATSDR's Division of Toxicology in Atlanta, GA.

- Previously served as the EPA Office of Research and Development (ORD) Superfund Technical Liaison in EPA Region IV in Atlanta and ATSDR Senior Toxicologist in EPA Region VII in Kansas City, KS.

- Served for 11 years on committees which establish levels of substances considered to represent no threat to human health [ATSDR's Minimal Risk Level (MRL) Workgroup; EPA's Reference Dose/Reference Concentration (RfD/RfC) Workgroup].

- Have authored environmental health criteria documents for EPA and the World Health Organization, toxicological profiles for ATSDR, and over 20 articles in the environmental health area in peer reviewed scientific journals.

- Hold adjunct assistant professorships at Georgia Military College (est. 1879) in Milledgeville, GA, and Shorter College in Rome, GA; instruct courses in general biology, environmental studies/quality, anatomy and physiology, and nutrition at Atlanta area extensions/distant learning centers.

Mercury in Humans

Seychelles Study
Drs. Phillip Davidson and Gary Myers
University of Rochester School of Medicine and Dentistry

The Seychelles Child Development Study: Background, Design, and Results Through 66 Months of Age

Philip W. Davidson
University of Rochester School of Medicine and Dentistry

The Seychelles Child Development Study (SCDS) is testing the hypothesis that there is an association between prenatal exposure to methylmercury (MeHg) from maternal fish consumption and child development. We are longitudinally following a large inception cohort (n=779) of mother-child pairs in the Republic of Seychelles where 85% of the population consumes marine fish daily. The mean prenatal exposure in maternal hair is about 7 ppm while other toxic exposures (e.g., lead, alcohol, PCBs and pesticides) are too low to be confounders. This presentation describes the study design, test batteries, analysis plan, and reviews the results of developmental and neurodevelopmental examinations through 66 months of age. Test results show the expected associations between co-variates and developmental endpoints. No adverse association between prenatal exposure and any developmental endpoint has been found.

The Seychelles Child Development Study: Testing and Results at 9 years of Age

Gary J. Myers
University of Rochester School of Medicine and Dentistry

The Seychelles Child Development Study (SCDS) is testing the hypothesis that there is an association between prenatal exposure to methylmercury (MeHg) from maternal fish consumption and child development. This longitudinal study enrolled 789 mother-child pairs at six months of age and this talk describes the results of the children's fifth neurodevelopmental evaluation at 9 years of age. The evaluation consisted of two three-hour test batteries given individually. All of the tests have been commonly used in previous developmental neurotoxicological studies. The tests examined global and domain specific abilities and included nearly all of the tests previously reported to show an adverse association with prenatal MeHg exposure. They specifically tested cognition (memory, attention, executive functions) and learning, perceptual, motor, social and behavioral abilities.

A total of 21 primary endpoints were analyzed for their relationship with prenatal MeHg exposure. Test results showed the expected associations between co-variates and developmental endpoints, as have tests from previous evaluations of this cohort. Two out of 21 endpoints showed a significant association with prenatal exposure; one association was adverse (the grooved pegboard, non-dominant hand) and the other was beneficial (Conner's Teacher Rating Scale, ADHD Index). No other significant associations between exposure and outcome were found. These findings do not support an association between prenatal exposure to MeHg from uncontaminated ocean fish consumption and adverse neurodevelopmental consequences in a population not exposed to other neurotoxins.

Philip W. Davidson
Professor of Pediatrics and Psychiatry
University of Rochester School of Medicine and Dentistry
Rochester, NY

Philip W. Davidson is Professor of Pediatrics and Psychiatry at the University of Rochester School of Medicine and Dentistry. He is director of the Strong Center for Developmental Disabilities (a University Center of Excellence in Developmental Disabilities Education, Research and Service), a position that he has held since 1975.

Dr. Davidson received his doctorate in experimental psychology from the George Washington University in 1970. Between 1970 and 1973 he was Assistant Professor of Psychology at Washington College in Chestertown, Maryland. After a Postdoctoral Fellowship in Pediatric Psychology at the Division for Disorders of Development and Learning at the University of Carolina at Chapel Hill in 1973-1974, Dr. Davidson worked for two years at the Monroe Developmental Center in Rochester, New York before joining the faculty of the University of Rochester.

Dr. Davidson's research reflects an interest in lifespan and aging effects on health and mental health outcomes in persons with developmental disabilities. For the past 14 years, he has been a senior investigator on the Seychelles Child Development Study examining the developmental neurotoxicity of methylmercury. He has 83 peer-reviewed publications and 53 book chapters and is the editor or co-editor of two books. He serves on the editorial boards of the *Journal of Intellectual Disability Research*, the *American Journal on Mental Retardation*, and the *International Review of Research in Mental Retardation*, and was Associate Editor of *Applied Research in Mental Retardation.*

Dr. Davidson has served as President of the Psychology Division of the American Association on Mental Retardation. He was Secretary-Treasurer of APA Division 12, Section 5 (the Society of Pediatric Psychology), and held the same office on the American Association of University Affiliated Programs. Currently he is President of Division 33 (Mental Retardation and Developmental Disabilities) of the American Psychological Association. In 1988, he served as Chairperson of the American Association on Mental Retardation *Task Force on Aversive Therapy.* He is a member of the Society of Pediatric Research.

Dr. Gary J. Myers
Professor of Neurology and Pediatrics
University of Rochester Medical Center, Rochester, NY

Dr. Myers is professor of Neurology and Pediatrics at the University of Rochester Medical Center, Rochester, New York. He trained in both subspecialties at Children's Hospital Medical Center in Boston, Massachusetts and later trained in Neonatology at the University of Rochester. For the past 30 years he has been actively involved in the practice of clinical Pediatric Neurology. He is engaged in teaching medical students and residents training in both Pediatrics and Neurology. He is also the medical director of the follow up clinic for high risk neonates who have been in the Neonatal Intensive Care Unit.

In the 1970's he became involved in studying the methyl mercury (MeHg) poisoning in Iraq. He and his colleague, Dr. David Marsh, traveled to Iraq on multiple occasions to examine children who were in utero at the time of the poisoning. This led to a long standing interest in environmental toxins and the present study in the Republic of Seychelles testing the hypothesis that mothers who expose their fetus to MeHg during pregnancy by fish consumption may adversely affect their children's neurodevelopment. He examined all of the children in Seychelles for the pilot study and lived in Seychelles for a year to enroll the main study children. Dr. Myers has authored a number of scientific papers related to pediatric neurology and neurotoxicology.

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Twenty-Seven Years Studying the Human Neurotoxicity of Methylmercury Exposure[1]

G. J. Myers,* P. W. Davidson,† C. Cox,‡ C. Shamlaye,§ E. Cernichiari,¶ and T. W. Clarkson¶

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Research at the University of Rochester (U of R) has been focused on mercury for nearly half a century. Initially studies focused on dosimetry, especially the accuracy of measuring exposure, and experimental work with animal models. Clinical studies in human populations started when the U of R mercury group was asked to assist with dosimetry in the Iraq epidemic of 1971–1972. Initial clinical studies described the effects of methylmercury (MeHg) poisoning on adults and children. A dose–response curve for prenatal exposure was determined and it suggested that relatively low exposures might be harmful to the fetus. Since most human exposure to MeHg is dietary from fish consumption, these theoretical dangers had far-reaching implications. After Iraq, the Rochester team pursued exposure from fish consumption in both adults and children. Populations with high fish consumption were identified in Samoa and Peru for studying adults and in Peru and the Seychelles islands for studying children. The possible health threat to the fetus from maternal fish consumption quickly became the focus of research efforts. This paper reviews the Rochester experience in studying human exposure to MeHg from fish consumption. © 2000 Academic Press

Key Words: **Mercury; methyl mercury; clinical research; child development; Seychelles.**

INTRODUCTION

The study of mercury has been a priority at the University of Rochester (U of R) since the 1950s. Initially studies concentrated on measurement of exposure and the consequences of exposure to various forms of mercury on experimental animals (Magos *et al.*, 1964; Clarkson and Rothstein, 1964; Clarkson *et al.*, 1965; Clarkson and Magos, 1966, 1967; Clarkson and Greenwood, 1968). However, in 1972 following the Iraq poisoning research interest turned toward the human health effects of methylmercury (MeHg) exposure. Immediately after the poisoning was discovered there was great concern about the safety of commercial food since MeHg is tasteless and odorless and usually has a latency period of weeks before clinical effects are seen. The U of R team was asked by the Iraqi government to establish a laboratory and measure mercury in food to determine whether it was safe to consume. Fortunately, the commercial food was safe and the primary exposure was the consumption of MeHg-treated seed grain by large numbers of rural families. This presented a unique opportunity to examine the clinical outcomes and to relate symptoms and signs to accurate measures of exposure.

Initial studies in Iraq provided extensive experience in measuring exposure and documenting the effects of varying exposure levels at different ages (Clarkson *et al.*, 1974, 1976, 1981a,b; Cox *et al.*, 1989; Rustam and Hamdi, 1974; Rustam *et al.*, 1975; Von Burg and Rustam, 1974a,b; Bakir *et al.*, 1973, 1976; Magos *et al.*, 1976; Greenwood *et al.*, 1977, 1978; Greenwood, 1985; Amin-Zaki *et al.*, 1974a, b, c, 1976, 1978, 1979, 1980, 1981; Elhassani *et al.*, 1978; Marsh *et al.*, 1977, 1980, 1981, 1987). In adults the earliest symptom reported was paresthesias and the earliest clinical finding was ataxia (Bakir *et al.*, 1973). For prenatal exposure, a dose–response curve was found (Marsh *et al.*, 1987; Cox *et al.*, 1989, 1995). A series of studies examining the effects of human exposure subsequently followed in Canada (Wheatley *et al.*, 1979; Kershaw *et al.*, 1980; Phelps *et al.*, 1980). As experience grew in measuring mercury in

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biological samples, the Environmental Health Sciences clinical laboratory became recognized for its expertise. A number of research opportunities related to mercury exposure arose from cooperation in measuring exposure (Davis *et al.*, 1994; Grandjean *et al.*, 1992; Gotelli *et al.*, 1985; Englender *et al.*, 1980; Fagan *et al.*, 1977; Nierenberg *et al.*, 1998).

However, the primary human exposure to MeHg is dietary from fish consumption. Consequently, research efforts turned to the study of individuals consuming large amounts of fish and whether low-level chronic exposure from this source could present a health risk. Could exposure at any age adversely affect the nervous system? The theoretical danger to the fetus from MeHg exposure following maternal fish consumption had the most far-reaching implications. This review will focus primarily on the clinical studies our team members have participated in which relate to human dietary exposure to MeHg from fish consumption.

Studies with Adults

Iraq. The Iraq poisoning epidemic occurred during the winter of 1971–1972 and presented a unique opportunity to study the effects of this neurotoxin. Iraqi physicians immediately recognized the cause of the poisoning since they had experience with MeHg poisoning from an outbreak in 1960 when nearly 1000 patients were affected (Al-Damluji, 1976). The Iraqi government promptly asked the U of R team to establish a laboratory to analyze food supplies for mercury contamination and to measure exposure in biological specimens. The Iraqi government promptly warned the population, collected the remaining treated grain, and prohibited the slaughter of animals (Al-Tikriti and Al-Mufti, 1976). The early recognition of MeHg as the cause and public action by the authorities limited the time of exposure to a few months. Even so, there were 6530 patients with poisoning admitted to hospital, 459 who died, and perhaps as many as 50,000 actually exposed (Bakir *et al.*, 1973; Greenwood, 1985). Fortunately, commercial food sources were not contaminated. The primary exposure was MeHg-treated seed grain which had been disseminated for planting and which rural farmers used to bake bread.

Following the epidemic, both blood and hair levels of MeHg were measured. Blood levels fell quickly when exposure stopped and hair levels were more useful in recapitulating the exposure (Bakir *et al.*, 1973). Hair was measured segmentally and the peak mercury exposure level was determined. This allowed an accurate determination of both the timing and the level of exposure in affected individuals. In collaboration with Iraqi colleagues the Rochester group carried out a series of clinical studies examining the effects of MeHg on adults (Bakir *et al.*, 1 1976; Clarkson *et al.*, 1974, 1976, 1981a, b; Greenwood *et al.*, 1978; Magos *et al.*, 1976; Rustam *et al.*, 1975; Smith *et al.*, 1976; Von Burg and Rustam, 1974a, b). These studies confirmed the devasting effects of this neurotoxin on adults and provided valuable information on the association between level of exposure and clinical effects. Paresthesias were found to be the first clinical symptom reported by patients (Bakir *et al.*, 1973). The first clinical finding was ataxia. If the exposure was sufficiently high, dysarthria, deafness, and eventually death followed ataxia.

Samoa. U of R team members next sought an adult population that consumed large quantities of fish with high mercury levels to see whether clinical symptoms or signs could be detected (Marsh *et al.*, 1974). They selected Samoa and examined two fish-eating populations there. The first study consisted of 88 men working on tuna fishing boats. These men were at sea for up to 47 weeks a year during which time their primary diet was the tuna they caught and rice. Their fish consumption was estimated at 10.4 oz daily. The second study consisted of 45 Samoans working in a tuna-packing factory. Their pri mary dietary protein was also fish, but in sm amounts and with a more varied diet. Fish consumption was about 7 oz per day in males and 3.7 oz in females. The evaluations of both study groups consisted of a history and neurological examination along with biological samples to determine exposure. Hair mercury values ranged up to 24 ppm among shore workers (mean 8 ppm) and 48 ppm among the tuna fishermen (mean 17 ppm). No individual had any symptoms or signs suggestive of MeHg poisoning. In this population adults with MeHg exposure from consuming large quantities of fish reported no symptoms compatible with poisoning and showed no associated neurological abnormalities on examination.

Peru. The next adult population studied was from Northern Peru (Turner *et al.*, 1974, 1980). One hundred ninety individuals from a coastal fishing community who consumed over 1 kg of fish weekly for many years were evaluated. They ranged in age from 1.5 to 82 years and had a mean blood MeHg level of 82 ppb (range of 11 to 275). Sixty-eight (35%) reported paresthesias, but there was no evidence of neurological impairment on examination. Therefore, 93 subjects from inland who consumed only small quantities of fish were examined. They had a me blood MeHg of 9.9 ppb (range 3.3 to 25.1) and 56 (60%) reported paresthesias. No association between paresthesias and MeHg exposure from regular long-term fish consumption could be established.

Studies with Children

Iraq. The opportunity to study prenatal and postnatal exposure to MeHg with accurate exposure data led to a number of studies on children (Amin-Zaki *et al.*, 1974a,b,c, 1976, 1978, 1979, 1980, 1981; Elhassani *et al.*. 1978; Cox *et al.*, 1989, 1995; Marsh *et al.*, 1977, 1980, 1981, 1987). Initial studies were observational ones of prenatal and postnatal exposure and the children's outcomes. In addition, studies of breast-feeding and treatment regimens were carried out (Greenwood *et al.*, 1978; Elhassani *et al.*, 1978). The concentration of MeHg in breast milk and its relation to maternal blood levels was determined and breast-feeding was found to slow the clearance of mercury (Amin-Zaki *et al.*, 1974b, 1976, 1981).

One of the most important studies carried out was of prenatal exposure in mothers who were pregnant during the poisoning (Marsh *et al.*, 1987; Cox, 1989, 1995). The prenatal exposure level was determined by measuring the mercury level in the maternal hair growing during pregnancy. Iraqi women had long hair and exposures could be determined over a period of years. The mothers were located and interviewed about their pregnancy and the children's development. Specifically the mothers were asked the age at which the child walked independently and first used two or more meaningful words. The children were then examined neurologically. Data were gathered on over 80 mother–infant pairs. A dose–response curve for the association between prenatal exposure and attainment of developmental milestones (walking unaided before or after 18 months of age and using two meaningful words before or after the age of 24 months) and neurological findings was determined. A dose–response curve for both developmental milestones and a score from the neurological examination suggested that prenatal exposure as low as 10 ppm peak mercury in maternal hair growing during pregnancy might be associated with adverse fetal consequences.

These findings raised concern about a possible public health issue as reviewed by Marsh (1994). It was previously known that most human exposure to MeHg was dietary and mainly from fish consumption. It was also known that individuals who consumed fish regularly often had hair mercury levels of 10 ppm or higher. If the dose–response curves from Iraq were applicable to prenatal exposure from fish consumption, as well as poisoning, then a significant public health problem might exist.

Although concerning in theory, it was unclear how applicable data from a poisoning episode were to exposure from dietary sources. In addition, the Iraq study had some limitations. Interviews of the mother were done through interpreters at a mean child age of 30 months. Birth dates were ascertained in relation to other events (i.e., seasons or holidays) since they are not important in the Arabic culture. The background rate of neurological abnormalities in the population was unknown. Covariates such as social and economic differences were not determined. There were substantial cultural differences between the families since they were widely scattered throughout Iraq. It seemed clear that studies in populations exposed to MeHg from consuming large amounts of fish were needed. Fortunately, such populations exist.

Peru. A study of prenatal exposure and its association with the child's development was first undertaken on the coast of Peru (Marsh *et al.*, 1995b). A total of 131 mother–infant pairs from a fishing village were enrolled. The mothers regularly consumed fish and had a mean hair MeHg level of 7 ppm (range 0.9 to 28 ppm). The same protocol that was used in Iraq was employed. Mother's were interviewed to determine the developmental milestones and the children had a neurological examination. No association was found between the children's prenatal exposure to MeHg and their development or neurological findings. However, it was unclear how definitive these findings were. The study was cross sectional, the evaluations were limited, and it was not possible to follow the children longitudinally.

Seychelles. The U of R team next sought a population with prenatal MeHg exposure from consuming fish which could be studied intensively, longitudinally, and with a minimum of confounding factors. Matthews (1983) had described such a population from the Republic of Seychelles and this became the study site (Marsh *et al.*, 1995a; Shamlaye *et al.*, 1995). In the Republic of Seychelles most individuals consume fish daily and do not consume marine mammals. The islands are 1000 miles from the nearest continent and there is minimal local industry with no known local pollution. Basic and preventive health care is free, readily available, and of high quality. Over 90% of women have prenatal care and deliver in a single central hospital. Maternal consumption of alcohol and use of tobacco are very low. Perinatal mortality is very low (13.4/1000 in 1990) and children's immunization rates are high (over 90%). There is no malaria or malnutrition. Education begins at age 3.5 years and is free, readily available, and of good quality. Over 90% of the population resides on the main island of Mahe and transportation around the island is excellent. In addition, the government, health authorities, and the people are cooperative, and the current conditions have been similar for many years.

In the early 1980s we started to monitor mercury exposure during pregnancy by measuring it in maternal hair samples taken at delivery. In 1987–1988 we enrolled a cross sectional pilot cohort on whom there was accurate prenatal exposure data (Cernichiari *et al.*, 1995a; Myers *et al.*, 1995a). Mother–child pairs were recalled and evaluated in their local health clinics. The assessment team consisted of a Seychellois nurse responsible for translating and a pediatric neurologist. All evaluators and personnel in Seychelles were blinded to the mother's mercury exposure. The evaluation consisted of a standardized questionnaire, a neurological examination, and administration of the Denver Developmental Screening Test–Revised. A total of 804 mother–child pairs were evaluated over a 1-year time period. After 15 exclusions for maternal or child medical conditions highly associated with developmental problems there were 789 children for analysis. No association between mercury exposure and any endpoint was seen. However, Kjellstrom and colleagues (1986) had proposed a nonstandard scoring procedure (questionable scores combined with abnormal scores) in an earlier study from New Zealand. Using the nonstandard procedure they found an association with mercury exposure, and when the Seychelles data were grouped in this manner an association was present (Myers *et al.*, 1995a). As the children's prenatal MeHg exposure increased there was an increasing number of these nonstandard combined scores. The response rates were higher in males and decreased as the children got older. It appeared that an association between exposure and endpoints was present using more specific developmental testing than in Iraq or Peru. The use of nonstandard scoring to determine a relationship with MeHg exposure during the enrollment was concerning and plans for a more detailed main study continued.

Subsequently, a subset of 217 of the pilot cohort was tested at 66 months of age (Myers *et al.*, 1995b). An association between development and prenatal MeHg exposure was present. However, when a small number of outliers and influential points were removed to normalize the data only one association remained. The associations were dependent on a few outliers and influential scores.

Eighty-eight pilot cohort children were evaluated at the age of 9 years with some of the test battery used for the main cohort study. These pilot data are now available and show associations between prenatal MeHg exposure and neurodevelopmental outcomes but in a beneficial direction (Davidson *et al.*, 2000). For males, performance on the Boston Naming test increased 4 points for every 10 ppm of maternal hair MeHg. Also, for males timed scores on the Grooved Pegboard improved (i.e., decreased) 10 s for every 10 ppm of maternal hair MeHg, and scores on the Beery Buktenica Developmental Test of Visual Motor Integration improved (increased) 6 points for every 10 ppm of MeHg. The small sample size and the presence of some influential points in these analyses make us cautious in interpreting these data.

In 1989 enrollment of the main cohort began. Like the pilot study, exposure was measured in maternal hair samples growing during pregnancy and all examiners and Seychellois were blinded to the exposure level. A number of modifications were made to the protocol based on our experience with the pilot study and a review of the literature (Davidson *et al.*, 1994). Evaluations of the children took place in age windows to reduce the problems of comparing developmental differences in children of different ages. The age windows were ± 2 weeks for evaluations at 6.5, 19, and 29 months, ± 3 months at 66 months, and ± 6 months at about 9 years of age. To accomplish this logistically, enrollment took place over a 12-month time period. The main study was restricted to the island of Mahe where 95% of the children reside. A Child Development Center was established where the children could be examined in an environment conducive to optimal performance. The questionnaire concerning history and covariates was expanded, as were the evaluations. Tests that had previously been reported to show associations with mercury exposure in humans or animals were added and the testing battery was expanded (Gunderson *et al.*, 1988; Marsh *et al.*, 1995a). The children's health records were obtained and examined for medical exclusions. Children with medical conditions highly associated with developmental problems (major congenital anomalies, perinatal seizures, epilepsy, significant head trauma, etc.) were excluded from analysis.

At 6.5 months we enrolled 779 mother–infant pairs (Myers *et al.*, 1995c). Thirty-nine children met *a priori* exclusion criteria (15 with inadequate maternal hair to recapitulate exposure, 18 for medical exclusion criteria, and 6 twins). The final cohort for analysis at enrollment was 740. Testing has been carried out at regular intervals (6.5, 19, 29, 66, and

TABLE 1

Tests and Endpoints Evaluated in the Main SCDS Study through 66 Months and Those Examined at 108 Months

6 Months
- Neurological examination
  - Overall neurological score
  - Muscle tone
  - Deep tendon reflexes
- Fagan test of visual recognition memory
  - Memory score
  - Attention subscale
  - Denver Developmental Screening Test

19 Months
- Developmental milestones (by maternal history)
  - Age child first walked
  - Age child first said two words
- Bayley Scales of Infant Development
  - Mental Developmental Index
  - Physical Developmental Index
  - Kohen-Raz (perceptual motor subscale)

29 Months
- Bayley Scales of Infant Development
  - Mental Developmental Index
  - Physical Developmental Index
  - Infant Behavior Record
- Activity
  - Attention
  - Cooperation
  - Happiness
  - Response to examiner

Response to mother

66 Months
- McCarthy Scales of Children's Abilities
  - General Cognitive Index
  - Verbal
  - Perceptual-performance
  - Memory
  - Quantitative
  - Motor
- Preschool Language Scale
  - Total language
- Woodcock-Johnson Achievement Test
  - Letter-word
  - Applied problems
- Bender-Gestalt
  - Errors
- Child Behavior Checklist
  - Overall
  - Internalization
  - Externalization
  - Attention
  - Anxiety
  - Withdrawal
  - Social problems
  - Learning problems
  - Conduct problems
  - Sexual problems

108 Months
- Wechsler Intellegence Scale for Children III
  - Full scale IQ
  - Verbal IQ
  - Performance IQ
  - Verbal comprehension
  - Perceptual organization
  - Processing speed
  - Freedom from distractibility
- Test of motor development (Bruinincks-Oseretsky)
  - Total score
- Berry-Buktenica Developmental Test of Visual Motor Integration
  - Developmental score
- Woodcock-Johnson Achievement Test
  - Letter-word
  - Applied problems
- Child Behavior Checklist
  - Overall
- Connors Teacher Rating Scale
  - Total score
- California Verbal Learning Test
  - Trials 1-5 total
  - List A, Short delay recall
  - List A, Long delay recall
- Wide Range Assessment of Memory & Learning
  - Design memory subtest
- Trial Making
  - Time to complete
  - Errors
- Finger Tapping
  - Average time-Preferred hand
  - Average time—Nonpreferred hand
- Grooved Pegboard
  - Average time—Preferred hand
  - Average time—Nonpreferred hand
  - Drops—Preferred hand
  - Drops-Nonpreferred hand
- Boston Naming Test
  - Total score
- Haptic discrimination test
  - Errors
- Connors Continuous Performance Task
  - Number of hits
  - Number of omissions
  - Number of commissions
  - Hit reaction time
  - Attentiveness (d')
  - Risk-taking (B)

96 months). The tests appear to be working well in this population (Davidson *et al.*, 1995a), and attrition at evaluations of the cohort over the first 66 months was minimal (738 at 19 months, 736 at 29 months, and 711 at 66 months). All of the endpoints evaluated to date in Seychelles are listed in Table 1.

The results of primary and some secondary analyses through the 66-month evaluations have been reported (Myers *et al.*, 1995c, d, 1997a, b; Davidson *et al.*, 1995b, 1998, in press; Axtell *et al.*, 1998). Developmental milestones were specifically examined since they were a key endpoint from the Iraq study (Marsh *et al.*, 1987). In Seychelles there was no significant delay in milestone achievement (Myers *et al.*, 1997a). A similar result has been reported from the Faroe Islands (Grandjean *et al.*, 1995). A number of associations between both prenatal and postnatal indices of MeHg exposure and endpoints have been found in the Seychelles (Table 2). Birth weight was associated with prenatal exposure to MeHg. In the reduced model the gender interaction was statistically significant (0.05), and both slopes were positive. The slope (SE) for males was 0.015 (0.005) and for females 0.0008 (0.005), but only the slope for males was significant ($P = 0.0038$). At 29 months prenatal mercury exposure was associated with decreasing activity in males on the Infant Behavior Record from the Bayley Scales of Infant Development (Davidson *et al.*, 1995b). As prenatal exposure increased the activity level decreased. The behavior rating scale is a subjective assessment and the significance of this finding is unclear. Prenatal exposure was associated with effect modification as described by Bellinger (2000) at the 19-month evaluation (Davidson *et al.*, 1999). Children had higher scores on the BSID-MDI when their caregiver IQ fell in a higher category. This relationship was present at several levels of family income. At 66 months associations were present with prenatal and postnatal mercury exposure, but all were in a beneficial direction (Davidson *et al.*, 1998). The total score from the Preschool Language Scale (PLS) was associated with both prenatal and postnatal exposure indices. Postnatal exposure was associated with improvements in the Applied Problems subtest from the Woodcock–Johnson Achievement Test and the error score from the Bender Gestalt Test, but the latter was present in males only.

The SCDS 9-year reevaluation of the Main Study cohort is now complete. It included a 4-h-long battery of neuropsychological tests given in two separate sessions. Many of the tests used were the same or similar to those in the Faroe Islands study (Grandjean *et al.*, 1997). The data are currently being analyzed.

TABLE 2

Associations Found between Prenatal and Methylmercury Exposure and Endpoints in the Seychelles Child Development Study during the First 5.5 Years of Life

Cohort	Age	Test	Exposure	Males	Females	Reference
Main	Birth	Birth weight	Prenatal	B	NE	NIEHS Conference on MeHg 11/99 Available at www.niehs.nih.gov
	19 months	Enhanced BSID-MDI with increasing MeHg exposure in higher caregiver IQ groups at several levels of family income	Prenatal	B	B	Davidson *et al.*, 1999
	29 months	BSID-IBR—Activity[a]	Prenatal	?	NE	Davidson *et al.*, 1995b
	66 months	PLS—Total score	Prenatal	B[b]	B[b]	Davidson *et al.*, 1998
		PLS—Total score	Postnatal	B[b]	B[b]	Davidson *et al.*, 1998
		W-J Applied Problems	Postnatal	B[b]	B[b]	Davidson *et al.*, 1998
		Bender-Gestalt—Errors	Postnatal	B	NE	Davidson *et al.*, 1998
Pilot	96 months	Boston Naming Test	Prenatal	B	NE	Davidson *et al.*, 2000
		Beery-Buktenka (VMI)	Prenatal	B		Davidson *et al.*, 2000
		Grooved Pegboard	Prenatal			Davidson *et al.*, 2000
		Preferred hand		B	A	
		Nonpreferred hand		B	NE	

Note. A, adverse; B, beneficial; NE, no effect; BSID, Bayley Scales of Infant Development; MDI, Mental Developmental Index; IBR, Infant Behavior Record; W-J, Woodcock–Johnson Test of Achievement; PLS, Preschool Language Scale; MSCD, McCarthy Scales of Children's Development; GCI, MSCD general cognitive index, VMI, Visual motor integration (analogous to Bender).

[a] Activity during the testing session was rated by the tester. For males, activity decreased with increasing maternal MeHg. No association was seen for females. It is unclear whether this result was adverse or beneficial.

[b] Single slope. Gender × MeHg interaction was not significant.

In addition to clinical studies, we have sought pathological evidence that might suggest that MeHg exposure from fish consumption had adverse effects on the nervous system. Earlier work from the U of R indicated that MeHg exposure at measured brain tissue levels below 2 ppm might affect the central nervous system of animals (Rodier *et al.*, 1984; Sager *et al.*, 1984). Consequently, we examined neuropathological material from stillbirths and natural deaths in Seychelles (Lapham *et al.*, 1995). No association between the mercury content of brain and histopathological changes was found. However, associations were found between the mercury content of various biological tissues (Cernichiari *et al.*, 1995b). The concentration of mercury in six brain regions was highly correlated with hair mercury levels.

To date in the SCDS we have found no adverse associations between either prenatal or postnatal exposure from fish consumption and neurological, developmental, or neuropathological endpoints. The SCDS is continuing to follow the pilot and main cohorts as they mature and is testing the children with increasingly sensitive test measures.

DISCUSSION

The results of clinical studies carried out following the MeHg poisoning in Iraq confirmed the neurological deficits reported from Japan and provided data on the level of exposure associated with neurological and developmental findings. These data raised concern that exposure to MeHg from fish consumption might be associated with adverse effects. However, our subsequent studies in Samoa, Peru, and the Seychelles have consistently found no evidence to support this hypothesis. Our research has not identified any adverse associations between MeHg exposure from fish consumption and clinical symptoms or signs. However, our studies of both prenatal and postnatal measures of MeHg exposure from fish consumption in Seychellois children have been associated with beneficial effects.

These results differ from those found in a similar epidemiologic study being carried out in the Faroe Islands (Grandjean *et al.*, 1997, 1998). The Faroe study reported adverse associations between prenatal MeHg exposure and tests of memory, attention, language, motor function, and visual spatial perception. There are many similarities between these two epidemiological studies. Both are double-blind studies examining large cohorts with prenatal dietary exposure to MeHg. However, there are also substantial differences including the data analysis. Table 3 outlines some of the important differences between these studies, and one or more of these may explain the differing conclusions.

Exposure to MeHg from fish consumption differs in a number of important ways from MeHg poisoning. With fish consumption the exposure is to very small amounts of MeHg over a long time period. The concentration of MeHg present in oceanic fish in the Seychelles averages about 0.3 ppm. In North America MeHg levels in fish are generally similar. However, ocean fish from polluted waters such as those at Minamata Bay in Japan had MeHg levels as high as 40 ppm, and freshwater fish from North America have been reported with concentrations as high as 10 ppm (Swedish Expert Group 1971; Shephard, 1976). The small amount of MeHg consumed with each exposure and spreading the exposure over a longer time period may alter the way the human body handles it. Clarkson (1995) has suggested that the liver may excrete or detoxify small amounts, but may be unable to handle larger amounts. Exposure to MeHg in conjunction with other components of fish such as selenium and amino acids may also

TABLE 3

Differences between the Seychelles and Faroe Island Epidemiologic Studies of Dietary Prenatal MeHg Exposure

Issue	Seychelles	Faroe Islands
Genetic/ethnic composition	African, Asian, and mixed	Scandinavian
Source of exposure to MeHg	Fish	Pilot whale and fish
Exposure to other toxins	None known	PCBs, possibly others
Measure of exposure	Maternal hair	Cord blood and maternal hair
Age at evaluation	6.5, 19, 29, 66, and 96 months	7 years
Exclusions	Medical problems highly associated with developmental delay	None
Covariates used in this study, but not in the other	7	4
Composition of test battery	Neurological Developmental Psychological	Neurological Neuropsychological Neurophysiological

influence its potential toxicity in other ways. Selenium may decrease any potentially toxic effects and amino acids may compete with MeHg for transport into the brain (Clarkson, 1995; WHO, 1990).

In addition, fish consumption may provide important nutrients and is an important source of calories and protein to many populations around the world, especially indigenous ones. Long-chain polyunsaturated fatty acids, mainly docosahexanoic and other omega 3 fatty acids, are high in fish and believed to be important in brain development (Innis, 1991, Uauy-Dagach and Valenzuela, 1996). Omega 3 fatty acids may simply improve brain performance enough that any adverse effects from this level of MeHg exposure are not apparent. Fish consumption has also been reported to have beneficial effects at later ages (Kromhout *et al.*, 1985; Daviglus *et al.*, 1997). We agree with Egeland and Middaugh (1997) that the benefits, alternatives, and possible risks of fish consumption should be weighed carefully before public health actions are taken that might reduce fish consumption.

SUMMARY

The clinical studies that our team has carried out in Samoa, Peru, and the Seychelles provide no evidence that consuming large quantities of fish is associated with adverse effects on adults or children. Our studies do show an association between test performance and MeHg exposure, but it is enhanced performance associated with both prenatal and postnatal exposure. Since MeHg is clearly neurotoxic there must be some factor in fish that covaries with exposure to account for improved performance. The absence of adverse effects is reassuring in terms of any significant risk to the child from prenatal or postnatal MeHg exposure from fish consumption. However, we are continuing to study the Seychelles cohorts with increasingly sensitive and sophisticated tests at older ages to identify associations that might appear as they mature. Restricting fish consumption without clear justification could potentially adversely affect children's development. This is especially true in societies where fish is the primary source of protein.

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REFERENCES

Al-Damluji, S. F. (1976). Organomercury poisoning in Iraq: History prior to the 1971–1972 outbreak. Conference on intoxication due to alkylmercury-treated seed, Baghdad, Iraq, 9–13 September, 1974. *Bull. WHO* **53** (Suppl.), 11–14.

Al-Tikriti, K., and Al-Mufti, A. W. (1976). An outbreak of organomercury poisoning among Iraqi farmers. Conference on intoxication due to alkylmercury-treated seed, Baghdad, Iraq, 9–13 September, 1974. *Bull. WHO* **53** (Suppl.), 15–21.

Amin-Zaki, L., Elhassani, S., Majeed, M. A., Clarkson, T. W., Doherty, R. A., and Greenwood, M. R. (1974a). Intra-uterine methylmercury poisoning in Iraq. *Pediatrics* **54**, 587–595.

Amin-Zaki, L., Elhassani, S., Majeed, M. A., Clarkson, T. W., Doherty, R. A., and Greenwood, M. R. (1974b). Studies of infants postnatally exposed to methylmercury. *J. Pediatr.* **85**, 81–84.

Amin-Zaki, L., Elhassani, S., Majeed, M. A., Clarkson, T. W., Doherty, R. A., and Greenwood, M. R. (1974c). Prenatal methylmercury poisoning in Iraq. *In* "Proceedings of the First International Mercury Conference," Vol. II, pp. 319–325. Fabrica National de Moneda U Timbre, Madrid, Spain.

Amin-Zaki, L., Elhassani, S., Majeed, M. A., Clarkson, T. W., Doherty, R. A., Greenwood, M. R., and Giovanoli-Jakubczak, T. (1976). Prenatal methylmercury poisoning in Iraq. *Am. J. Dis. Child.* **130**, 1070–1076.

Amin-Zaki, L., Majeed, M. A., Clarkson, T. W., and Greenwood, M. R. (1978). Methylmercury poisoning in Iraqi children: Clinical observations over two years. *Br. Med. J.* **1**, 613–616.

Amin-Zaki, L., Majeed, M. A., Elhassani, S. B., Clarkson, T. W., Greenwood, M. R., and Doherty, R. A. (1979). Prenatal methylmercury poisoning: Clinical observations over five years. *Am. J. Dis. Child.* **133**, 172–177.

Amin-Zaki, L., Elhassani, S. B., Majeed, M. A., Clarkson, T. W., Doherty, R. A., and Greenwood, M. R. (1980). Methylmercury poisoning in mothers and their suckling infants. *In* "Mechanisms of Toxicity and Hazard Evaluation" (B. Holmsted, R. Lauwerys, M. Mercier, and M. Roberfroid, Eds.), pp. 75–78. Elsevier/North-Holland Biomedical Press, Amsterdam.

Amin-Zaki, L., Majeed, M. A., Greenwood, M. R., Elhassani, S. B., Clarkson, T. W., and Doherty, R. A. (1981). Methylmercury poisoning in the Iraqi suckling infant: A longitudinal study over five years. *J. Appl. Toxicol.* **1**, 210–214.

Axtell, C. D., Myers, G. J., Davidson, P. W., Choi, A. L., Cernichiari, E., Sloane-Reeves, J., Shamlaye, C., Cox, C., and Clarkson, T. W. (1998). Semiparametric modeling of age at achieving developmental milestones after prenatal exposure to methylmercury in the Seychelles Child Development Study. *Environ. Health Perspect.* **106**(9), 559–564.

Bakir, F., Damluji, S. F., Amin-Zaki, L., Murtadha, M., Khalidi, A., Al-Rawi, N. Y., Tikriti, S., Dahir, H. I., Clarkson, T. W., Smith, J. C., and Doherty, R. A. (1973). Methylmercury poisoning in Iraq. *Science* **181**, 230–241.

Bakir, F., Al-Khalidi, A., Clarkson, T. W., and Greenwood, M. R. (1976). Clinical observations on treatment of alkylmercury poisoning in hospital patients. Conference on intoxication due to alkylmercury-treated seed, Baghdad, Iraq, 9–13 September 1974. WHO, Geneva. *Bull. WHO* **53** (Suppl.), 87–92.

Bellinger, D. (2000). Effect modification in epidemiologic studies of low-level neurotoxicant exposures and health outcomes. *Neurotoxicol. Teratol.* **22**, 133–140.

Cernichiari, E., Toribara, T. Y., Liang, L., Marsh, D. O., Berlin, M. W., Myers, G. J., Cox, C., Shamlaye, C. F., Choisy, O., Davidson, and Clarkson, T. W. (1995a). The biological monitoring of mercury in the Seychelles study. *Neurotoxicology* **16**(4), 613–628.

Cernichiari, E., Brewer, R., Myers, G. J., Marsh, D. O., Lapham, L. W., Cox, C., Shamlaye, C. F., Berlin, M., Davidson, P. W., and Clarkson, T. W. (1995b). Monitoring methylmercury during pregnancy: Maternal hair predicts fetal brain exposure. *Neurotoxicology* **16**(4), 705–710.

Clarkson, T. W., and Rothstein, A. (1964). The excretion of volatile mercury by rats injected with mercuric salts. *Health Phys.* **10**, 1115–1121.

Clarkson, T. W., Rothstein, A., and Sutherland, R. (1965). The mechanism of action of mercurial diuretics in rats: The metabolism of 203 Hg-labeled chlormerodrin. *Br. J. Pharmacol. Chemother.* **24**, 1–13.

Clarkson, T. W., and Magos, L. (1966). Studies on the binding of mercury in tissue homogenates. *Biochem. J.* **9**, 62–70.

Clarkson, T. W., and Magos, L. (1967). The effect of sodium maleate on the renal deposition and excretion of mercury. *Br. J. Pharmacol. Chemother.* **31**, 560–567.

Clarkson, T. W., and Greenwood, M. R. (1968). Simple and rapid determination of mercury in urine and tissues by isotope exchange. *Talanta* **115**, 54–555.

Clarkson, T. W., Smith, J. C., and Bakir, F. (1974). A preliminary report on a population that received a heavy exposure to methylmercury. *In* "Trace Substances in Environmental Health" (D. D. Hemphill, Ed.), pp. 127–135. Univ. of Missouri, Columbia.

Clarkson, T. W., Amin-Zaki, L., and Al-Tikriti, S. (1976). An outbreak of methylmercury poisoning due to consumption of contaminated grain. *Fed. Proc.* **35**(12), 2395–2399.

Clarkson, T. W., Magos, L., Cox, C., Greenwood, M. R., Amin-Zaki, L., Majeed, M. A., and Damluji, S. F. (1981a). Tests of efficacy of antidotes for removal of methylmercury in human poisonings during the Iraq outbreak. *J. Pharmacol. Exp. Ther.* **218**(10), 74–83.

Clarkson, T. W., Cox, C., Mash, D. O., Myers, G. J., Al-Tikriti, S., Amin-Zaki, L., and Dabbagh, A. R. (1981b). Dose-response relationships for adult and prenatal exposures to methylmercury. *In* "Measurement of Risk" (G. G. Berg and H. D. Maillie, Eds.), pp. 111–130. Plenum, New York.

Clarkson, T. W. (1995). Environmental contaminants in the food chain. *Am. J. Clin. Nutr.* **61** (Suppl.), 682S–686S.

Cox, C., Clarkson, T. W., Marsh, D. O., Amin-Zaki, L., Tikriti, S., and Myers, G. J. (1989). Dose-response analysis of infants prenatally exposed to methylmercury. An application of a single compartment model to single-strand hair analysis. *Environ. Res.* **31**, 640–649.

Cox, C., Marsh, D. O., Myers, G. J., and Clarkson, T. W. (1995). Analysis of data on delayed development from the 1971–1972 outbreak of methylmercury poisoning in Iraq: Assessment of influential points. *Neurotoxicology* **16**(4), 727–730.

Davidson, P. W., Myers, G. J., Cox, C., Shamlaye, C. F., Sloane-Reeves, J., Cernichiari, E., Marsh, D., Clarkson, T. W., and Tanner, M. A. (1994). Measuring neurodevelopmental outcomes of young children following prenatal dietary low dose methyl mercury exposures. *Environ. Sci.* **3**(1), 55–65.

Davidson, P. W., Myers, G. J., Cox, C., Shamlaye, C. F., Choisy, O., Sloane-Reeves, J., Cernichiari, E., Marsh, D. O., Berlin, M., Tanner, M. A., and Clarkson, T. W. (1995a). Neurodevelopmental test selection, administration and performance in the main Seychelles Child Development study. *Neurotoxicology* **16**(4), 665–676.

Davidson, P. W., Myers, G. J., Cox, C., Shamlaye, C. F., Marsh, D. O., Tanner, M. A., Berlin, M., Sloane-Reevesm, Cernichiari, E., Choisy, O., Choi, A., and Clarkson, T. W. (1995b). Longitudinal neurodevelopmental study of Seychellois children following *in utero* exposure to methylmercury from maternal fish ingestion: Outcomes at 19 and 29 months. *Neurotoxicology* **16**, 677–799.

Davidson, P. W., Myers, G. J., Cox, C., Axtell, C., Shamlaye, C., Sloane-Reeves, J., Cernichiari, E., Needham, L., Choi, A., Wang, Y., Berlin, M., and Clarkson, T. W. (1998). Effects of prenatal and postnatal methylmercury exposure from fish consumption on neurodevelopment. *J. Am. Med. Assoc.* **280**(8), 701–707.

Davidson, P. W., Myers, G. J., Shamlaye, C., Cox, C., Gao, P., Axtellm, C., Morris, D., Sloane-Reeves, J., Cernichiari, E., Choi, A., Palumbo, D., and Clarkson, T. W. (1999). Association between prenatal exposure to methylmercury and developmental outcomes in Seychellois children: Effect modification by social and environmental factors. *Neurotoxicology.* **20**(5), 833–842.

Davidson, P. W., Palumbo, D., Myers, G. J., Cox, C., Shamlaye, C. F., Sloan-Reeves, J., Cernichiari, E., Wilding, G. E., and Clarkson, T. W. (2000). Neurodevelopmental outcomes of seychellois children from the pilot cohort at 108 months following prenatal exposure to methylmercury from a maternal fish diet. *Environ. Sci.*, in press.

Daviglus, M. L., Stamler, J., Orencia, A. J., Dyer, A. R., Liu, K., Greenland, P., Walsh, M. K., Morris, D., and Shekelle, R. B. (1997). Fish consumption and the 30-year risk of fatal myocardial infarction. *N. Engl. J. Med.* **336**, 1046–1053.

Davis, L. E., Kornfeld, M., Mooney, H. S., Feidler, K. J., Haaland, K. Y., Orrison, W. W., Cernichiari, E., and Clarkson, T. W. (1994). Methylmercury poisoning: Long-term clinical, radiological, toxicological, and pathological studies of an affected family. *Ann. Neurol.* **35**, 680–688.

Egeland, G. M., and Middaugh, J. P. (1997). Balancing fish consumption benefits with mercury exposure. *Science* **278**, 1904–1905.

Elhassani, S., Amin-Zaki, L., Majeed, M. A., Clarkson, T. W., Doherty, R. A., Greenwood, M. R., and Kilpper, R. W. (1978). Exchange transfusion treatment of methylmercury-poisoned children. *J. Environ. Sci. Health* **13**(1), 63–80.

Englender, S. J., Atwood, R. G., Landrigan, P. J., Clarkson, T. W., Greenwood, M. R., and Smith, J. C. (1980). Organic mercury exposure from fungicide contaminated eggs. *Arch. Environ. Health* **35**(4), 224–228.

Fagan, D. G., Prichard, J. S., Clarkson, T. W., and Greenwood, M. R. (1977). Organ mercury levels in infants with omphaloceles treated with organic mercurial antiseptic. *Arch. Dis. Child.* **52**, 962–964.

Gotelli, C. A., Astolfi, E., Cox, C., Cernichiari, E., and Clarkson, T. W. (1985). Early biochemical effects of an organic mercury fungicide on infants: "Dose makes the poison." *Science* **22**, 638–640.

Grandjean, P., Weihe, P., Jorgensen, P. J., Clarkson, T., Cernichiari, E., and Videro, T. (1992). Impact of maternal seafood diet on fetal exposure to mercury, selenium, and lead. *Arch. Environ. Health* **47**, 185–195.

Grandjean, P., Weihe, P., and White, R. F. (1995). Milestone development in infants exposed to methylmercury from human milk. *Neurotoxicology* **16,** 27–34.

Grandjean, P., Weihe, P., White, R. F., Debes, F., Araki, S., Yokoyama, K., Murata, K., Sorensen, N., Dahl, R., and Jorgensen, P. J. (1997). Cognitive deficit in 7-year-old children with prenatal exposure to methylmercury. *Neurotoxicol. Teratol.* **19,** 417–428.

Grandjean, P., Weihe, P., White, R. F., and Debes, F. (1998). Cognitive performance of children prenatally exposed to "safe" levels of methylmercury. *Environ. Res.* **77,** 165–172.

Greenwood, M. R., Dhahir, P., Clarkson, T. W., Farrant, J. P., Chartrand, A., and Khayat, A. (1977). Epidemiological experiences with the Magos reagents in the determination of different forms of mercury in biological samples by flameless atomic absorption. *J. Anal. Toxicol.* 1(6), 265–269.

Greenwood, M. R., Clarkson, T. W., Doherty, R. A., Gates, A. H., Amin-Zaki, L., Elhassani, S., and Majeed, M. A. (1978). Blood clearance half-times in lactating and non-lactating members of a population exposed to methylmercury. *Environ. Res.* **16,** 48–54.

Greenwood, M. R. (1985). Methylmercury poisoning in Iraq. An epidemiological study of the 1971–1972 outbreak. *J. Appl. Toxicol.* **5**(3), 148–159.

Gunderson, V. M., Grant-Webster, K. S., Burbacher, T. M., and Mottet, N. K. (1988). Visual recognition memory deficits in methylmercury-exposed Macaca fasicularis infants. *Neurotoxicol. Teratol.* **10,** 373–379.

Innis, S. M. (1991). Essential fatty acids in growth and development. *Progress Lipid Res.* **30,** 39–103.

Kershaw, T. G., Clarkson, T. W., and Dhahir, P. H. (1980). The relationship between blood levels and dose of methylmercury in man. *Arch. Environ. Health* **35**(1), 28–36.

Kjellstrom, T., Kennedy, P., Wallis, S., and Mantell, C. (1986). Physical and mental development of children with prenatal exposure to mercury from fish. Stage 1. Preliminary tests at age 4. *Solna, National Swedish Environmental Board*. Report No. 3080, pp. 1–96.

Kromhout, D., Bosschieter, E. B., and Coulander, C. L. (1985). The inverse relation between fish consumption and 20-year mortality from coronary heart disease. *N. Engl. J. Med.* **312,** 1205–1209.

Lapham, L. W., Cernichiari, E., Cox, C., Myers, G. J., Baggs, R. B., Brewer, R., Shamlaye, C. F., Davidson, P. W., and Clarkson, T. W. (1995). An analysis of autopsy brain tissue from infants prenatally exposed to methylmercury. *Neurotoxicology* **16**(4), 689–704.

Magos, L., Tuffery, A. A., and Clarkson, T. W. (1964). Volitalization of mercury by bacteria. *Br. J. Ind. Med.* **21,** 294–298.

Magos, L., Bakir, F., Clarkson, T. W., Al-Jawad, A. M., and Al-Soffi, M. H. (1976). Tissue levels of mercury in autopsy specimens of liver and kidney. *Bull. World Health Org.* **53,** 93–96.

Marsh, D. O., Turner, M., Smith, J. C., Choi, W. J., and Clarkson, T. W. (1974). Methyl mercury in human populations eating large quantities of marine fish. II. American Samoa: Cannery workers and fishermen. *In* "Proc. First International Mercury Conf., May 1974, Barcelona," Vol. II, pp. 235–239. Fabrica National de Moneda y Timbre, Madrid, Spain.

Marsh, D. O., Myers, G. J., Clarkson, T. W., Amin-Zaki, L., and Tikriti, S. (1977). Fetal methylmercury poisoning: New data on clinical and toxicological aspects. *Trans. Am. Neurol. Asso.* **102,** 1–3.

Marsh, D. O., Myers, G. J., Clarkson, T. W., Am[illegible] L Tikriti, S., and Majeed, M. A. (1980). Fetal me[illegible] mercur poisoning: Clinical and toxicological data on 29 cases. *Ann Neurol.* **7,** 348–353.

Marsh, D. O., Myers, G. J., Clarkson, T. W., Amin-Zaki, L Tikriti, S., and Majeed, M. A. (1981). Dose–response relationship for human fetal exposure to methylmercury. *Clin. Toxicol.* **18,** 1311–1318.

Marsh, D. O., Clarkson, T. W., Cox, C., Myers, G. J., Amin-Zaki L., and Al-Tikriti, S. (1987). Fetal methylmercury poisoning *Arch. Neurol.* **44,** 1017–1022.

Marsh, D. O. (1994). Organic mercury: Clinical and neurotoxicological aspects. *In* "Handbook of Clinical Neurology, Interactions of the Nervous System" (F. A. deWolff, Ed.), Part 1 Vol. 20 (64), pp. 413–429. Elsevier, Amsterdam.

Marsh, D. O., Clarkson, T. W., Myers, G. J., Davidson, P. W., Cox, C., Cernichiari, E., Tanner, M. A., Lednar, W., Shamlaye, C. F., Choisy, O., Hoareau, C., and Berlin, M. (1995a). The Seychelles study of fetal methylmercury exposure and child development: Introduction. *Neurotoxicology* **16**(4), 583–596.

Marsh, D. O., Turner, M. D., Smith, J. C., Allen, P., and Richadale, N. (1995b). Fetal methylmercury study in a Peruvian fish-eating population. *Neurotoxicology* **16,** 717–726.

Matthews, A. D. (1983). Mercury content of commercially important fish of the Seychelles, and hair mercury levels of a selected part of the population. *Environ. Res.* **30,** 305–312.

Myers, G. J., Marsh, D. O., Cox, C., Davidson, P. W., Shamlaye, C. F., Tanner, M. A., Choi, A., Cernichiari, E., Choisy, O., and Clarkson, T. W. (1995a). A pilot neurodevelopment [illegible] of Seychellois children following *in utero* exposure t[illegible] mercury from a maternal fish diet. *Neurotoxicol* [illegible] **16**(4), 629–638.

Myers, G. J., Davidson, P. W., Cox, C., Shamlaye, C. F., Tanner, M. A., Choisy, O., Sloane-Reeves, J., Marsh, D. O., Cernichiari, E., Choi, A., Berlin, M., and Clarkson, T. W. (1995b). Neurodevelopmental outcomes of Seychellois children sixty-six months after *in utero* exposure to methylmercury from a maternal fish diet: Pilot study. *Neurotoxicology* **16**(4), 639–652.

Myers, G. J., Marsh, D. O., Davidson, P. W., Cox, C., Shamlaye, C. F., Tanner, M. A., Choi, A., Cernichiari. E., Choisy, O., and Clarkson, T. W. (1995c). Main neurodevelopmental study of Seychellois children following *in utero* exposure to methylmercury from a maternal fish diet: Outcome at six months. *Neurotoxicology* **16**(4), 653–664.

Myers, G. J., Davidson, P. W., Cox, C., Shamlaye, C. F., Tanner, M. A., Marsh, D. O., Cernichiari, E., Lapham, L. W., Berlin, M., and Clarkson, T. W. (1995d). Summary of the Seychelles Child Development Study on the relationship of fetal methylmercury exposure to neurodevelopment. *Neurotoxicology* **16**(4), 711–716.

Myers, G. J., Davidson, P. W., Shamlaye, C. F., Axtell, C. D., Cernichiari, E., Choisy, O., Choi, A., Cox, C., and Clarkson, T. W. (1997a). Effects of prenatal methylmercury exposure from a high fish diet on developmental milestones in the Seychelles Child Development Study. *Neurotoxicology* **18,** 819–830.

Myers, G. J., Davidson, P. W., Cox, C., Shamlaye, C. F., Choisy, O., Cernichiari, E., Choi, A., Sloane-Reeves, J., Axtell, C., Gao, P., and Clarkson, T. W. (1997b). The Seychelles Child Development Study: Results and new directions through twenty-ni[illegible] months. *Water Air Soil Pollut.* **97,** 53–61.

Nierenberg, D. W., Nordgren, R. E., Chang, M. B., Siegler, R. W., Blayney, M. B., Hochberg, F., Toribara, T. Y., Cernichiari, E., and Clarkson, T. W. (1998). Delayed cerebellar disease and death after accidental to dimethylmercury. *N. Engl. J. Med.* **338,** 1672-1676.

Phelps, R. W., Clarkson, T. W., Kershaw, T. G., and Wheatley, B. (1980). Interrelationships of blood and hair mercury concentrations in a North American population exposed to methylmercury. *Arch. Environ. Health* **35,** 161-168.

Rodier, P. M., Aschner, M., and Sager, P. R. (1984). Mitotic arrest in the developing CNS after prenatal exposure to methylmercury. *Neurobehav. Toxicol. Teratol.* **6,** 379-385.

Rustam, H., and Hamdi, T. (1974). Methyl mercury poisoning in Iraq. A neurologial study. *Brain* **97**(3), 500-510.

Rustam, H., Von Burg, R., Amin-Zaki, L., and Elhassani, S. (1975). Evidence for a neuromuscular disorder in methylmercury poisoning: Clinical and electrophysiological findings in moderate to severe cases. *Arch. Environ. Health* **30,** 190-195.

Sager, P. R., Aschner, M., and Rodier, P. M. (1984). Persistent, differential alterations in developing cerebellar cortex of male and female mice after methylmercury exposure. *Dev. Brain Res.* **12,** 1-11.

Shamlaye, C. F., Marsh, D. O., Myers, G. J., Cox, C., Davidson, P. W., Choisy, O., Cernichiari, E., Choi, A., Tanner, M., and Clarkson, T. W. (1995). The Seychelles Child Development Study on neurodevelopmental outcomes in children following *in utero* exposure to methylmercury from a maternal fish diet: Background and demographics. *Neurotoxicology* **16**(4), 597-612.

Shephard, D. A. E. (1976). Methyl mercury poisoning in Canada. *Can. Med. Assoc. J.* **114,** 110-114.

Smith, J. C., Farris, F. F., and Von Burg, R. (1976). Chemical type of mercury in patients in the outbreak of organomercury poisoning in Iraq. *Bull. WHO* **53,** 61-64.

Swedish Expert Group. (1971). Methylmercury in fish. A toxicological-epidemiological evaluation of risks. *Nord. Hyg. Tidskr.* **4**(Suppl.), 19-364.

Turner, M. D., Marsh, D. O., Rubio, C. E., Chiriboga, J., Chiriboga, C., Smith, J. C., and Clarkson, T. W. (1974). Methylmercury in populations eating large quantities of marine fish. I. Northern Peru. *In* "Proc. First International Mercury Conf., May 1974, Barcelona," Vol. II, pp. 229-234. Fabrica National de Moneda y Timbre, Madrid, Spain.

Turner, M. D., Marsh, D. O., Smith, J. C., Clarkson, T. W., Inglis, J. B., Rubino, E. C., Chiriboga, J., and Chiriboga, C. C. (1980). Methylmercury in populations eating large quantities of marine fish. *Arch. Environ. Health* **35,** 367-378.

Uauy-Dagach, R., and Valenzuela, A. (1996). Marine oils: The health benefits of n-3 fatty acids. *Nut. Rev.* **54**(110), S102-S108.

Von Burg, R., and Rustam, H. (1974a). Conduction velocities in methylmercury poisoned patients. *Bull. Environ. Contam. Toxicol.* **12**(1), 81-85.

Von Burg, R., and Rustam, H. (1974b). Electrophysiological investigations of methylmercury intoxication in humans. Evaluation of peripheral nerve by conduction velocity and electromyography. *Electroencephalogr. Clin. Neurophysiol.* **37**(4), 381-392.

Wheatley, B., Barbeau, A., Clarkson, T. W., and Lapham, L. (1979). Methylmercury poisoning in Canadian Indians—The elusive diagnosis. *Can J. Neurol. Sci.* **6**(4), 417-422.

World Health Organization. (1990). Environmental Health Criteria 101 Methylmercury. Geneva, Switzerland, pp. 1-144.

Mercury in Humans

Faroe Islands Studies

Drs. Richard Clapp and Philippe Grandjean
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Health effects of seafood contamination with methylmercury in the Faroes

Philippe Grandjean
University of Southern Denmark and Boston University

Neurotoxicity due to methylmercury is well documented from unfortunate poisoning incidents. However, the dose-effect relationships have been poorly documented, and the impact of imprecision and potential bias is unclear from past epidemiological studies. We first generated a cohort of 1022 consecutive singleton births during 1986-1987 in the Faroe Islands, where increased methylmercury exposure is mainly due to consumption of pilot whale meat. We measured total mercury concentrations in cord blood and maternal hair collected at parturition. Because prenatal neurotoxic effects would be permanent, assessment of neurobehavioral functions was postponed to the age of 7 years, where the children could undergo detailed testing. A total of 917 of the cohort children underwent thorough examinations. Significant exposure-related dysfunctions were seen in most neuropsychological tests and were most pronounced in the domains of language, attention, and memory. Mercury-associated effects were also seen in delayed latencies for evoked potentials and in blood pressure regulation. The associations remained significant after adjustment for covariates and also after exclusion of children with high maternal hair-mercury concentrations (corresponding to the benchmark dose) or high PCB exposures. Results from examinations conducted of the same children at age 14 years are currently underway. As expected, the cord-blood mercury concentration was the best risk indicator. However, statistical analyses suggested that even this parameter was associated with an error variance that substantially exceeded the analytical imprecision, although it was much less than that associated with the maternal hair-mercury concentration. Such imprecision leads to an underestimation of the true mercury effects.

A second Faroese cohort of 182 singleton term births was generated in 1994 with more detailed exposure documentation. In this cohort, we have documented mercury-associated decreases in the neonatal Neurological Optimality Score and in postnatal growth. Detailed statistical analyses of both prospective cohort studies have failed to identify any covariates that could account for the mercury-associated effects, which remained robust when using different analytical strategies. Although exposure misclassification may be more likely in cross-sectional studies, we have also seen adverse effects in studies of mercury-exposed children from Brazil and Madeira, where developmental exposure levels were determined from current hair-mercury concentrations. In conclusion, we have obtained evidence of subtle adverse effects on neurobehavioral functions, blood pressure, and growth. At age 7 years, a doubling of the mercury exposure corresponds to a developmental delay of up to 2 months. Although IQ tests were not done, such delays would be comparable to a loss of about 1.5 IQ points. These dysfunctions are detectable at exposure levels prevalent in many parts of the world where contaminated seafood or freshwater fish constitutes an important part of the diet.

Richard W. Clapp
Associate Professor of Public Health
Boston University School of Public Health

Education

Dartmouth College, A.B. degree in Biology, 1967
Harvard School of Public Health, M.P.H. degree in Health Services, 1974
Boston University School of Public Health, Sc.D. in Epidemiology, 1989

Recent Professional Experience

1977-1978	Director, Childhood Lead Poisoning Prevention, Massachusetts Dept. of Public Health, Boston, MA
1979-1980	Acting Director, Occupational and Environmental Health Studies, Equifax Health Systems Division, Reading, MA
1980-1989	Director, Massachusetts Cancer Registry, Massachusetts Department of Public Health, Boston, MA
1989-1994	Director, Center for Environmental Health Studies, John Snow, Inc., Boston, MA
1994-present	Consultant, John Snow, Inc., Boston, MA
1992-1995	Assistant Professor of Public Health, B.U. School of Public Health
1995-present	Associate Professor of Public Health, B.U. School of Public Health
2002	Senior Environmental Health Scientist, Tellus Institute, Boston, MA

Teaching Appointments

1989-1995	Assistant Clinical Professor, Tufts University School of Medicine,
1989-1996	Boston, MA
1990-1993	Adjunct Assistant Professor, Boston University School of Public
Health,	
	Boston, MA
1993-1995	Assistant Professor, B.U. School of Public Health, Boston, MA
1995-present	Associate Professor, B.U. School of Public Health, Boston, MA

Recent Publications

Geller A, Miller D, Lew R, Clapp R. Cutaneous Melanoma: Another Cancer with Mortality Disproportionately Affecting the Socioeconomically Disadvantaged. Am J Public Health 86:538-43, 1996.

Baron JA, Newcomb PA, Longnecker MP, Mittendorf R, Storer BE, Clapp RW, Bogdon G, Yuen J. Cigarette Smoking and Breast Cancer. Cancer Epi Biomarkers Prev 5:399-403, 1996.

Coogan PF, Clapp RW, Newcomb PA, Wenzl TB, Bogdan G, Baron JA, Longnecker MP. Occupational Exposure to 60 Hz Magnetic Fields and Risk of Breast Cancer in Women. Epidemiology 7:459-64, 1996.

Coogan PF, Clapp RW, Newcomb PA, Mittendorf R, Bodgan G, Longnecker MP. Variation in Female Breast Cancer Risk by Occupation. Am J Ind Med 30:430-37, 1996.

Newcomb PA, Longnecker MP, Storer BE, Mittendorf R, Baron J, Clapp RW, Trentham-Dietz A, Willett WC. Recent Oral Contraceptive Use and Breast Cancer (submitted) Cancer Causes and Control 7:525-32, 1996.

Coogan PF, Clapp RW, Newcomb PA, Baron J, Longnecker MP, Trentham-Dietz A. Physical Activity in Usual Occupation and Risk of Breast Cancer. Cancer Causes and Control 8:626-31, 1997.

Colton T and Clapp RW. Proportional Mortality Studies. In Encyclopedia of Biostatistics, Armitage P and Colton T, eds. Volume 5, pp. 3556-8. John Wiley and Sons, London, 1998.

Clapp RW. The Decline in U.S. Cancer Mortality from 1991 to 1995. Int J Health Services 28:747-55, 1998.

Ozonoff DM and Clapp RW. Cancer Survival is No Lottery. Lancet 353:1379-80, 1999.

Fikfak M, Clapp RW, Kriebel D. Health Risks of Decommissioning Nuclear Facilities. New Solutions 9(2):153-161, 1999.

Chie W-C, Hsieh C-C, Newcomb PA, Longnecker MP, Mittendorf R, Greenberg ER, Clapp RW, et al. Age at Any Full-term Pregnancy and Risk of Breast Cancer. Am J Epidemiol 151:714-22, 2000

Clapp RW and Coogan PF. Leukemia in Petroleum Refinery Workers: A Review of Recent Studies. New Solutions 9(4):375-87, 1999.

Clapp RW and Ozonoff DM. Where the Boys Aren't: Dioxin and the Sex Ratio. Lancet 355:1838-39, 2000.

Clapp RW. Environment and health: 4. Cancer. Can Med Assoc J 163(8):1009-1012, 2000.

Philippe Grandjean, M.D., D.M.Sc.
Professor of Environmental Medicine
University of Southern Denmark, Odense, Denmark

Dr. Grandjean is Professor of Environmental Medicine at the University of Southern Denmark, Odense, Denmark. He is also an Adjunct Professor of Environmental Health and Neurology at Boston University Schools of Medicine and Public Health. He received his academic degrees from the University of Copenhagen, Denmark. Following university fellowships and a two-year Fulbright senior research scholarship at Mount Sinai School of Medicine in New York, he became the first Director of the Department of Occupational Medicine at the Danish National Institute of Occupational Health in 1980. After moving to Odense University as full Professor in 1982, he also became the Consultant in Toxicology for the National Board of Health, Denmark. He serves on editorial boards of ten scientific journals, was European Editor of Archives of Environmental Health during 1986-92, and recently became Editor-in-Chief of the new web-based journal Environmental Health. He has previously served as Vice-Dean of the medical faculty in Odense, as institute chairman and member of the Danish Medical Research Council. Internationally, he has served on or chaired several committees under the auspices of WHO, IARC, EC, IUPAC, and other organizations and served on the Endocrine Disruptor Screening Program Subcommittee of the U.S.EPA. The research on marine contaminants and their effects on child development was initiated with Dr. Pal Weihe in the mid-1980s, and the Faroese birth cohort studies have since then given rise to several international cooperative projects. Of the 300+ scientific publications almost half are published in international journals with peer review.

Presentation in Mobile, 20 May 2002
by Philippe Grandjean, MD
(pgrand@bu.edu)

Important (and difficult) questions:

- What kinds of effects are caused by (methyl)mercury?
- How do we assess exposure levels?
- How do we determine the adverse effects?
- How is a safe exposure level determined?
- Is this pollution really a problem?
- Do we really know what we need to know?

Why MeHg research in the Faroes?

- Exposure to MeHg from pilot whale meat is like a natural experiment - highest level 1000x the lowest
- Exposure only weakly associated with confounders
- Homogeneous, western culture
- High participation rate (88% at 14 yrs)

Faroese cohort studies

- 1. Mercury cohort (N= 1022): born 1986-1987
- 2. PCB cohort (N = 182) born 1994
- 3. POP Cohort (N ~ 650) born 1999-2000

Exposure biomarkers (Hg concentrations)

- Cord blood
- Maternal hair at parturition (c. 9 cm)
- Same, first 2-cm segment
- Child hair at 12 months
- Child blood and hair, 7 years
- Child blood and hair, 14 years

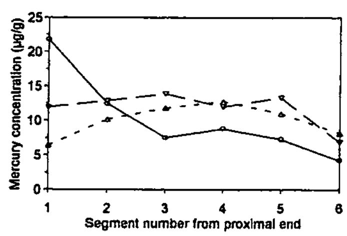
Validity of exposure biomarkers

- Analytical imprecision and accuracy
- Sample characteristics
- Timing of sampling
- Toxicokinetic patterns (T½ $\geq$45d)
- Developmental vulnerability of brain functions
- Predictive validity

Sources of variation (hair-Hg)

- Variation in hair weight (100 mg of hair may include 500 to 2,000 strands each 2.5 cm)
- Variation with hair color (in gray-haired subjects, concentration lower in white hair than in pigmented)
- Variation with hair growth rate and hair structure (unknown, studies of non-Caucasians needed)

Mercury in 1.5-cm segments from 3 samples with CV > 25%



Statistical error analysis

- Total measurement error may be assessed in a factor analysis model
- Linear functional model is assumed
- Only two prenatal exposure variables available: cord blood and maternal hair
- Addition of questionnaire response on pilot whale meat dinners as third variable
- Total error for all variables may be calculated

Total measurement error

Indicator*	Loading	Variance	CV
Blood	1.0000	0.0175	30%
Hair	0.8395	0.0515	52%
Whale	0.8820	0.2622	>100%

*log transformed

Conclusions on exposure assessment

- Hair is excellent for monitoring
- Developmental toxicity risk best determined by Hg in cord blood
- The less precise the exposure assessment, the more the effects are underestimated
- Prenatal exposure is most relevant
- Postnatal exposure also a concern

Considerations on assessing developmental neurotoxicity

- Neurotoxicity may not be immediately apparent
- Nervous system must mature to express relevant functions
- Participation in clinical tests difficult at preschool age
- Reversibility or compensation

Criteria for clinical tests

- Sensitive to toxic exposures
- Reflecting functional domains
- Reasonably specific, with limited potentials for confounding
- Appropriate for age and culture
- Highly skilled professional examiners
- Computer-assisted methods

Number of responses and p for association with mercury

Tests reflecting attention		
Catching a ball	3	0.74
Digit span score	10	0.02
Reaction time	312	<0.001

Examiner effect on WISC-R Similarities score (beta for mercury and p)

Neuropsychologist	(N=282)	**-1.53**	**0.043**
Technician	(N=578)	0.24	0.59
Both*	(N=860)	-0.05	0.90

*adjusted for examiner effect

Conclusions on neurobehavioral effects

- Mainly involve attention, memory and language, but also visuospatial and motor functions
- Prenatal more toxic than early postnatal exposure (except for visuospatial?)
- Subtle effects may be difficult to demonstrate
- Concerns about residual confounding

P values for PCB (and Hg) after adjustment for Hg

Test*	PCB	Hg
CPT-reaction time	0.67	0.002
Boston Naming	0.27	0.06
CVLT long delay	0.64	0.08

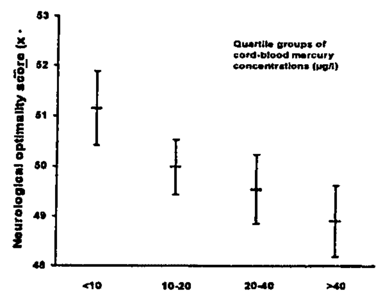
* where PCB (alone) is significant

Betas for PCB in Hg exposure tertile groups

Test*	I	II	III
CPT-reaction time	-6.6	2.8	38.4
Boston Naming	-1.0	-0.6	-1.6
CVLT long delay	-0.2	-0.1	-0.9

* where PCB (alone) is significant

Neonatal neurological optimality (Cohort 2)



Methylmercury toxicity in current perspective

- Neurotoxic effects at low exposure levels - like lead?
- Cardiovascular effects – possibly due to neurotoxicity?
- Effects on postnatal growth - indicate systemic toxicity?

<u>Over</u>estimation of a mercury effect has been stressed

- Association with other neurotoxic seafood toxicant(s)
- Other residual confounding (residence, transportation)
- Failure to adjust for multiple comparisons

Underestimation of a mercury effect needs consideration

- Imprecise exposure assessment
- Protective nutrients in seafood
- Imprecise outcome measures
  - Psychometric test properties
  - Test administration
- Overadjustment for confounders?
- Two-sided p-values and limited power

An important health risk?

- Adverse effects are unlikely to be apparent from national health statistics
- Loss of brain function has social and economic consequences
- Increased blood pressure in childhood predicts risk in adult life
- True significance may yet be unclear

Mercury effect as delay in development (months, age 7) for each doubling of exposure

Motor (Finger tapping, PH)	0.9
Attention (CPT-reaction time)	1.3
Visuospatial (Bender errors)	0.6
Language (Boston Naming)	1.6
Verbal memory (CVLT short delay)	2.0

(~10% of s.d. or ~~1.5 IQ points)

Mercury in Humans

EPA Fish Consumption Advisories

Mr. Joel Hansel
U.S. Environmental Protection Agency – Region 4

The states have primary responsibility for protecting their residents from the health risks of consuming contaminated non-commercially caught fish. They do this by issuing consumption advisories for the general population, including recreational and subsistence fishers, as well as for sensitive subpopulations (such as pregnant women/fetus, nursing mothers and their infants, and children). These advisories inform the public that high concentrations of chemical contaminants, such as mercury, have been found in local fish. The advisories recommend either limiting or avoiding consumption of certain fish from specific waterbodies or, in some cases, from specific waterbody types (such as lakes or rivers).

As of December 2000, mercury was the chemical contaminant responsible, at least in part, for the issuance of 2,242 fish consumption advisories by 41 states. Almost 79% of all advisories issued in the United States are at least partly due to mercury contamination in fish and shellfish. Advisories for mercury have increased steadily, by 149% from 899 advisories in 1993 to 2,242 advisories in 2000. The number of states that have issued mercury advisories also has risen steadily from 27 states in 1993 to 41 states in 2000. Advisories for mercury increased nearly 8% from 1999 (2,073 advisories) to 2000 (2,242 advisories).

Thirteen states have issued statewide advisories for mercury in their freshwater lakes and/or rivers: Connecticut, Kentucky, Indiana, Maine, Massachusetts, Michigan, Minnesota, New Hampshire, New Jersey, North Carolina, Ohio, Vermont and Wisconsin. Another ten states (Alabama, Florida, Georgia, Louisiana, Maine, Mississippi, North Carolina, South Carolina and Texas) have statewide mercury advisories in effect for their coastal marine waters.

On January 12, 2001, EPA and FDA jointly issued a press release notifying the public of a national fish consumption advisory due to mercury contamination. EPA's advice is that if you are pregnant or could become pregnant, are nursing a baby, or if you are feeding a young child, limit consumption of freshwater fish caught by family and friends to one meal per week. For adults one meal is six ounces of cooked fish or eight ounces uncooked fish; for a young child one meal is two ounces cooked fish or three ounces uncooked fish. Many states collect data on mercury levels in fish from local waters. Check with your state or local health department for specific advice on waters where your family and friends are fishing.

In addition, the Food and Drug Administration (FDA) has issued advice on mercury in fish bought from stores and restaurants, which includes ocean and coastal fish as well as other types of commercial fish. FDA advises that women who are pregnant or could become pregnant, nursing mothers and young children not eat shark, swordfish, king mackerel, or tilefish. FDA also advises that women of childbearing age and pregnant women may eat an average of 12 ounces of fish purchased in stores and restaurants each week. Therefore, if in a given week you eat 12 ounces of cooked fish from a store or restaurant, then do not eat fish caught by your family or friends that week. This is important to keep the total level of methylmercury contributed by all fish at a low level in your body.

EPA recommends that women who are or could become pregnant, nursing mothers and young children follow the FDA advice for coastal and ocean fish caught by family and friends. Check with your local or state health department for specific advice.

As noted earlier, States have primary responsibility for protecting their residents from the health risks of consuming contaminated non-commercially caught fish. Distribution of these state fish consumption advisories/guidelines is typically done through one of three routes. First, this information is contained within most state fishing regulations that are distributed at the time that an angler purchases a fishing license. Second, signs may be posted at common public access points to inform anglers of the chemical contaminant and the species which are affected. Third, the public can request such information directly from the appropriate resource management agency. This information is also compiled in a national database of fish and wildlife advisories which can be found at www.epa.gov/waterscience/fish.

When EPA issues an advisory as it did related to mercury in January, 2001, the information was directly distributed to media outlets throughout the Nation. Also, EPA, along with ATSDR, has initiated an effort to inform medical professionals about the danger posed to their patients by consuming contaminated fish. All of this information can be found at the EPA website noted above.

Mr. Joel Hansel
U.S. EPA Region 4

Mr. Hansel serves at the EPA Region 4 Fish Advisory Coordinator, Regional BEACH Coordinator, Regional Human Health Criteria Expert, and Water Quality Standards Coordinator. He also serves on the EPA Gulf of Mexico Program's Public Health Focus Team and as chairman of the Laboratory Quality Assurance and Education Committees for the Interstate Shellfish Sanitation Conference. Mr. Hansel has a B.S. in Biology with subspecialties in Human Physiology and Microbiology from the University of Akron in Akron, Ohio and is currently completing a Master's of Public Health in Epidemiology and Environmental/Occupation Health from the Rollins School of Public Health at Emory University in Atlanta, Georgia.

USEPA's

National Fish and Wildlife Contamination Program

Joel Hansel, Regional Fish Advisory Coordinator
Standards, Monitoring, and TMDL Branch
Water Management Division
United States Environmental Protection Agency, Region 4

USEPA's Fish Contamination Program

- Provides technical assistance to State, Federal and Tribal agencies on matters related to health risks associated with exposure to chemical contaminants in fish and wildlife
- Activities include:
  - National guidance documents
  - National databases
  - National conferences and workshops
  - Grants for sampling and analysis
  - Conduct special studies
  - Issue advisories

EPA

Historical Perspectives and Rationale

- Contamination of aquatic resources has been documented for many regions of the United States
- While concentrations of some pollutants have decreased, others have increased due to increased urbanization, industrial development, and agricultural activities
- Toxic contaminants can accumulate in fish and shellfish tissues and may reach concentrations much higher than in the water column

EPA

Historical Perspectives and Rationale, cont'd.

- **Monitoring of contaminants in fish tissue provides an indicator of water quality problems and contaminated sediments**
- **Monitoring can indicate whether fish contaminant concentrations pose an unacceptable risk to humans consuming fish**
- **Fish advisories or bans can be issued based on the results of the monitoring of fish contaminants**

EPA

2000 Advisory Listing

The 2000 NLFWA database lists 2,838 advisories in 48 states, the District of Columbia, and the US Territory of American Samoa. The number of waterbodies under advisory represents:

- **23% of the Nation's total lake acres or over 63,288 lakes**
- **9.3% of the Nation's total river miles or over 325,500 river miles**
- **71% of the Nation's contiguous coastal waters including 92% of the Atlantic coast and 100% of the Gulf coast**
- **100% of the Great Lakes and their connecting waters**

There were 23 states with statewide advisories in effect, including new statewide advisories for Kentucky (lakes and rivers), Wisconsin (lakes), and Georgia, South Carolina and North Carolina (Coastal).

Source: NLFWA December 2000

EPA

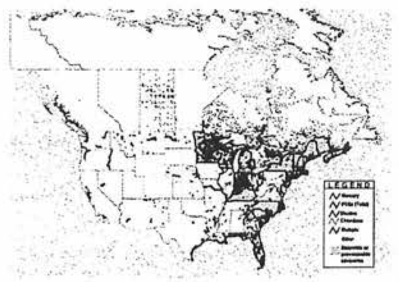
Bioaccumulative pollutants

- Although current advisories in the United States have been issued for 36 different pollutants, most advisories involve five primary bioaccumulative contaminants:
  - Mercury = 2,242 advisories in 40 statesactive in 2000 (up 8% from 1999. up 149% from 1993).
  - PCBs = 726 advisories active in 2000 in 38 states (up 3% from 1999. up 128% from 1993).
  - Chlordane = 99 advisories active in 2000 (unchanged from 1999).
  - Dioxins = 76 advisories active in 2000 (up 3% from 1999. after a 25% increase from 1998 to 1999).
  - DDT and metabolites = 44 advisories active in 1999 (up 4 from 1999).
- The increase in advisories issued by the states generally reflects an increase in the number of assessments of contaminants in fish and wildlife tissues.

Source: NLFWA December 2000

EPA

Location of Waterbodies Under Consumption Advisory



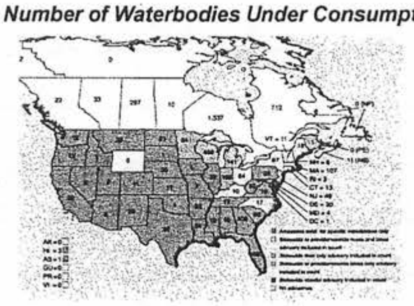
- Current advisories issued for:
  - Mercury
  - PCBs
  - Chlordane
  - Dioxins
  - 36 other chemicals.
- Advisories are in effect in:
  - 63,288 lakes, representing 23% of our Nation's lake acres
  - 9.3% of our Nation's rivers
  - 100% of the Great Lakes
  - 71% of coastline of contiguous 48 states.
- Number of advisories in the US in 2000 represents a 7% increase over 1999; 124% increase since 1993.
- 48 states, the District of Columbia, and 1 US territory and 8 Canadian provinces and 1 Canadian territory have fish advisories.
- 48 states and 1 US territory and 7 Canadian provinces and 1 Canadian territory have waterbody-specific advisories.
- 14 states and the District of Columbia have statewide advisories and 2 Canadian provinces have provincewide advisories for lakes and/or rivers.
- 15 states have all of their marine coastal waters under advisory.

NOTE: This map depicts waterbodies where fish consumption advisories were in effect in 2000 based on information provided to the USEPA by the states in December 2000 and Canadian provinces in December 1997. Because only selected waterbodies are monitored, this map may not reflect the full extent of chemical contamination of fish tissue

Source: NLFWA December 2000

EPA

Number of Waterbodies Under Consumption Advisory



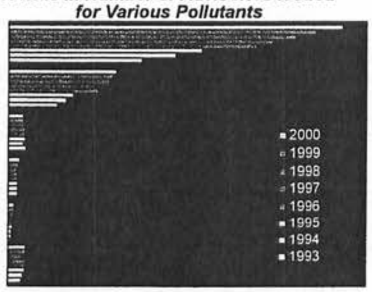
- Advisories are designed to reduce health risks associated with consumption of chemically contaminated fish and wildlife.
- Most states are developing advisories using a risk-based approach designed for noncommercial harvesting of fish and wildlife; some, however, still use the FDA action level approach designed for commercial fisheries.
- State and province advisory data should not be used for:
  - Characterizing geographic distribution of chemical contaminants
  - Making interstate or international comparisons.
- Advisory database is updated annually by USEPA's Fish and Wildlife Contamination Program.
- Learn more about USEPA's Fish and Wildlife Contamination Program at www.epa.gov/ost/fish
-

NOTE: This map depicts the number, by state or province, of waterbodies where fish consumption advisories were in effect in 1999 based on information provided to the USEPA by the states in December 1999 and by the Canadian provinces in December 1997. Because only selected waterbodies are monitored, this map does not reflect the full extent of chemical contamination of fish tissues in each state or province.

Source: NLFWA December 2000

EPA

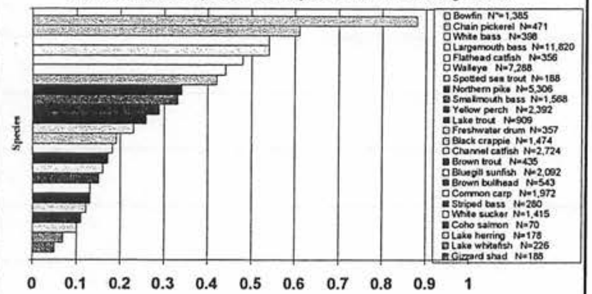
Trends in Number of Advisories Issued for Various Pollutants



Source: NLFWA December 2000

EPA

Mean Mercury Concentration in Tissues of Selected Fish Species Sampled from Advisory Sites



Source: NLFWA February 2002, data from 1987-2001

EPA

Fish Advisories Due to Mercury

EPA

Guidance for Assessing Chemical Contamination Data for Use in Fish Advisories

Volume 1: Fish Sampling and Analysis

Volume 2: Risk Assessment and Fish Consumption Limits

Volume 3: Overview of Risk Management

Volume 4: Risk Communication

EPA

Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories

Volume 1
Fish Sampling and Analysis
Second Edition

FISH AND HUMAN HEALTH

EPA

Volume 1-Fish Sampling and Analysis

Purpose and Objectives

- To provide guidance on assessing chemical contaminants in fish and shellfish
  - sampling methods
  - chemical analysis
  - statistical design
  - monitoring strategy
  - quality assurance/quality control

EPA

Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories

Volume 2
Risk Assessment and Fish Consumption Limits
Second Edition

FISH AND HUMAN HEALTH

EPA

Volume 2-Risk Assessment and Fish Consumption Limits

Purpose and Objectives

- **Provide guidance on the development of risk-based fish consumption limits.**
- **Describe EPA's four-step risk assessment process for consumption of fish and shellfish.**
- **Provide toxicological profiles for 25 chemicals of concern**

EPA

Risk Assessment Methods

EPA

Risk Assessment Methods

- **Risk assessment can be divided into four main steps:**
  - **Hazard Identification**
  - **Dose - Response Assessment**
  - **Exposure Assessment**
  - **Risk Characterization**

EPA

Development and Use of Risk-Based Consumption Limits

EPA

Risk-Based Fish Consumption Limits

- Definition:
  - Risk-based fish consumption limits are intended to provide guidance on the maximum number of meals of fish from a defined area that can be eaten, over a specified time period, by defined groups of consumers.
  - These limits are intended to protect human health by limiting exposure to chemical contaminants in fish tissue

EPA

Daily Consumption limits for NonCarcinogens

$$CR_{lim}\ \text{(kg/day)} = \frac{RfD \times BW}{C_m} \qquad \text{(Eq. 3-3)}$$

- CR_{lim} = Maximum allowable daily fish consumption rate (kg/day
- RfD = Reference dose (mg/kg-day)
- BW = Consumer body weight (kg)
- C_m = Measured concentration of chemical contaminant "m" in a given species of fish (mg/kg)

EPA

Meal Consumption Limits

$$CR_{mm}\text{ (meals/month)} = \frac{CR_{lim} \times T_{ap}}{MS} \quad \text{(Eq. 3-2)}$$

- CR_{mm} = Maximum allowable fish consumption rate (meals/month)
- CR_{lim} = Maximum allowable daily fish consumption rate (kg/day)
- T_{ap} = Time averaging period (365.25 days/12 months = 30.44 days/month)
- MS = Meal size (kg fish/meal)

EPA

Mercury Table

Consumption Limit	Noncancer Health Endpoint
Fish Meals/Month	Fish Tissue Conc. (ppm,ww)
Unrestricted (>16)	0 – 0.029
16	>0.029 – 0.059
12	>0.059 – 0.078
8	>0.078 – 0.12
4	>0.12 – 0.23
3	>0.23 – 0.31
2	>0.31 – 0.47
1	>0.47 – 0.94
0.5	>0.94 – 1.9
None (<0.5)	>1.9

Assumed meal size equals 8oz, RfD = 1x10-4 mg/kg-d, and 70 kg body weight.

EPA

EPA's National Mercury Advisory

If you are pregnant or could become pregnant, are nursing a baby, or if you are feeding a young child, limit consumption of freshwater fish caught by family and friends to one meal per week. For adults one meal is six ounces of cooked fish or eight ounces uncooked fish; for a young child one meal is two ounces cooked fish or three ounces uncooked fish. Many states collect data on mercury levels in fish from local waters. EPA recommends that women who are or could become pregnant, nursing mothers and young children follow the FDA advice for coastal and ocean fish caught by family and friends. Check with your state or local health department for specific advice on waters where your family and friends are fishing.

EPA

FDA's National Advisory

The Food and Drug Administration (FDA) has issued advice on mercury in fish bought from stores and restaurants, which includes ocean and coastal fish as well as other types of commercial fish. FDA advises that women who are pregnant or could become pregnant, nursing mothers and young children not eat shark, swordfish, king mackerel, or tilefish. FDA also advises that women of childbearing age and pregnant women may eat an average of 12 ounces of fish purchased in stores and restaurants each week.

EPA

FISH AND HUMAN HEALTH

EPA

Mercury in Humans

Development of Methylmercury Reference Dose
Dr. Kathryn Mahaffey
Office of Prevention, Pesticides and Toxic Substances
U.S. Environmental Protection Agency

Methylmercury: Current Understanding of Health Risks and US Exposures

Methylmercury is widely recognized as a neurotoxin affecting adults, children, and the developing fetus. Until the mid-1990s, peripheral neuropathy was considered to be the critical effect for methylmercury among adults. Paresthesias formed the basis of multiple organizations' evaluations of adverse effects; i.e., EPA, Food and Drug Administration, and the World Health Organization. Data from poisoning episodes during the 1960s and 1970s had shown that neurological problems among children exposed *in utero* to methylmercury whose mothers themselves demonstrated no or minimal symptoms. The vulnerability of the developing nervous system to a myriad of adverse effects following methylmercury exposures has been described in a broad array of studies in rodents, nonhuman primates, and humans. In 1995 EPA revised it's Reference Dose for methylmercury to be based on fetal protection.

- Between 1995 and present, there have been varied estimates of the methylmercury exposure level likely to be without adverse effects in populations including sensitive groups. The Committee on Toxicology of Methylmercury (organized under the auspices of the United States National Academy of Sciences) issued a report in July of 2000 which recommended the following: Neurodevelopmental effects observed in the Faroe Islands cohort study form the basis of US EPA's Reference Dose (RfD) for methylmercury.
- The preferred Benchmark Dose Lower Bound (BMDL) was 58µug/L of cord blood.
- An uncertainty factor (UF) of not less than 10 would be used in setting a RfD.

In the 2001 revision of US EPA's RfD for methylmercury, the BMDLs are based data showing adverse effects of methylmercury exposure on multiple tests of child development. The RfD is based on data from the Faroese cohort, with supporting analyses from the New Zealand study, and the integrative analysis of the two preceding studies and the Seychelle Islands study. The UF was 10 and the Modifying Factor (MF) was 1. The BMDL exposure parameters selected are associated with a doubling of the number of children with scores in a range considered clinically subnormal (i.e., the lowest 5% of the distribution) on multiple tests of neurobehavioral function. Multiple endpoints yielded BMDLs in the range of 32 to 79 µg/L in maternal blood for different neuropsychological effects in the offspring at 7 years-of-age corresponding to a range of daily maternal intakes of 0.596 to 1.472 µg/kg. The RfD remains 0.1 µg/kgbw/day associated with a cord blood of approximately 6 µg/L. This is not a "no observed adverse effect level". Within the Faroese cohort's data, effects at exposures less than those associated with a maternal hair mercury concentration of less than 10 ppm have been reported raising questions about whether or not a threshold for methylmercury's effects exists.

Blood mercury data from the first year of the fourth National Health and Nutrition Examination Survey (NHANES IV-99) indicated that the 90^{th} percentile value for women ages16 through 49 years was 6.2 (95% CI 4.7 - 7.9) µg/L. These data are from a survey that is intended to be representative of the United States population as a whole. The 1999 data indicated that approximately 10% of adult women of child-bearing age had blood mercury levels above a level that US EPA considers protective from adverse effects of methylmercury on children's neurological development. Data from various locations in the United States indicate that more elevated exposures exist in some geographic areas. Case reports of blood mercury concentrations considerably in excess of the Reference Dose have been reported from Wisconsin and Massachusetts. The prevalence of these more serious elevations in blood mercury concentrations has not yet been determined.

Kathryn R. Mahaffey, Ph.D.
Director of the Division of Exposure Assessment, Coordination and Policy
Office of Science Coordination and Policy of OPPTS, US EPA

Dr. Mahaffey's professional career is in exposure assessment and toxicology of metals. She has worked extensively in the area of food safety. Following graduate training in nutritional biochemistry and physiology at Rutgers University, she completed post-doctoral training in neuro-endocrinology at the University of North Carolina School of Medicine. Her research has been on susceptibility to lead toxicity with greatest focus on age and nutritional factors resulting in more than 100 publications in this area. During her long career with the United States Government she has been influential in lowering lead exposures for the United States population through actions to removal lead from foods and beverages, and from gasoline additives during the 1970s and 1980s.

In the past decade, Dr. Mahaffey has been actively involved in risk assessments for mercury. She was the author of the NIH Report to Congress on Mercury, and a primary author of US EPA's *Mercury Study Report to Congress*. These reports emphasized risk of developmental deficits caused by methylmercury exposure during development of the nervous system. Dr. Mahaffey was one of the primary developers of US EPA's *Mercury Research Strategy* which was released in late 2000. Along with other team members, she was responsible for the 2001 EPA/FDA national advisory on fish consumption. Dr. Mahaffey was one of a group of three EPA health scientists who revised the basis for EPA's Reference Dose for Methylmercury which was used in developing the Methylmercury Water Quality Human Health Criterion. In 2002 she received EPA's Science Achievement Award in Health Sciences for this work. This is EPA's highest health sciences award and is presented in conjunction with the Society of Toxicology.

Currently Dr. Mahaffey is the Director of the Division of Exposure Assessment, Coordination and Policy within the Office of Science Coordination and Policy of OPPTS, US EPA. This division runs US EPA's Endocrine Disruptor Screening and Validation Program. Dr. Mahaffey remains active in research and developing US EPA's policies on methylmercury.

Mercury in Humans

Development of Consistent Mercury Advisories in the Gulf of Mexico

Dr. Frederick Kopfler
EPA Gulf of Mexico Program

From the first meeting of the Gulf of Mexico Program Public Health Committee in March, 1989, the members recognized that some toxic substances introduced into Gulf Coast waters, may bioaccumulate in the food chain and pose a hazard to consumers. They included this as one of the four public health issues that should be addressed by the Gulf of Mexico Program. The goal recommended by the Public Health Team was: Prevent illness by reducing exposure to toxic substances in seafood while maintaining the beneficial effects of seafood consumption.

The Team funded an assessment of the occurrence of toxic substances in the fishery resources of the Gulf and concluded that the only contaminant that appeared to occur in a pervasive manner was mercury. In 1999 the Gulf of Mexico Program Management Committee charged the Program Office to "? provide more detailed information on the occurrence of mercury in the fishery resources of the Gulf of Mexico." In January 2000, a report was produced which indicated that mercury occurred in the edible tissues of many fish taken from the Gulf waters and sometimes at levels of concern. It was also apparent that for some species there was little or no data available.

After the development of an issue paper by the Gulf Sates Marine Fisheries Commission which included several recommendations, the Gulf of Mexico Program Management Committee charged the Program Office to ? "develop a project team to work in cooperation with the Gulf Sates Marine Fisheries Commission in developing recommendations for consumption advisories, as needed, for mercury in marine fish from the Gulf of Mexico that are consistent in all five States bordering the Gulf."

The team is to contain members from the Gulf State Health Agencies; Other state agencies as appropriate; Gulf States Marine Fisheries Commission; US EPA; FDA; NMFS; Gulf of Mexico Fishery Management Council; the Gulf Business Coalition; the Coastal Conservation Association; the Southeast Fisheries Association; and the Gulf of Mexico Program Citizens Advisory Committee. The Team is to be formed and develop a work plan with appropriate activities and submit for review by the Management Committee and should complete its work and present a final report within one year.

Fred Kopfler
EPA-Gulf of Mexico Program

Fred Kopfler is a native of Louisiana. He received a BS in chemistry from Southeastern Louisiana University in Hammond and a Masters and PhD in Biochemistry and Food Science and Technology from Louisiana State University.

After a two year post-doctoral at the US Department of Agriculture's Protein Pioneering Laboratory in Philadelphia, PA, he worked at the US Public Health Laboratory at Dauphin Island, Alabama investigating the pesticide and trace metal contaminants in shellfish.

When the US EPA was formed, he became one of the charter employees and moved to Cincinnati, OH where he worked until 1989 on the health effects of chemical contaminants and disinfection by-products in drinking water.

In 1989 he joined the newly formed Gulf of Mexico Program with offices at Stennis Space Center in Mississippi. At the Gulf of Mexico Program he has worked on public health issues associated with the use of the Gulf's waters and its seafood products including chemical contaminants of seafood; sewage pollution of shellfish growing waters and recreational waters; and harmful algal blooms.

Mercury in the Environment

Historical Background of Mercury in the Environment

Mr. Charles Moore
South Carolina Department of Natural Resources

Mercury is a basic chemical element of our solar system. There is a fixed amount on earth that cannot be created or destroyed. Mercury cycles through the earth's biosphere; including the atmosphere, surface waters, aquatic sediments, soils, as well as all plant and animal life. Mercury emissions into the environment can be characterized by three sources: the natural release and cycling of geologically bound mercury, anthropogenic releases, and the re-emission of mercury to the atmosphere from that previously deposited. EPA estimates 50 to 75 percent of the mercury released annually comes from human activities. Of approximately 200,000 tons of mercury emitted to the atmosphere since 1890, about 95 percent resides in terrestrial soils, 3 percent in ocean surface waters and 2 percent in the atmosphere. Mercury is a known toxicant, affecting growth, reproductive success, and development of both plant and animal life. It is a neurotoxin that bioaccumulates through the food chain with its primary pathway to humans being through the consumption of fish.

The natural global bio-geochemical cycling of mercury involves the degassing of mercury from soils and surface waters, atmospheric transport, and the deposition of mercury back to the land and open water. It may then be either re-volatized into the atmosphere or converted to insoluble mercury sulfide, that is absorbed to the soil, or bio-converted into more volatile or soluble forms that re-enter the atmosphere or are bioaccumulated in aquatic and terrestrial food chains.

Mercury occurs in three oxidation states. Metallic or elementary mercury has no charge and quickly vaporizes from its liquid form. Over 50% to 95% of the mercury found in the atmosphere is gaseous mercury (HgO) that has a residence time in the atmosphere of between 6 days and 2 years. During this time it is transported great distances, circulating globally. Elementary mercury is not very soluble and atmospheric water (rain and snow) does not serve as a significant means of transfer. Elemental mercury in the atmosphere is oxidized by ozone, hydrogen peroxide, hypochlorite or organoperoxide compounds.

Reactive gaseous mercury (mercuric, with a double electric charge, and mercurous, with a single positive charge) occur at much lower levels than the elementary form representing approximately 3% of the total gaseous mercury in the air. These forms are water-soluble and are removed from the air by gravity (dry deposition), and by rain, snow, dew and humidity (wet deposition). They have an atmospheric residence time of hours to days. Mercury adsorbed onto organic and inorganic microparticulates, may range from less than one percent to 40% of the total ambient mercury level in industrialized areas. Particulate forms are effectively removed by rain and have a relatively short residence time in the atmosphere.

Man's activities that release mercury into the environment are a complex combination of (a) activities that directly emit or inject mercury into the air, soil or water and (b) industrial utilization in products that eventually may be returned to the environment though landfills, combustion, or other means. Estimates of the annual total global input to the atmosphere from all sources including natural, anthropogenic, and oceanic emissions is about 5,500 – 6,000 tons with US sources estimated to have contributed about 3 percent. Mercury today is utilized in the electrical industry (switches, thermostats, batteries etc.), dentistry (dental amalgams, which are 50% mercury), medicinal products including antiseptics (mercurochrome), laxatives, worming medications, teething powers, pharmaceutical preservative products (thimerosal), a red tattoo dye, measuring devices, (thermometers), numerous industrial processes (the production of chlorine and caustic soda), in nuclear reactors, as an anti-fungal agent (wood processing), a solvent for reactive and precious metal, a coloring agent for paint as well as numerous other uses.

Global production of mercury, primarily from cinnabar (mercuric sulfide) mines, has declined 38% from 5,356 tons in 1990 to 3,337 tons in 1996. U.S. mercury production has declined from more than 2,000 metric tons per year in the 1970's, to less than 500 metric tons in 1996, most resulting from secondary sources and industrial recovery. Although the domestic use of mercury has shown a downward trend since the early 1970s mercury imports (277 metric tons in 1995) have escalated in recent years as a result of the suspension of mercury sales from the National Defense Stockpile in 1994, which had formerly been a major supplier of mercury to the domestic market.

Of the estimated 158 tons of mercury emitted annually into the atmosphere by human activities in the United States, approximately 87 percent is from combustion point sources, 10 percent from manufacturing, and 3 percent from all other sources. Of this total, about one-third (52 tons) is deposited within the lower 48 states and two-thirds (107 tons) is transported outside of U.S. borders. An additional 35 tons is deposited within US borders from the global reservoir for a total annual mercury deposition of 87 tons. Four specific source categories (all high temperature waste combustion of fossil fuel processes) account for approximately 80 percent of total mercury emissions in the U.S.: coal-fired utility boilers (33 percent), municipal combustion (19 percent), commercial /industrial boilers (18 percent) and medical waste incinerators (10 percent). In 1994, electric power plants built during the 1940's to 1970's emitted an estimated total of 91,422 pounds of mercury. The vast majority (95%) came from coal–burning plants, and most of that was from plants built prior to 1977 (77%).

According to EPA documents the amount of mercury in the atmosphere is estimated to have increased by 200 % to 500 % since the beginning of the industrial revolution. Others report that there is 3 to 6 times more mercury today vs. pre-industrial times in Atlantic Ocean water, Atlantic bird feathers, peat bogs, soils and lake sediments. Whereas mercury deposition rates have decreased in the vicinity of some localized sources in the western United States during the 1990s, measurements continue to increase in remote sites in northern Canada and Alaska indicating that the global atmospheric burden is continuing to increase.

The production and utilization of mercury are decreasing both on a worldwide and national level. However, based on past mercury releases, it may take decades and perhaps longer, before we observe measurable declines in the environment and affected biological systems. Increasing background levels of mercury increase the potential impact of emissions from local point sources to affected areas.

Charles J. Moore
Marine Biologist
South Carolina Department of Natural Resources, Charleston, SC

Charles Moore is a marine biologist with the South Carolina Department of Natural Resources in Charleston, South Carolina. After receiving a Masters Degree in Marine Biology from the University of Delaware in 1968, he spent 9 years studying estuarine fish populations in Chesapeake Bay for the Academy of Natural Sciences of Philadelphia.

During his 25-year career with the Maine Resources Division he managed marine recreational and commercial finfish resources. He served on advisory panels for the South Atlantic Fishery Management Council, Atlantic States Marine Fisheries Commission, and the National Maine Fisheries Service and is a representative of the International Game Fish Association. He was Conservationist of the year (1986) for the Coastal Conservation Association and for the South Carolina Wildlife Federation (Water Award) in 1998. Attached to the Office of Environmental Management, his recent efforts have been with understanding and communicating to the public the impact of mercury on the environment. His review of this subject lead to a symposium, Methylmercury (Impacts on Wildlife and Human Health), that he organized and moderated, April 9-10, 2001 in Charleston, South Carolina.

Historical Background Of Mercury In The Environment

Four Topics

- What are the sources of mercury entering environment?
- What quantities are being discharged?
- Importance of mercury's physical state at release.
- Evidence of environmental increases.

- •Basic element - solar system: sun, moon, meteorites.
- •Fixed amount - not created - destroyed.
- •Found throughout biosphere: atmosphere, surface waters, soils, sediments, all plant and animal life.
- •All classes of rocks - 10 to 50 ppb.
- •Cinnabar ore (mercury-sulfide) 86.6% mercury stable - insoluble.

MERCURY

Mercury – What Is It ?

- Heavy, silvery - white liquid.
- Only metal that is liquid at ambient temperature.
- Highest solubility in water of all metals.
- Easily vaporizes in air.
- Low viscosity - highly mobile droplets that amalgamate when they collide.
- Superconductivity observed in 1911.
- Environmental toxicant, affecting growth, reproductive success, and development in both plant and animal life.
- Neurotoxin - bioaccumulates through the food chain - primary pathway to man is consumption of fish.

Sources of Mercury in Today's Environment

- Natural releases of geological bound mercury.
- Anthropogenic (man's activities).
- Re-emission of mercury from above sources.

The Mercury Cycle

- Degassing of hg – rocks, soil, and water both by natural processes and mans activities.
- Movement of gaseous forms through atmosphere.
- Deposition of mercury on land and surface waters.
- Revolatilized to atmosphere or absorbed to soil or sediment particles.
- Converted to insoluble mercury sulfide and precipitated or bioconverted into more volatile or soluble forms that re-enter atmosphere or are bioaccumulation up the food chain(methylmercury).

Elemental Mercury

- Vaporizes from metallic mercury.
- Has no electrical charge.
- 50% to 90% of mercury found in atmosphere.
- Residence time in atmosphere – 6 days to 2 years.
- Insoluble – not effectively removed from atmosphere by rain or other precipitation.
- Distributed globally to even the most remote areas of earth.

Hg^{0} ⇄ (Oxidation / Reduction) Hg^{+1} or Hg^{+2} ⇄ (Methylation / Demethylation)

Reactive gaseous mercury

- Oxidized - chlorine/bromine oxides.
- Approximately 3% of Hg in atmosphere.
- Residence time – hours to days.
- Is water soluble.
- Attaches or absorbed to inorganic and organic micro-particulates.
- Removed from air by gravity (dry deposition) and by rain, snow, dew (wet deposition).
- Local or regional deposition.

The Mercury Cycle

1) Hg is constantly being cycled though the environment.
2) Hg in air is primarily elemental.
3) Hg in water, sediments and soil is primarily mercuric and mecurous inorganic salts
4) Hg in soils has a long retention time.
5) Regardless of source the same pathways.
6) Nearly impossible to separate contributions to biosphere from sources.

Annual global input to the atmosphere estimated to be 5,500 – 6,000 metric tons

Anthropogenic Sources of Mercury

- Direct
  - Air
    - Smelting of mercury ore / burning fossil fuel
  - Water
    - Gold mining
  - Soil
    - Fertilizers
- Indirect
  - Products containing mercury
    - Electrical wiring & switches
    - Medical and dental products
    - Pharmaceutical products

EPA Estimates

50% To 75% Of Mercury Releases To Atmosphere Are Result Of Human Activities.

Of The Estimated 200,000 Tons Of Mercury Emitted Since 1890:

1) About 95% Resides In Terrestrial Soils
2) About 3% Resides In Ocean Surface Waters
3) About 2% Resides In The Atmosphere

Uses Of Mercury

1500BC - mercury ore - China and Egypt

Today - electrical products - switches, wiring, thermostats, batteries, etc.

- dentistry - dental amalgams (50% mercury).
- medical products - antiseptics, laxatives, teething powers.
- measuring devices – thermometers.
- pharmaceutical preservatives – thimerosal.
- industrial processes - chlorine/ caustic soda.

The Materials Flow of Mercury in the Economies of the United States and the World

By John L. Sznopak and Thomas G.Goonan

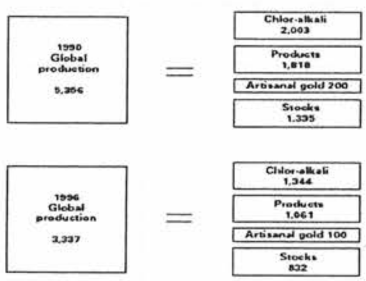
U.S. Geological survey Circular 1197

U.S. Department of the Interior

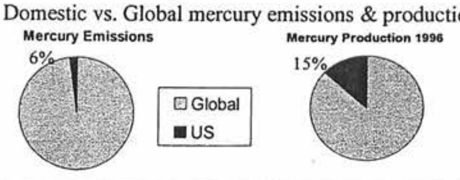
U.S. Geological Survey

June 14, 2000

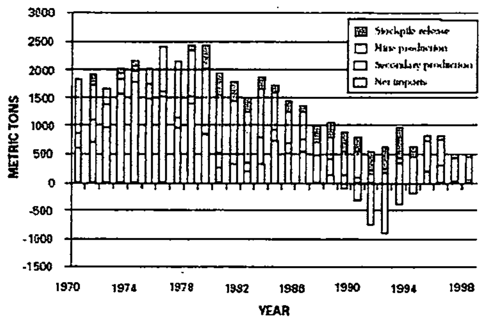
Global flow by Use, 1990 vs. 1996, Metric Tons



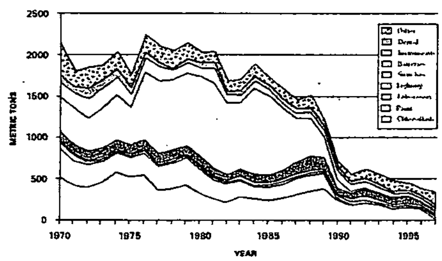
- Global mercury flow - concerns
  - Coal is worlds primary fuel, accounting for 37% of fuel used for electrical production and may be world's single greatest source of anthropogenic mercury to the atmosphere.
  - People's Republic of China is world's largest importer of hg and largest combustor of coal.
  - Chlor-alkali plants are centered in areas of world where environmental control is lacking or at best, is unknown.
  - There is unchecked use of mercury by artisanal gold miners in brazil, Ghana, Philippines and other countries.
- Domestic vs. Global mercury emissions & production



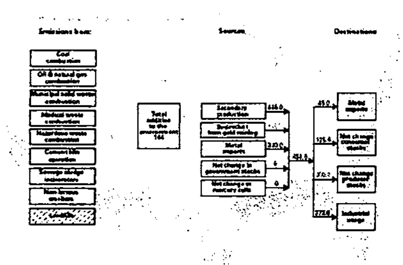
Components of U.S. Apparent Supply of Hg (1970-1998)



U.S Industrial Reported Consumption of Hg (1970-1997)



Domestic Flow of Hg in 1996, in Metric Tons



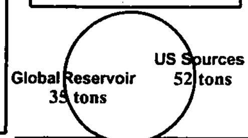
Annual U.S. Anthropogenic Mercury Emissions
158 Metric Tons

Sources

- 87% combustion
  - 33% coal fired utility
  - 19% municipal waste
  - 18% C/I boilers
  - 10% medical waste
  - 7% other sources
- 10% manufacturing
- 3% all other sources

Deposited

- 33.3% in US
- 66.6% outside US
- An additional 35 tons deposited in US from global reservoir



TOTAL DEPOSITION OF MERCURY IN US – 87 TONS

US Electric Power Plant Emissions

- **1994 - 91,422 pounds emitted**
  95% from coal burning plants.
  75% from plants built prior to 1977.
  (Stanfield and Lopez, 2000)
- **1995 - 1,128 plants emitted 42 tons**
  58% elementary
  42 % was oxidized
  1% was particulate
  (EPRI, 2001)

NOAA Atmospheric Modeling Division Laboratory (1997)

- 83% anthropogenic emissions originate from combustion sources.
  - 41% was elementary.
  - 41% was oxidized.
  - 18 % was particulate.

Is the Level of Hg in Environment Increasing ?

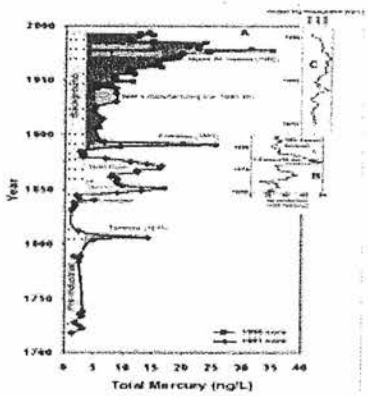
Since industrial revolution:

- In the atmosphere
  - 200 to 500 % - EPA, 1997
  - 300 to 600% - Mason, 1995
  - 370% or about 2% a year – Swain et al., 1992
  - 5.5 to 17% between (1990 – 1996) – Glass & Sorenson, 1999

- In Atlantic ocean water
  - 1.2 to 1.5 % per year since 1970 (Mason et al.,1970)
- In Atlantic sea bird feathers
  - 1.1% to 4.8% per year (Monterio and Funess, 1977)
- In tree rings, soil, sediments
  - 400 to 500% (Travis and Blaylock, 1992)
- In peat bogs
  - 200 to 300% (Zillioux et al., 1993)
- In lake sediments (Sweden)
  - 500% (Lindyish, 1991)

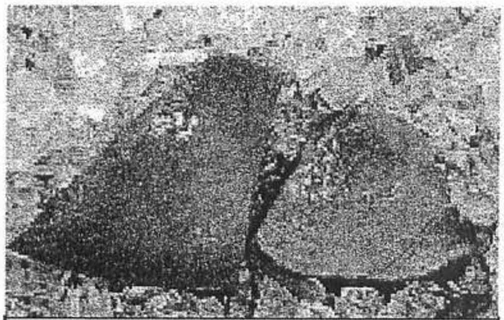
Schuster et al. 2002 Atmospheric Mercury Deposition During the Last 270 Years: A Glacial Ice Core Record of Natural and Anthropogenic Sources, Environ. Sci. Technol. April, 2002

- U. S. Geological survey - total hg in 97 ice-core samples from upper Fremont glacier, Wyoming
- Regional and global contributions – remote & high elevation
  - Over 270-year period
    - 42% background
    - 52% anthropogenic sources
    - 6% volcanic events
  - Over past 100 years
    - 20 fold increase in hg deposition
    - 70% from anthropogenic sources
  - Over past 10 years
    - Deposition has declined – currently a 11 fold increase over pre-industrial levels



FINAL THOUGHTS

- There is no quick fix.
  - Decades and generations may be required to reduce environmental mercury levels to acceptable levels.
  - It may require far longer than that for such reductions to be reflected in the food chain and particularly within those fish species near the top that are most frequently eaten by man.
- Local point sources are becoming more important.
  - Increasing background levels of mercury increase the potential impact of emissions from local point sources to effected areas.



The US Food and Drug Administration (FDA) advises that women of childbearing age,nursing mothers and young children should not eat shark, swordfish, king mackerel and tilefish and should limit consumption of other fish to no more than 12 ounces of cooked fish per week.

FINAL IMPRESSION

A Review Of Mercury In The Environment
(Its Occurrence In Marine Fish)

BY

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November 1, 2000

OFFICE OF ENVIRONMENTAL MANAGEMENT

MARINE RESOURCES DIVISION

SOUTH CAROLINA DEPARTMENT OF NATURAL RESOURCES

INTRODUCTION

A recent mercury advisory on consumption of king mackerel in South Carolina has resulted in numerous questions and concerns by the fishing public as well as the general public. To address these questions and concerns, the Department of Natural Resources (DNR) determined that a workshop for regional managers, biologists, the fishing public and the general public should be convened in early 2001. This document is a first step in planning that workshop. This paper is intended to collect facts and to objectively state the issues in terms that the layman can understand. Additionally, this report will serve as a guide for DNR and South Carolina Department of Health and Environmental Control (DHEC) in selecting the workshop's topics and speakers. Workshop proceedings including conclusions and recommendations will be published.

This report summarizes available information concerning the level and sources of mercury in the environment, particularly in marine fish; its transformation to methylmercury; its ability to bioaccumulate in the food chain; and its toxicity to man. A primary issue of concern addressed in this report is the adequacy and level of safety being provided to the American seafood consumer by federal and state agencies concerning mercury contamination in marine fish.

The Clean Water Act (Amendment 112(n)(1) B of 1990) required the United States Environmental Protection Agency (EPA) to conduct and report to Congress a study concerning: the rate and mass of mercury emissions from electrical utility steam generating units and other sources; the health and environmental effects of such emissions and the available technologies and the potential costs to control such emissions. This report, Mercury Study Report to Congress (EPA, 1997a), provides up-to-date information concerning mercury and emphasizes that "the typical U.S. consumer of fish is not in danger of consuming harmful levels of methylmercury and is not being advised to reduce fish consumption".

In March 1999, the U.S. Department of Health and Human Services, in accordance with guidelines developed by the Agency for Toxic Substance and Disease Registry (ATSDR) and EPA, published an updated Toxicological Profile for Mercury (Risher and Woskin, 1999). This report increased the minimal risk level (MRL) of 0.1 microgram per kilogram of body weight per day (ug/kg/d) established in 1994 for ingestion of methylmercury to 0.3 ug/kg/d. This increase, however, did not result in a change in advice provided by the Food and Drug Administration (FDA) regarding consumption of commercially caught fish. An individual of average weight is still advised to consume no more than about 7 ounces of fish containing 1 ppm or 14 ounces of fish containing 0.5 ppm per week. The report states: "Commercial fish sold through interstate commerce that are found to have levels of methylmercury above an "action level" of 1 ppm (established by FDA) cannot be sold to the public".

A subsequent report, prepared by the Mercury Policy Project and cosponsored by the Sierra Club and Clean Water Action entitled "The One That Got Away" (Bender and Williams, 2000), concludes that the FDA seafood mercury monitoring program is severely inadequate; that some commercially sold fish are above the FDA action limit; the health of the American consumer, particularly women and children, is being threatened; and that the American people are not being made aware of the risks associated with methylmercury in seafood.

In July 2000, the National Research Council (National Academy of Sciences) published a report (NRC, 2000) endorsing EPA's MRL or reference dose (RfD) for MeHg of 0.1 ug/kg per day as a scientifically justifiable level for the protection of public health. Congress requested the study be conducted prior to the establishment of the new, more stringent levels for mercury emissions from coal-burning power plants. This report concludes that American children of women that consume large amounts of fish and seafood during pregnancy may be at special risk of brain and nerve damage resulting in neurological problems, including learning disabilities.

The key points presented in the following pages of this review are:

1) Methylmercury is a worldwide pollutant originating largely from the burning of fossil fuels,

primarily in the generation of electrical power;

2) It is estimated that should all anthropogenic sources of mercury pollution be eliminated, it would require more than 50 years for methylmercury in fish to return to pre-industrial levels;

3) Methylmercury is a potent neurotoxin that can cause birth defects, learning disabilities, blindness, paralysis, loss of muscular control and death;

4) Methylmercury bioaccumulates through the food chain with the primary source of risk to human health being the consumption of fish (freshwater and marine);

5) Methylmercury in many freshwater and marine fish has been documented at levels that exceed those generally agreed upon by federal agencies (EPA and FDA), state agencies and recently by the National Academy of Sciences (National Research Council) and methylmercury constitutes a health risk that should be limited or avoided by man;

6) Pregnant women, women of child bearing age (15-44 years of age), and children aged 12 and under are of special concern. Eating ten grams (a quarter cup) of fish a day with an average mercury concentration of 0.1 to 0.15 ppm is up to twice the average EPA recommended reference dose; at a 1.0 ppm level the mercury intake range could be 6 to 12 times the exposure recommended by EPA;

7) There is a general misconception that commercially harvested fish and seafood can not be sold (seafood markets, restaurants, etc.) in this country if it contains more than the FDA action limit of 1.0 ppm of mercury;

8) South Atlantic states do not have a program to examine and document methylmercury contamination in marine fish and other wildlife such as exists in the San Francisco Bay region and the Gulf of Mexico,

9) No effective national education campaign exists for focusing on a factual and realistic evaluation of the dangers in consuming certain types of freshwater and marine fish and seafood, particularly in regards to that consumed by children under 12 years old and by women of childbearing age.

SOURCES AND MOVEMENT OF MERCURY

Mercury - What Is It?

Mercury is a basic chemical element of which there is a fixed amount on earth. It is a heavy, silvery-white liquid that vaporizes quickly at ambient temperatures. It exists in three oxidation states: metallic, mercurous and mercuric. Most mercury occurring in the atmosphere is in the form of elemental vapor. Most mercury in water, soil, sediments or biota is in the form of inorganic salts or organic (methylmercury) forms (EPA, 1997a).

Uses of Mercury

Mercury is utilized in the electrical industry (switches, thermostats, batteries etc.), dentistry (dental amalgams), numerous industrial processes including the production of chlorine and caustic soda, in nuclear reactors, as an anti-fungal agent for wood processing, a solvent for reactive and precious metal, and as a preservative of pharmaceutical products. Industrial demand for mercury peaked in 1964 and fell 74% between 1980 and 1993 and by another 75% between 1988 and 1996. This decline was largely a result of federal bans on mercury additives in paint and pesticides and the reduction of mercury in batteries (EPA, 1997a).

Sources of Mercury in the Environment

Mercury emissions into the environment can be characterized by three sources. These are: the natural release and cycling of geologically bound mercury, anthropogenic releases, and, thirdly, the re-emission of mercury to the atmosphere from that deposited to earth's surface in the past by the other two sources. Recent EPA estimates place the annual amount of mercury released into the air by human activities at 50 to 75 percent of the yearly total (EPA, 1997a). Of approximately 200,000 tons of mercury emitted to the atmosphere since 1890, about 95 percent resides in terrestrial soils, about 3 percent in ocean surface waters and 2 percent in the atmosphere. The amount of mercury in the atmosphere is estimated to have increased by 200 % to 500 % since the beginning of the industrial revolution (EPA, 1997a). Whereas mercury deposition rates have decreased in the vicinity of some localized sources in the western United States during the 1990s, measurements continue to increase in remote sites in northern Canada and Alaska indicating that the global atmospheric burden is continuing to increase (Monterio and Furness, 1997).

Between 1990 and 1996, atmospheric mercury levels have risen between 5.5% and 17% in the upper Midwest, depending on the season, with an average annual increase of 8% (Glass and Sorenson, 1999). Studies conducted in the Atlantic Ocean estimate a rise in mercury levels of 1.2 % – 1.5 % per year since 1970 (Mason et al. 1994). Recent studies indicate that mercury contamination in the marine environment is increasing at a rate of up to 4.8% a year (Monterio and Furness, 1997). Mercury concentrations in the feathers of seabirds breeding in the Azores, Madeira and Salvages islands, a tropical sector of the northeast Atlantic remote from mercury emissions due to human activity, were compared to preserved museum specimens dating back to 1886. Birds that typically feed on fish within the epipelagic layer (upper 100m of the ocean) showed an increase of an average of 1.1-%-1.9%. Feathers from birds that fed primarily on fish from the mesopelagic zone (below the epipelagic layer) showed average increases of 3.5% to 4.8% per year (Monterio and Furness, 1997).

Anthropogenic Sources of Mercury in the United States

Of the estimated 158 tons of mercury emitted annually into the atmosphere by human activities in the United States, approximately 87 percent is from combustion point sources, 10 percent from manufacturing, and 3 percent from all other sources (EPA, 1997a). Of this total, about one-third (52 tons) is deposited within the lower 48 states and two-thirds (107 tons) is transported outside of U.S. borders. An additional 35 tons is deposited within US borders from the global reservoir for a total annual mercury deposition of 87 tons. Four specific source categories (all high temperature waste combustion of fossil fuel processes) account for approximately 80 percent of total mercury emissions in the U.S.: coal-fired utility boilers (33 percent), municipal combustion (19 percent), commercial /industrial boilers (18 percent) and medical waste incinerators (10 percent). When fully implemented, current EPA emission limits established during recent years will reduce mercury emission by waste combustion and medical waste incinerators by 90 percent over 1995 levels (EPA, 1997a).

Electrical power plants built in the 1940s to 1970s are the largest industry source of mercury emitted into the environment. In 1994, such plants emitted a total of 91,422 pounds of mercury (Stanfield and Lopez, 2000). The vast majority (95%) came from coal–burning plants, and most of that was from those plants built prior to 1977 (77%). These plants have been and continue to be unregulated in regard to mercury emissions. The Clean Air Act passed by Congress in 1970, and amended in 1977 and 1990, exempted such plants from new air pollution standards. In fact, until 2000, all electrical utilities emitting less than 25,000 pounds of mercury a year were exempt from reporting (Sandfield and Lopez, 2000). As this was 12 times the annual emission level of the highest emitting plant in the U.S., all plants were therefore exempt. This reporting threshold was changed for the 2000 reporting year (report not due until 2002) to require that facilities that release 10 or more pounds of mercury annually must now report their releases.

Reduction and Associated Costs of Anthropogenic Mercury

Mercury emissions at 129 municipal waste combustion facilities could be reduced by 90% (26 tons) annually by material separation, product substitution, carbon filter beds, etc. at an estimated national annual cost between $11 and 47 million (EPA, 1997a). Fifteen tons of mercury emissions could be eliminated (a 95% reduction) at approximately 2,400 medical waste incinerators at a cost of $60 to 120 million. Seven tons of mercury could be eliminated at 14 chlor-alkali plants at a cost of $65 million. It will require an estimated national annual cost of $5 billion to remove 48 tons of mercury emissions per year (a 90% reduction) at 426 coal-fired utility facilities (EPA, 1997a).

Movement of Mercury in the Environment

Mercury in the form of vapor and/or inorganic salts may be transported great distances over several months in the atmosphere prior to falling out or being deposited by precipitation. It may be emitted back into the atmosphere as a gas or associated with dust particles to be re-deposited elsewhere. Thus, mercury is distributed to even the most remote areas of the earth. Mercury in soils has a long retention time, possibly hundreds of years and may continue to be released into the air and surface waters for many years to come. An expert panel on mercury and atmospheric processes concluded that if all mercury releases were stopped today it could **take at least 50 years for the methylmercury levels in fish to return to pre-industrial levels** (Standfield and Lopez, 2000).

THREATS TO WILDLIFE

Movement of Mercury into the Food Chain

Plants and animals, including man, are exposed to mercury as it cycles between the air, water and land by direct contact and by ingesting mercury-contaminated food. Elemental and inorganic forms of mercury are poorly absorbed in the digestive tract of higher animals. Very large quantities of inorganic mercury would have to be swallowed to cause toxicity in man. Less than 0.01% of any inorganic mercury that passes through the digestive system is absorbed and even that is rather quickly eliminated (EPA, 1997a). However, inorganic forms of mercury are efficiently bio-transformed by bacteria and other chemical processes to methylated forms that are almost completely absorbed within the digestive system and move efficiently through the food chain from the smallest organisms to top predators, including man. Methylmercury is eliminated from living tissue very slowly. Several months to years are required to reduce only half the mercury contaminant level within living tissue. **Thus, nearly 100% of the mercury that bioaccumulates at various trophic levels of the food chain is methylmercury (EPA, 1997a).**

Bioaccumulation

Mercury accumulates in living tissue when the rate of uptake exceeds the rate of elimination. Top aquatic predators such as freshwater largemouth bass, pike and walleye and marine fish such as king mackerel, sharks, and swordfish may contain concentrations of mercury 10,000 to 100,000 times greater than that found in the surrounding water (EPA, 1999). The bioaccumulation factor of methylmercury for all fish may be nearly 3 million and may approach more than 7 million for top predators. High levels of mercury contamination have been found in fish-eating birds such as the wood stork, loon, osprey and bald eagle, and mammals such as minks, otters, and the endangered Florida panther (EPA, 1997a). Similarly, the primary source of mercury contamination in man is through eating fish.

Toxic Impacts on Plants and Animals

Methylmercury concentrations in plant and animal tissue have been associated with sublethal effects and death (Risher and DeWoskin, 1999). Sublethal effects to plants include inhibition of growth, decreased chlorophyll, and leaf and root damage. Sublethal effects in animals include impaired growth and development, reduced reproductive success, liver and kidney damage and behavioral abnormalities. Laboratory studies have been utilized to assess the effects of methylmercury from fish to mink, otter and several avian species (EPA, 1997a). Effects can occur at a dose of 0.25 ug/g of body weight / per day with death occurring in some species at 0.1 to 0.5 ug/g body weight/ per day. Smaller animals, such as mink and monkeys are generally more susceptible to mercury poisoning than are larger animals, such as mule deer or harp seals (EPA, 1997a). Mercury is a known human toxicant (Mad Hatters Disease) with neurotoxic effects ranging from decreased motor skills, tremors, the inability to walk and convulsions to death (EPA, 1997a).

Mercury Levels in Fish

When an organism contaminated with methylmercury is ingested by a predatory fish (bird, man etc.) it is quickly absorbed and circulated by the animal's circulatory system. Methylmercury readily attaches to protein sodium ions throughout the fish's musculature (Minnesota Department of Health, 2000). **Skinning and trimming fish does not significantly reduce the mercury concentration in fillets, nor is it removed in the cooking process.** In fact, as cooking removes moisture, mercury concentrations are higher in fish flesh after cooking (EPA, 1999).

Mercury in Freshwater Fish

As part of the 1984-85 National Contaminant Biomonitoring Program, the U.S. Fish and Wildlife Service sampled freshwater fish for mercury contamination from 109 random stations nationwide. The maximum, geometric mean, and the 85th percentile concentrations for mercury were 0.37, 0.10 and 0.17 ppm (wet weight), respectively (Kidwell et al, 1995). In EPA's 1987 National Study of Chemical Residues in Fish, mercury was detected in fish at 92% of the

374 sites sampled (EPA, 1999). Maximum, arithmetic mean and median concentrations in fish tissue were 1.77, 0.26 and 0.17 ppm (wet weight). Freshwater sport fish (walleye, chain pickerel, largemouth and smallmouth bass) analyzed during the 1980s to 1996 in Canadian provinces consistently contained mean mercury concentrations greater than 0.5 ppm with individual fish exceeding 2.0 ppm. One largemouth bass was found to contain 8.94 ppm of mercury (EPA, 1999). In a separate study, largemouth bass in Florida measured as high as 4.4 ppm (Dukes, 98).

Most (68%) of all health advisories issued in the United States are the result of mercury contamination in freshwater fish (EPA, 1999). Mercury advisories in fish increased 115% from 1993 (899 advisories issued by 27 states) to 1998 (1,931 advisories issued by 40 states). Ten states have issued statewide advisories for mercury in their freshwater lakes and rivers. Eleven states have issued more than 90% of all mercury advisories: Minnesota (821), Wisconsin (402), Indiana (126), Florida (97), Georgia (80), Massachusetts (58), South Carolina (49) New Jersey (30), New Mexico (26), and Montana (22) (EPA, 1997a). The greater number of advisories concerning mercury in Minnesota and Wisconsin result from an active mercury sampling program of freshwater lake-fish in the 1970s and do not necessarily reflect greater levels of mercury contamination within their waterways.

Mercury in Marine Fish

Between 1963 and 1970 the average annual commercial catch (domestic and international fleet) of north Atlantic swordfish was about 22 million pounds. In 1969, FDA, in response to mercury poisonings in Japan, set an administrative guideline of 0.5 ppm for mercury in fish and shellfish moving in interstate commerce. In December 1970, as a result of the publication that most swordfish contained mercury in excess of this limit, what had been a flourishing swordfish fishery went into a period of decline (Booz et al., 1979). From 1971 to 1978, some U.S. fishermen continued to fish for swordfish in spite of the threat that their catches would be confiscated by the FDA for sampling and testing, and that most fish would not pass the 0.5 ppm restriction (SAFMC, 1985). Landings data for this period were considered unreliable (SAMFC, 1985).

In 1978, FDA's mercury content control was challenged in court (U.S. District Court, North District of Florida - Anderson vs. FDA and FDA vs. Anderson). Based on more detailed analysis of seafood consumption patterns prepared by the National Marine Fisheries Service, the allowable mercury content level was raised to 1.0 ppm (SAFMC, 1985). From 1978 to 1982, as consumers' fear of mercury contamination waned and consumption increased, the annual swordfish catch increased to 26 million pounds in 1983 and to 37 million pounds in 1989 (NMFS, 1997).

FDA currently advises that pregnant women and women of childbearing age who may become pregnant limit their consumption of shark and swordfish to no more than one meal per month. FDA further advises that persons other than pregnant women and women of child bearing age in the general population limit their regular consumption of shark and swordfish (which typically contains methylmercury around 1.0 ppm) to about 7 ounces per week (about one serving) to stay below the acceptable daily intake for methylmercury.

According to EPA's Report to Congress (EPA, 1997a), "mercury levels in marine fish have been monitored for more than 20 years by the National Marine Fisheries Service (NMFS) and have remained relatively constant in various species." However, the only NMFS data concerning mercury in marine fish which could be located by this author was a survey of trace elements in the fishery resources in mid 1970s (Hall et al., 1978). This comprehensive survey, initiated in 1971, examined the occurrence of 15 trace elements (including mercury) in 204 species of finfish, mollusca and crustaceans from 198 coastal United States sites. Those species reported as having a mean mercury level of 0.4 to 0.5 ppm or greater are listed below (Table 1.) The only marine species of commercial importance not sampled by this survey was swordfish, which, according to the authors, was not sampled in this study for "policy reasons" (Ahmed, 1991). The midpoint of the mean range for mercury within swordfish tissue from FDA surveillance samples during the 1970s was 0.95 ppm (Ahmad, 1991).

FDA sampling data, obtained in a 1999 Freedom of Information request and reported by Bender and Williams (2000), indicates that 36% of the swordfish, 33% of the shark and nearly 4 % of large tuna sold commercially in the United States between 1992 and

Table 1. Species reported in 1978 by the National Marine Fisheries Service - Survey of Trace Elements in the Fishery Resource (Hall et al., 1978) as having a mean mercury level of 0.4 to 0.5 ppm or greater.

Species	# in Sample	Range of mean mercury content in muscle, ppm
Atlantic barracuda	7	2.0 – 3.0
Atlantic bonito	15	1.0 – 2.0
Gafttopsail catfish	34	0.5 – 0.6
Smooth dogfish shark	95	1.0 – 2.0
Black grouper	33	0.7 – 0.8
Bluestriped grunt	16	0.6 – 0.7
Scalloped hammerhead shark	12	2.0 – 3.0
Smooth hammerhead shark	10	2.0 – 3.0
Jack crevalle	49	0.6 – 0.7
Ladyfish	2	2.0 – 3.0
Shortfin mako shark	3	2.0 – 3.0
Blue marlin	33	4.0 – 5.0
Striped marlin	40	1.0 – 2.0
White marlin	52	0.7 – 0.8
Sand perch	1	0.6 – 0.7
Red porgy	22	0.5 – 0.6
Sailfish	43	0.5 – 0.6
Atlantic sharpnose shark	1	0.8 – 0.9
Blacktip shark	16	0.7 - 0.8
American lobster (leg meat)	2	0.5 - 0.6
American lobster (tail meat)	2	1.0 - 2.0

1998 exceeded the 1.0 ppm action level for methylmercury. Approximately three-quarters of the sharks and swordfish and one-third of the large tunas sampled exceeded 0.5 ppm mercury. According to Bender and Williams (2000), FDA posed "detention" alerts for these three species in 1996 and 1997 but discontinued sampling these species for mercury, taking no samples in 1998 or 1999, and is no longer conducting a domestic monitoring program for these fish. Canned tuna (39 cans) sampled in 1992 revealed nearly 20% contained 0.3 to 0.5 ppm and ten percent exceeded 0.5 ppm mercury. The last testing of canned tuna (13 cans) by FDA for mercury was done in 1995 with 15% percent containing 0.3 to 0.5 ppm. Canned tuna is the most commonly consumed fish in the United States, averaging 10 cans per person per year (Johnson, 1999).

EPA studies also detected mean concentrations of methylmercury in muscle tissue of nine species of Atlantic sharks of 0.88 ppm (EPA, 1999). Mercury concentrations in these samples ranged from 0.06 to 2.87 ppm. Bluefin tuna from the northwest Atlantic Ocean were found to contain mercury at a mean concentration of 3.41 ppm (EPA, 1999). In 1994, EPA issued a chemical hazard alert for bonito, halibut, Spanish mackerel, king mackerel, shark, marlin and bluefin tuna based on a federal action level of 1 ppm mercury in the edible flesh of food fish bound for market. In 1998, Florida advisories for mercury included gafftopsail catfish, jack crevalle, spotted seatrout, ladyfish, sharks, and west coast king mackerel (Dukes, 1998).

EPA funded a program in 1988 to develop and implement voluntary, incentive-based management strategies to protect, restore, and maintain the health and productivity of the Gulf of Mexico ecosystem. The program is a partnership of Florida, Alabama, Mississippi, Louisiana, and Texas and 18 different Federal agencies, as well as numerous public and private organizations. A program report, while not making an evaluation or drawing conclusions about mercury-associated human health risks, provides existing knowledge of the mercury concentrations present in fish and shellfish within the Gulf of Mexico (Gulf of Mexico Program Report, 1999). Utilizing 6,620 records representing samples from 121 species (federal and state mercury monitoring samples) collected on or after January 1, 1990, mercury concentrations were mapped and made available on the internet (http://www.duxbury.battelle.org/gmp/newExecSum.htm). The report concludes that mercury is common in edible tissues of estuarine/marine fish and shellfish harvested from the Gulf of Mexico. Approximately 77 percent of the 24 species/species groups (including three size classes of king mackerel) analyzed in the study had a Gulfwide mean mercury concentration between 0.2 and 1.0 ppm. Species reported as containing mercury levels greater than 0.4ppm Gulfwide are listed in Table 2. No species or species/group had a Gulfwide mean mercury concentration greater than 1.0 ppm.

Tissues from seven fish species from San Francisco Bay were analyzed for mercury in 1994 and 1997 (Davis, 1997). More than half of the fish showed concentrations above 0.23 ppm. An overall average level of mercury for the seven species examined was 0.3 ppm with the highest levels occurring in leopard sharks, which exceeded 1.0 ppm, and in individual striped bass samples (0.9 ppm). A positive correlation of increasing mercury concentrations with increasing fish length (age) was noted in several species. Based on these studies, the California Office of Environmental Health and Hazard Assessment (1998) issued health advisories warning that: adults should eat no more than two eight-ounce meals per month of San Francisco Bay sport fish, including sturgeon and striped bass caught in the Delta; striped bass over 35 inches should not be eaten; and women who are pregnant or may become pregnant, nursing mothers and children under the age of six should not eat more than one meal of fish per month. No striped bass over 27 inches or any shark over 24 inches should be eaten.

Mercury in King Mackerel

Health advisories concerning the consumption of large king mackerel (over 43 inches total length) taken from the Gulf of Mexico were issued by all Gulf states during 1997-98 (Dukes, 1998). In response to the detection of high levels of mercury in Gulf Coast king mackerel, North Carolina sampled the mercury content of king and Spanish mackerels in November 1998 (Hale, 1999). The 22 Spanish mackerel samples ranged from 0.06 to 0.84 ppm mercury and the 30 king mackerel fillets ranged from 0.36 to 3.0 ppm. **In 1999, king mackerel examined by Florida, Georgia, South Carolina and North Carolina were found to contain mercury levels as high as 3.5 ppm and health advisories were issued by each state (DHEC, South Carolina, 2000).**

The 1999 king mackerel fillets were collected and independently analyzed for mercury concentration by

Table 2. Species and species groups reported as having Gulfwide mean mercury levels of 0.4 or greater in edible fish tissue collected since 1990 (Gulf of Mexico Program Report, 1999).

Species	Number of Samples	Mean of mercury in edible tissue (ppm)	Maximum site value (ppm)
Blacktip shark	73	0.86	2.0
Bonnethead shark	76	0.51	1.4
Groupers (*Mycteroperca*)	64	0.43	1.4
Jack crevalle	68	0.63	3.1
Sand seatrout	93	0.57	0.9
Largemouth bass	723	0.46	1.6
King mackerel(>39")	58	0.96	1.7
King mackerel (33-39")	89	0.69	1.1
King mackerel (<39")	77	0.60	1.7
Spanish mackerel	179	0.57	1.7
Common snook	190	0.50	1.5

North Carolina (112 fish), South Carolina (28 fish), Georgia (20 fish) and Florida (21 fish). Mercury levels were similar in each state's samples and were correlated with fish length (Figure 1). Health advisories were jointly issued by each state and were based on fish length:

1) No consumption limits were placed on king mackerel less than 33 inches (fork length);

2) Individuals should eat no more than four 8-ounce servings of king mackerel from fish between 33 and 39 inches (fork length) per month;

3) Children (up to 12 years of age) and women of child bearing age should consume no more than one 8-ounce serving per month from fish between 33 and 39 inches;

4) King mackerel more than 39 inches in fork length should not be eaten.

To determine the possible consequences of mercury levels in large king mackerel exceeding the 1.0 ppm FDA action level, fisheries representatives contacted FDA for guidance as to their possible future actions to restrict the distribution and /or sale of such fish (Gregory M. Cramer, Center for Food Safety and Applied Nutrition, FDA, pers. comm. 2000). Information provided to FDA indicated that king mackerel landed commercially average 27-34 inches, only 10 to 15% fish exceed 39 inches and overall mercury level of commercially landed fish of around 0.6ppm. States were advised that FDA's policy for MeHg focused on time weighted exposures rather than on exposures from an individual meal or individual fish. FDA concluded that the information provided showed that there is only a 10 to 15 percent chance that commercially harvested king mackerel will have MeHg levels exceeding 1 ppm and that the average contamination level is 0.6 ppm. Since FDA's 1 ppm limit focuses on a lot average and there is little likelihood of exceeding that limit, FDA would not prohibit the sale of king mackerel over 39 inches.

The mean mercury level by size category for the 181 king mackerel sampled by all four states is given in Table 3. The length frequency of king mackerel caught commercially between 1995 and 1999 in South Carolina is provided in Figure 2. The length frequency of king mackerel taken recreationally from North Carolina, South Carolina, Georgia and Florida based upon

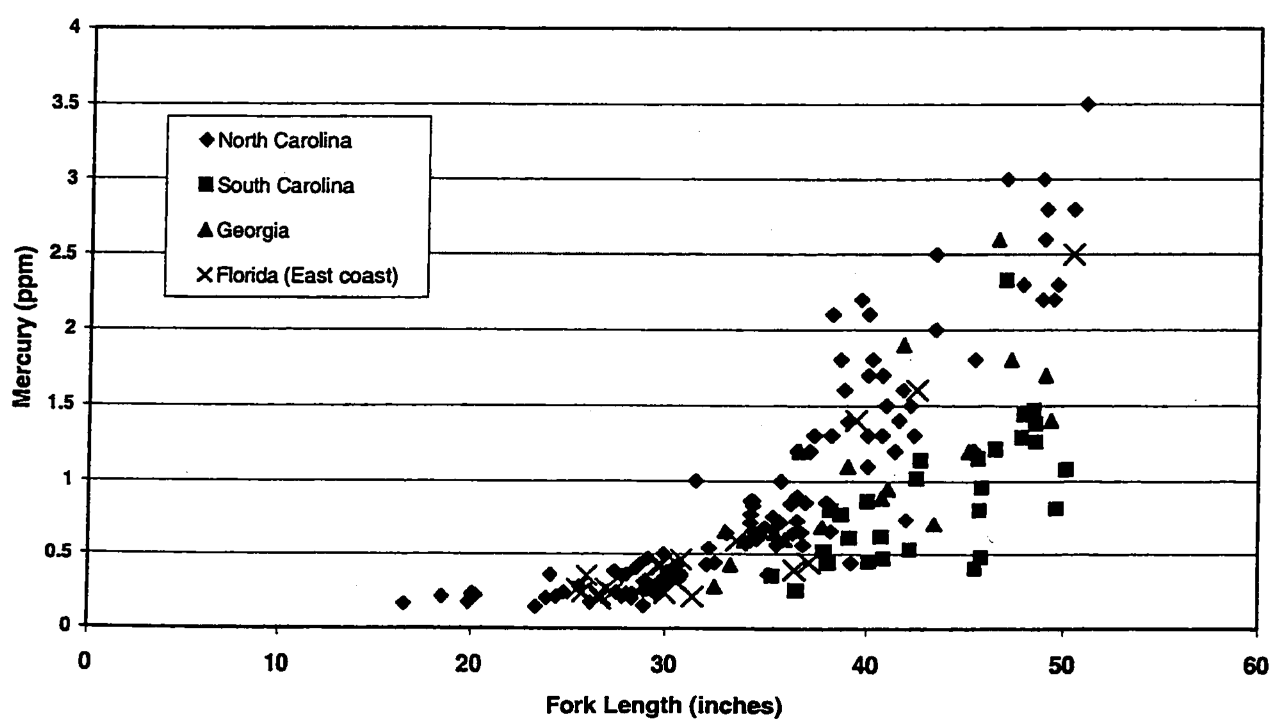

Figure 1. Mercury concentrations in the edible tissue of king mackerel collected in North Carolina, South Carolina, Georgia and Florida (East Coast).

the NMFS Marine Recreational Fishing Survey (1999) is presented in Figure 3.

THREATS TO HUMANS

Methylmercury Poisoning

Two major cases of methylmercury poisoning through fish consumption have been documented, both in Japan (EPA, 1997a). The first occurred in Minamata, Kyushu, Japan during the late 1950s and 1960s. Methylmercury in waste sludge from a chemical factory that used mercury as a catalyst drained into Minamata Bay. Mercury concentrations in fish, which were a primary diet item of local residents, were between 10 and 30 ppm wet weight. Thousands of individuals complained of symptoms, now known as Minamata disease, including impairment of: peripheral vision, speech, hearing, and walking; a feeling of "pins and needles" in the hands and feet; uncoordination of movements as in writing; and mental disturbances. Many people (adults and children) died. It was recognized that nervous system damage could occur to the fetus if the mother ate fish contaminated with high concentrations of methylmercury during pregnancy.

Table 3. The mean methylmercury level in 181 king mackerel sampled in 1999 by North Carolina, South Carolina, Georgia and Florida, by size category:

Size Category (Fork length)	(Number of fish)	Average MeHg	Range
< 27 inches (legal size limit)	19	0.22 ppm	0.14 – 0.36 ppm
27 to 32 inches	43	0.34 ppm	0.15 – 1.00 ppm
33 to 39 inches	53	0.80 ppm	0.25 – 2.10 ppm
> 39 inches	66	1.54 ppm	0.40 – 3.50 ppm

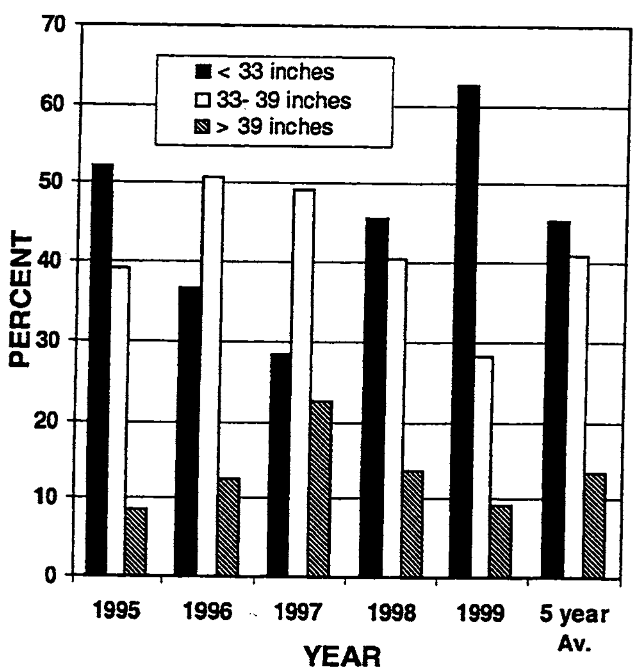

Figure 2. Length frequency of king mackerel caught commercially in South Carolina (SCDNR Marine Resources Statistics Program Annual Reports 1995-1999).

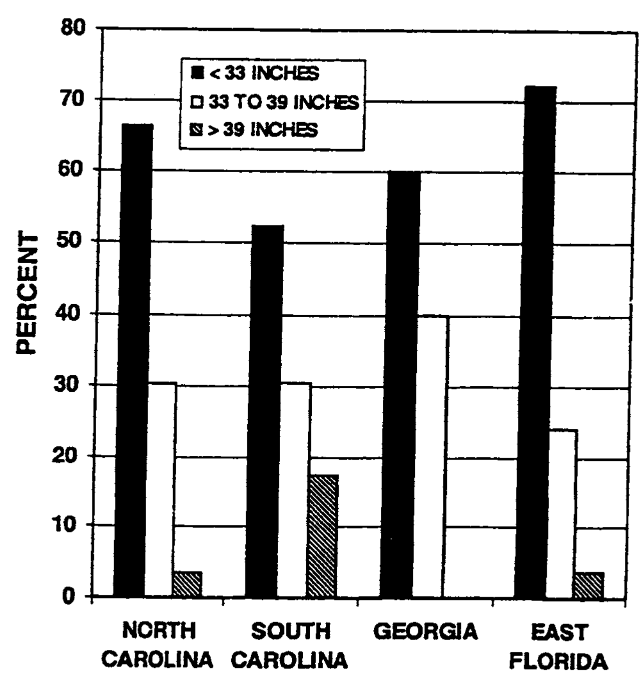

Figure 3. King mackerel length frequency distributions (NMFS Marine Recreational Fisheries Survey, 1999).

In 1965, a second methylmercury-poisoning outbreak was traced to a chemical factory releasing methylmercury into the Agano River in Japan (EPA, 1997a).

Two additional poisoning incidents have been documented from the consumption of seed grain treated with a fungicide containing methylmercury. Severe human poisoning occurred in Iran in 1960, and again in Iraq in 1970, which was estimated to have hospitalized approximately 6,500 people with 459 fatalities reported (EPA, 1997a).

Characterization of Risk to Human Populations

The characterization of risk to U.S. human populations focuses on exposure to methylmercury over time. Ingestion of fish tissue is the dominant exposure pathway. The critical elements in estimating the risk of methylmercury exposure from fish are: the species of fish consumed, the concentration of methylmercury in the fish, the quantity and frequency of consumption, and the sex and age of the individual eating the fish (EPA, 1997a). There has been a 25 % increase in fish consumption in the United States since 1980 (Bender and Williams, 2000). On average, Americans eat about 19 pounds of fish each year and approximately 15 pounds (75%) is marine (Bender and Williams, 2000).

MeHg Reference Dose

A reference dose (RfD) is defined as an estimate of a daily exposure to the human population (including sensitive subpopulations) that is likely to be without an appreciable risk of deleterious effects during a lifetime (Risher and DeWoskin, 1999). The RfD for methylmercury has been determined by EPA to be 0.0001 mg per kg of body weight per day, meaning a person could consume 0.1 microgram (ug) methylmercury for every kg of his/her body weight every day for a lifetime without anticipation of risk of adverse effect. A recent study mandated by the U.S. Congress, including an evaluation of three large epidemiological studies in the Seychelles Islands, Faroe Islands and New Zealand by the National Academy of Sciences, endorsed EPA's RfD for MeHg and found it to be scientifically justifiable for the protection of public health (NCR, 2000).

General Population

The FDA advises the general population to limit their consumption of fish species which have methylmercury levels around 1.0 ppm to about 7 ounces or about one serving per week (Risher and DeWoskin, 1999). Fish consumed with levels averaging around 0.5 ppm should be limited to 14 ounces per week or two servings. The most recent U.S. Department of Health and Human Services toxicological profile for mercury (Risher and DeWoskin, 1999) states the following:

> "The Food and Drug Administration (FDA) estimates that most people are exposed on average to about 50 ng of mercury per kilogram of body weight per day (50 ng/kg/day) in the food they eat. This is about 3.5 micrograms (ug) of mercury per day for an adult of average weight. This level is not thought to result in any harmful effects. A large part of this mercury is in the form of methylmercury and probably comes from eating fish. Commercial fish sold through interstate commerce that are found to have levels of methylmercury above an "action level" of 1 ppm (established by the FDA) cannot be sold to the public. This level itself is below a level associated with adverse effects. However, if you fish in contaminated waters and eat the fish you catch, you may be exposed to higher levels of mercury. Public health advisories are issued by state and federal authorities for local waters that are thought to be contaminated with mercury. These advisories can help noncommercial (sport and subsistence) fishermen and their families avoid eating fish contaminated with mercury."

EPA recommendations are based on an integrated risk information system. EPA recommends that an individual of average weight (158 pounds) in order to not surpass an RfD of 0.0001 mg/kg of body weight / day not to consume more than: one meal (8 oz. portion) of fish containing more than 0.5 ppm MeHg per month; or one meal every other month of fish containing 1.0 ppm or more. Fish contaminated with more than 1.9 ppm MeHg should never be eaten (Table 4).

Table 4. EPA recommended monthly fish consumption limits (number of 8 ounce portions) of fish containing various levels of MeHg for an individual weighting 72kg(158 pounds) in order to not exceed the recommended RfD of 0.0001mg/kg of body weight/d (EPA, 1999).

Concentration in fish tissue MeHg (ppm)	Fish meals/month (8 ounce portions)
>0.03 - 0.06	16
>0.06 - 0.08	12
>0.08 - 0.12	8
>0.12 - 0.24	4
>0.24 - 0.32	3
>0.32 - 0.48	2
>0.48 - 0.97	1
>0.97 - 1.9	0.5
>1.9	NONE

Subpopulations of Concern

Pregnant women, women of child bearing age (15-44 years of age), and children aged 14 and under are of special concern. EPA advises that anyone in this group who is eating ten grams (a quarter cup) of fish a day with an average mercury concentration of 0.1 to 0.15 ppm is at or up to twice the average EPA recommended RfD for mercury. Should the fish have a mercury concentration of 0.5 ppm, it may expose them to three to six times the interim RfD and at a 1.0 ppm level the mercury intake range could be at 6 to 12 times the recommended exposure (EPA, 1997a). Bender and Williams (2000) point out that EPA's Mercury Study to Congress (EPA, 1997b) estimates that 7 million women and children are at risk of mercury poisoning due to consumption of fish. Because swordfish, sharks and other large predatory fish may contain methylmercury levels which exceed the FDA 1.0 ppm limit, that agency's advice to consumers warns pregnant women and those of child bearing age to limit their consumption of such fish to no more that one meal a month (FDA, 1995). Four states (Vermont, Minnesota, Michigan and New Jersey) recommend that expectant mothers and children not eat swordfish or shark and limit consumption of canned tuna to 7 ounces per week. Seven states (Texas, Alabama, Mississippi, Florida, Georgia, South Carolina, and North Carolina) recommend that the public, and especially women and children, should limit consumption or not eat larger king mackerel because of their high mercury content.

Jurisdiction and Action Limits

The United States Food and Drug Administration (FDA) has jurisdiction of fish sold in commerce and has set an action level of 1.0 mg/kg body weight (ppm) (Federal Register 44:3990, January 19,1979). EPA reports the concentration of methylmercury in the ten most commercially important marine species (tuna, shrimp, pollack, salmon, cod, catfish, clam, flounder, crab, and scallop), on the average, to be close to ten times lower than the action limit. FDA originally set an administrative guideline of 0.5 ppm for mercury in 1969 for both fish and shellfish in interstate commerce. This was converted to an action level in 1974, increased to 1.0 ppm in 1979 and converted from a mercury standard to one based on methylmercury in 1984 (EPA, 1997a).

FDA public information concerning Action Limits indicates:

1) "Action levels and tolerances represent limits at or above which FDA will take legal action to remove products from the market. The blending of a food or feed containing a substance in excess of an action level or tolerance with another food or feed is not permitted." (FDA, 1998)

2) "FDA works with state regulators when commercial fish, caught and sold locally, are found to contain methyl mercury levels exceeding 1 ppm. The agency also checks imported fish at ports and refuses entry if methylmercury levels exceed the FDA limits." (FDA, 1995)

According to Bender* and Williams ** (2000), FDA is using guidance developed in the 1970s for protecting the public from mercury levels in seafood and the 1.0 ppm action level for mercury, established in 1979, is not legally enforceable and only serves as discretionary guidance to FDA and states. Public awareness of mercury exposure is significantly lacking. **Guidelines, programs and practices established by FDA are seldom implemented and provide the American public with a false sense of safety about the consumption of mercury contaminated seafood (Bender and Williams, 2000).** Whereas, 75% of the public responding to a recent survey in the Northeast indicated that they eat fish on a regular basis, only about one-half were aware of FDA or state advisories and only one-third knew their meaning (NESCAUM, 1999).

FDA and EPA state that each state has the primary responsibility for protecting its residents from the health risks of consuming contaminated, non-commercially caught fish. They do this by issuing recommendations to the public to either limit or avoid consumption of certain fish from specific waterways, or in some cases, from all state waters (EPA 1997a). However it is also acknowledged " that not all anglers heed such advice".

Southeastern states (North Carolina, South Carolina, Georgia, and Florida) each utilize different approaches for developing fish advisories for mercury in state waters (Manning, 2000). Florida bases its recommendations on 1976 toxicity criteria for mercury reported by the Word Health Organization. The general public should not consume more than one meal a week of fish containing approximately 0.5 ppm mercury. More sensitive individuals (women of child bearing age and children 12 years old and younger) should not consume more than one meal of such fish a month. Fish containing up to 1.5 ppm should not be eaten more than once a month and fish with greater amounts of mercury should not be consumed. South Carolina and Georgia set similar consumption guidance levels for fish containing mercury – fish containing 0.23 – 0.25 to 0.6 – 0.7 ppm should be limited to one meal per week, fish with 0.6 –0.7 to 2.3 – 3.0 ppm should not be consumed more than once a month and fish with 2.0 to 3.0 ppm should never be consumed. North Carolina has utilized a level of 1 ppm for issuing fish consumption advisories in the past but is currently recommending a mercury toxicity criteria of 0.2 ug/kg/day for sensitive populations and 0.5 ug/kg body weight /day for non-sensitive populations (Manning, 2000).

SUMMARY

The National Academy of Science's National Research Council (NRC) report (2000) concerning the toxicological effects of methylmercury (MeHg) recently endorsed EPA studies concerning its toxicological effects. The report states, "On the basis of its evaluation, the committee's consensus is that the value of EPA's current RfD for MeHg, 0.1 ug/kg (body weight) per day, is a scientifically justifiable level for the protection of pubic health". The Committee found that high-dose MeHg exposure effects included mental retardation, cerebral palsy, deafness, blindness and dysarthria. Low-dose prenatal exposure to MeHg from maternal consumption of fish has been associated with poor performance on neurobehavioral tests, particularly on tests of attention, fine motor function, language, visual-spatial abilities and verbal memory. They also found evidence in humans and animals that MeHg levels even lower than those associated with neurodevelopmental effects can have adverse effects on the developing and adult cardiovascular system (blood pressure regulation, heart-rate variability, and heart disease).

The NRC report (2000) confirms that public health concerns expressed by both federal and state agen-

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***Jane Williams is the executive director of California Communities Against Toxics. She has a degree in economics from the University of California and serves on the board of the California Environmental Research Group, the Clean Air Network, the Mercury Policy Project, and the Nonstockpile Chemical Weapons Forum.*

cies during the past forty years about the level of MeHg in fish and seafood are real and justified. More stringent requirements of mercury emissions from coal-burning power plant are needed. An obvious need for additional sampling exists for long term monitoring of MeHg in freshwater and marine fish as well as other types of seafood at both a state and federal level.

From a public health standpoint, the greatest need is to provide the American public with an effective education campaign focusing on a factual and realistic evaluation of the dangers in consuming certain types of freshwater and marine fish and seafood. This is particularly true in regards to fish and seafood consumed by children under 12 years old and by women of childbearing age. Federal and state mercury advisories that have been issued are poorly reported, generally ignored by the public and fail to adequately warn of the combined effects of consuming various types of meals containing mercury contamination. State mercury advisories (including South Carolina's) are primarily based on river systems and the number of meals that should not be exceeded for various types of fish. Few, if any, advisories indicate that if an individual eats a meal of contaminated fish or other food containing methylmercury, such as a tuna-fish sandwich, that all other foods which may also contain methylmercury should be avoided.

There is a general misconception that commercially sold (seafood markets, restaurants etc.) fish and seafood can not be sold in this country if it contains more than the FDA action limit of 1.0 ppm of MeHg. FDA's lack of sampling methylmercury content in marine fish and seafood as well as the policy of focusing on time weighted exposures rather than on exposures from an individual meal or fish makes it impossible for an individual to determine his level of exposure. All state and federal advisories recommend that more sensitive sub-populations not consume a single meal containing MeHg concentrations greater than 2.0 ppm.

Federal and state agencies in the Southeastern United States need to combine efforts and resources to develop a program similar to those being carried out by the California Regional Water Quality Control Board in the San Francisco Bay Region and by the Gulf of Mexico Program in the Gulf to examine and document environmental issues such as MeHg contamination in estuarine and marine fish as well as other seafood.

Program efforts are needed to:

1) establish an effective public education program particularly aimed at parents of young children and women of childbearing age as to the occurrence of mercury in fish and seafood and safe consumption levels;

2) identify primary species (size classes or sub-populations) that, based on feeding habits and life expectancy, should be analyzed and monitored for MeHg;

3) determine the level of MeHg contamination in water, sediments, fish and other aquatic organisms in the regions fresh and marine waterways;

4) and establish a long-term regional MeHg monitoring program.

LITERATURE CITED

Ahmed, F.E. (editor). 1991. Seafood Safety committee on evaluation of the safety of fishery products. National Academy Press, Washington, DC.

Bender, M. and J. Williams. 2000. The one that got away: FDA fails to protect the public from high mercury levels in seafood. Mercury Policy Project Report 19p.

Booz, Allen and Hamilton Inc. 1979. Description of the swordfish fishery. For: South Atlantic Fishery Management Council. Charleston, S.C.

California Office of Environmental Health and Hazard Assessment. 1998. Interim Fish Consumption Health Advisory.

Cramer, Gregory M. 2000. Guidance (FDA) to the State of North Carolina re: the sale of king mackerel with > 1ppm MeHg. Center for Food Safety and Applied Nutrition, FDA, email dated March 30, 2000).

Davis, J.A. 1997. Contaminant concentrations in fish from San Francisco Bay. Richmond, California, San Francisco Estuarine Institute. 99p.

DHEC (South Carolina Department of Health and Environmental Control). 2000. King Mackerel advisory issued. Press Release March 23, 2000 Columbia, SC. 2p.

Dukes, C. 1998. Heavy metal blunder. Texas Fish and Game Magazine. March 1998 2p.

EPA (U.S. Environmental Protection Agency). 1997. A Mercury Study Report to Congress, Washington DC Volume 1: Executive summary.

EPA (U.S. Environmental Protection Agency). 1997b. Mercury Study Report to Congress, Washington DC Vol. IV: An assessment of exposure to mercury in the United States.

EPA (U.S. Environmental Protection Agency). 1999. Fact Sheet - Mercury Update: Impact on fish advisories. 13p.

FDA (U.S. Food and Drug Administration). 1998. Action levels for poisonous or deleterious substances in human food and animal feed. Industry Activities Staff Booklet, March 1998.

FDA (U.S. Food and Drug Administration). 1995. Mercury in fish. Cause for concern?. In U.S. Consumer, Revised May 1995.

Glass, G. and J. Sorenson. 1999. Six year trend (1990-1996) of wet mercury deposition in the upper Midwest, USA. Jour. Envirn. Sci. Techn. September 1999.

Gulf of Mexico Program. 1999. The Occurrence of Mercury in the Fishery Resources of the Gulf of Mexico. Executive Summary. http://www.duxbury.battelle.org/gmp/new/ExecSum.htm

Hale, T.F. 1999 . Results of tissue mercury analysis for period 1 mackerel samples. State of North Carolina Department of Environment and Natural Resources: Inhouse Report 7p.

Hall, R.A., E.G. Zook and G. M. Meaburn. 1978. National Marine Fisheries Service survey of trace elements in the fishery resource. NOAA Tech. Rep. NMFS SSRF-721.

Johnson, H.M. 1999. Annual report on the U.S. seafood industry, 7th ed. Bellevue (WA): H. M. Johnson & Associates; 1999.

Kidwell, J.M., L.J. Phillips, and G.F. Birchard. 1995. Comparative analyses of contaminate levels in bottom feeding and predatory fish using the National Contaminant Biomonitoring Program Data. Bulletin Environmental Contamination and Toxicology. 54:919-923.

Mason, R., W. Fitzgerald and F. Morgan 1994 Gecehemico Cosmochinm Acta. 58(15):3191-3194.

Manning, R.O. 2000. Briefing for the South Atlantic Fishery Management council – Mercury assessment in king mackerel and resulting consumption advisory. Environmental Protection Division, Georgia DNR, 11p.

Minnesota Department of Health. 2000. Mercury in the Environment. Fish Facts.

Monteiro, L.R. and R.W. Furness. 1997. Accelerated increase in mercury contamination in North Atlantic mesopelagic food chains as indicated by time series of seabird feathers. Journal of Environmental Toxicology and Chemistry 16 (12): 2489-2493.

NESCAUM (Northeast States for Coordinated Air Use Management.) 1999 Preliminary findings from the northeast fish consumption and mercury awareness survey. May 1999.

NRC (National Research Council -Academy of Natural Sciences and Engineering). 2000.Toxicological effects of methylmercury (Executive Summary) National Academy Press 9p.

NMFS (National Marine Fisheries Service). 1997. Draft Amendment 1 to the Fishery Management Plan for Atlantic Swordfish including an environmental assessment and regulatory impact review. Highly Migratory Species Division (NMFS), NOAA. January 28, 1997. 107p.

NMFS (National Marine Fisheries Service). 1999. Marine Recreational Fishery Survey. N.O.A.A. (NMFS) Washington, DC. Cur. Fish. Stat.

Risher, J.R. and R. DeWoskin. 1999. Toxicological profile for mercury (update). U.S. Dept. Health & Human Services. 617p.

South Atlantic Fishery Management Council (SAFMC). 1985. Fishery Management Plan, Regulatory Impact Review, Initial Regulatory Flexibility Analysis and Final Environmental Impact Statement for Atlantic Swordfish. Southpark Circle, Suite 306, Charleston, South Carolina, February, 1985.

South Carolina Department of Natural Resources (Marine Resources Division –Office of Fisheries Management) Commercial Fisheries Statistics Program. 1995-1999. Annual reports.

Stanfield, R. and C. Lopez. 2000. Lethal legacy - the dirty truth about the Nation's most polluting power plants. U.S. Public Interest Research Group (U.S. PIRG), the National Campaign Against Dirty Power 42p.

Mercury in the Environment

Chemistry of Mercury to Methylmercury
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Biogeochemical Controls on Monomethyl Mercury Production in Aquatic systems

It is now well recognized that the chemical form or chemical speciation of an element in aquatic systems dictates the elements transport, fate, bioavailability, and toxicity in aquatic systems. The chemical and phase speciation of mercury in aquatic systems is especially complex. Mercury can be found in the environment in two oxidation states, Hg (0) and Hg (II), it has an affinity to interact with natural organic material, it is very particle reactive, and it can be converted to methylmercury, which bioconcentrates in aquatic food webs, potentially leading to concentration levels in top fish which exceed safe consumption advisory levels.

From a thermodynamic viewpoint, the inorganic chemical speciation of mercury is fairly well understood. Interactions of mercury with organic material in aquatic systems has long been recognized as potentially important, but currently is poorly understood. Recent investigations in our laboratory have suggested that a substantial portion of what is often considered "dissolved" mercury is actually mercury associated with macromolecular colloidal organic matter. In addition, using competitive ligand equilibration techniques, we have found that a major portion of the mercury present in Galveston Bay is complexed by ~10 pM of a natural organic ligand(s), with a conditional stability constant > 10^{29}. These findings suggest that >99% of the solution forms of mercury in oxic estuarine systems exist as mercury-organic complexes. How broadly representative these findings hold true remains to be investigated.

It is now fairly well accepted that the main pathway for the introduction of methylated mercury forms into aquatic systems is via in situ production, mediated by sulfate-reducing bacteria. There is often a temptation in assessing methyl mercury concentrations and production in aquatic systems to focus predominately on loading or abundance of inorganic Hg as the dominant controlling factor. While this is indeed an important factor, it is by no means the only important factor, nor necessarily the controlling factor. A number of parameters have been identified as important in influencing the production and abundance of methyl mercury in aquatic systems including: mercury loading, the chemical form of mercury (chemical speciation), temperature, the availability of organic substrate for sulfate-reducing bacteria (i.e. a food source), mercury de-methylation activity (by bacteria), in situ reduction-oxidation conditions and in some cases photo-demethylation. To complicate the issue even more, many of these parameters vary temporally and spatially in aquatic systems. Any of these parameters can potentially limit the abundance of methylmercury in an aquatic system.

Benoit, Gilmour, Mason and colleagues have recently proposed that sulfide levels in aquatic systems can be very important in controlling methylmercury production by sulfate-reducing bacteria. This influence arises from the strong interaction between inorganic mercury and sulfide to form mercury-sulfide complexes and the bioavailability of these complexes to sulfate-reducing bacteria. They hypothesize that only neutrally charged mercury complexes (e.g. Hg^0, HgS^0 or $HgCl_2{}^0$) are capable of readily passing bacterial membranes for intra-cellular mercury methylation. Hence, the in situ chemical speciation of mercury is very important in controlling Hg methylation. In anoxic systems, the inorganic speciation of mercury is dominated by sulfide complexes. At low sulfide levels (< 10 μM), neutrally charged HgS^0 dominates the Hg-sulfide speciation. Above this concentration level, polysulfide (charged) complexes of Hg dominate.

The role of oil and gas platform operations and practices in directly or indirectly promoting the formation of methylmercury and its incorporation into the food webs around the platforms is currently of great interest. Platforms are frequently characterized as "Oases for Marine Life in the Gulf" or "Islands or Life", due to the proliferation of marine life which concentrates around the platforms. It is possible to speculate that this phenomenon might be promoting environmental conditions in sediments around the platforms which enhance the production of methylmercury. If the deposition of organic debris from the marine life around the platforms is locally elevated, this might provide the "fuel" needed to drive down oxygen levels, promote sulfate-reduction, and enhance methylmercury formation. This condition would exist even without the current concern of the possible augmentation of mercury in sediments from barite drilling mud. Whether such a localized phenomena is important on a Gulf-wide basis remains to be determined.

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Research Interests and Specialties

The biogeochemistry of trace elements in natural waters, with particular experience and interest in environmental and analytical studies concerning mercury. Trace element analysis using ultra-clean laboratory and environmental sampling techniques. Chemical and biological interactions involving trace elements in aquatic environments. The chemical speciation of trace elements in natural waters. Understanding the role that chemical speciation plays in the interaction of trace elements with biota and particulate surfaces and the influence of this interaction on the geochemical cycling of trace elements in natural waters. Geochemical processes which control the temporal and spatial distribution of trace elements in estuaries, surface and deep waters of the ocean and other aquatic systems.

Professional Affiliations

- American Chemical Society
- American Geophysical Union
- Geochemical Society
- Oceanographic Society American
- Society of Limnology and Oceanography
- Texas Academy of Sciences
- Estuarine Research Federation

Mercury in the Environment

Atmospheric Deposition of Mercury

Dr. Jane Guentzel
Coastal Carolina University

The Importance of Chemical Speciation, Climate, and Meteorology

Mercury exists in many different physical and chemical forms in the environment and it is the interconversions between these species that mediate its distribution patterns and biogeochemical cycling. The most widely known conversion is the biological transformation of inorganic Hg (II) to organic (methyl) Hg and its subsequent biomagnification in piscivorous fish, which poses a risk to higher trophic level organisms and humans who consume these fish. As the atmosphere is considered the dominant pathway for the delivery of inorganic Hg to aquatic ecosystems (1), this presentation will discuss the chemical species of Hg in the atmosphere; the sources of Hg to the atmosphere; and the transport and deposition of inorganic Hg to aquatic ecosystems.

In the atmosphere, mercury exists predominantly in the zero oxidation state as gaseous elemental Hg (Hg^o) and in the +2 oxidation state as particulate Hg (Hg_p) or as reactive gaseous Hg (Hg(II)). Gaseous elemental Hg comprises 97-99% of the total mercury found in the atmosphere and has a residence time on the order of 1 year (1,2). The remaining 1-3% is comprised of Hg(II) and Hg_p,, with residence times on the order of days to weeks (2). Reactive gaseous Hg can be formed in the atmosphere through the oxidation of gaseous elemental Hg by ozone (3,4) or halogen radicals in the marine boundary layer and troposphere (5,6). Hg(II) is incorporated into cloud droplets or becomes attached to particulate material and is scavenged from the atmosphere by wet and dry deposition processes. Hg_P can dry deposit to surfaces, be incorporated into cloud droplets, or be scavenged by precipitation.

These various species of mercury in the atmosphere originate from natural processes (25-30%) and anthropogenic activities (60-75%) (7). Natural or background sources of atmospheric mercury, mainly in the form of Hg^o, include emissions from volcanoes, soils, vegetation, and the ocean (8). It has been estimated that 20-30% of the current oceanic emissions originates from mercury mobilized by natural sources, with the remaining 70-80% derived from recycled anthropogenic Hg (7). Forest fires may emit Hg^o and some partially oxidized species (8). Estimates of contributions from natural sources are limited by our uncertainties regarding the amount of Hg in the pre-industrial environment as well as uncertainties in estimating the amount of anthropogenic Hg that is recycled by the ocean and terrestrial environment (9).

Modeling calculations estimate that anthropogenic emissions have tripled the concentration of mercury in the atmosphere and surface ocean over the last century (7). Anthropogenic sources of mercury include fossil fuel combustion (coal, oil, gas), waste incineration, chloro-alkali production, metal extraction processes, and cement production. These sources emit Hg^o, Hg_p, and Hg(II), which can cycle within the atmosphere and be deposited to ecosystems mainly as Hg(II)and Hg_p. The distance that anthropogenically mobilized Hg is transported prior to deposition is determined largely by the speciation of Hg that is emitted (8). Hg(II) and Hg_p deposit locally (≤50 km), while a significant fraction of Hg^o can be transported over long distances (≤10,000 km) and enter the global mercury cycle (8). This mercury is subsequently available for oxidation to Hg(II) in the troposphere and marine boundary layer, resulting in a global or "background" contribution of Hg(II) to mercury deposition (5,10).

Chemical speciation, climate, and meteorology influence the extent to which local and or global sources contribute to Hg deposition. The sub-tropical climate and complex meteorology of Southern Florida provide us with a rather unique environment to investigate Hg deposition. The annual rainfall volumes across Southern Florida range from 128-150 cm, with greater than 70% of the rainfall occurring during the rainy season (May-Oct.) (10). The summertime wet season in Southern Florida is characterized by the almost daily occurrence of tall convective thunderstorms (12-16 km) and daily ventilation of background air by the strong synoptic southeasterly winds associated with the North Atlantic trade winds. Findings from the Florida Atmospheric Mercury Study (FAMS) suggest that the annual deposition of Hg in Southern Florida is mediated by long range transport of Hg (mainly Hg(II) resulting from the oxidation of Hg^{o} in the global atmosphere) coupled with strong convective thunderstorm activity during the wet season. Model calculations indicate that long range transport accounts for 54-70% of the summertime rainfall Hg deposition, with the remaining 30-46% attributable to local anthropogenic Hg_p and Hg(II) emissions (10). It is important to recall that 60-70% of the Hg^{o} in the modern atmosphere results from industrial activity (7) and reductions in Hg deposition will likely require reductions in local and global Hg emissions.

References

(1) Fitzgerald, W.F., Mason, R.P., and Vandal, G.M. (1991) Water Air Soil Poll. 56, 745-768.
(2) Lindqvist, O., Johansson, M., Aastrup, A., Andersson, L., Iverfeldt, A., Meili, M., and Timm, B. (1991) Water Air Soil Poll. 55, 1-262.
(3) Hall, B. 1995 Water Air Soil Pollut. 80, 301-315.
(4) Munthe, J. (1992) Atmos. Environ. 26A, 1461-1468.
(5) Mason, R.P., Guey-Rong, S., and Lawson, Nichole. (2001) Abstract submitted to the Symposium on Methylmercury: Impacts on Wildlife and Human Health. Charleston, South Carolina, 2001.
(6) Lindberg, S.E., Brooks, S., Lin, C.J., Scott, K.J., Landis, M.S., Stevens, R.K., Goodsite, M., and Richter, A. (2002) Environ. Sci. Technol. 36, 1245-1256.
(7) Mason, R.P., Fitzgerald, W.F., Morel, F.M.M. (1994) Geochim. Cosmochim. Acta. 58, 3191.
(8) Porcella, D.B., Chu, P., and Allan, M.A. (1996) Inventory of North American Hg Emissions to the Atmosphere; Relationship to the Global Hg Cycle. In; Global and Regional Mercury Cycles: Sources, Fluxes, and Mass Balances, 179-190.
(9) EPMAP (Expert Panel on Mercury Atmospheric Processes) (1994) Mercury Atmospheric Processes: A Synthesis Report. EPRI/TR-104214. EPRI, Palo Alto, CA 94304, 23p.
(10) Guentzel, J.L., Landing, W.M., Gill, G.A., and Pollman, C.D. (2001) Environ. Sci. Technol.35, 863-873.

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Coastal Carolina University

Dr. Jane Guentzel obtained her doctorate in chemical oceanography from the Florida State University. She is currently an assistant professor of marine and environmental chemistry in the departments of marine science and chemistry at Coastal Carolina Univeristy. She has conducted studies related to mercury cycling in Florida and South Carolina. Research conducted in the Florida Everglades by Dr. Guentzel and her colleagues, Dr. Landing, Dr. Gill, and Dr. Pollman, focused on quantifying the depositional loading of mercury to the Everglades and determining the contributions from long range transport of reactive gaseous Hg and local sources.

Studies conducted in South Carolina, by Dr. Guentzel and her students, include: determining the atmospheric loading of mercury to northeastern South Carolina; quantifying gaseous elemental formation and evasion in coastal salt marsh ecosystems; and investigating relationships between mercury and dissolved organic carbon in groundwater from coastal flood plain regions.

Mercury in the Environment

Offshore Oil and Gas Sources
Dr. Jerry Neff
Battele-American Petroleum Institute

Influence of Offshore Oil and Gas Platforms on Environmental Risks of Mercury in the Gulf of Mexico

Mercury is a metal that is present naturally at very low concentrations in the atmosphere, water, sediments, and tissues of all plants and animals. Mercury is released into the environment from a wide variety of natural and human activities. It is present in the oceans as mercury metal and as inorganic and organic mercury compounds. An organic form of mercury, methylmercury, is the most toxic to animals. Living animals can absorb into their tissues some inorganic and organic mercury compounds from food and water. Methylmercury is toxic to terrestrial, freshwater, and marine organisms, if a large amount accumulates in their tissues. The main pathway for human exposure to methylmercury is through consumption of freshwater and marine fish.

Recent newspaper articles suggested that offshore oil and gas operations might be a secondary source of the more toxic, organic form of mercury in marine fish and shellfish in the Gulf of Mexico. This presentation reviews the scientific literature on the sources of mercury in the Gulf of Mexico environment and the potential contribution of offshore oil and gas operations to mercury levels in fish and shellfish consumed by man. The scientific literature shows that:

- Mercury from offshore oil and gas activities represents less than 0.5 % of the mercury inputs to the Gulf of Mexico;
- Most of the mercury from offshore platforms is in barite in drilling mud where it is present in an insoluble form that can not be absorbed into the tissues of marine animals;
- Mercury concentrations in sediments near offshore oil and gas platforms are low;
- Mercury concentrations in edible muscle tissue of shrimp and fish caught near offshore platforms are similar to those in the same species caught at locations far from platforms. This indicates that mercury in tissues of fish from the Gulf of Mexico is not coming from offshore platform discharges.

The Gulf of Mexico environment receives inputs of mercury from natural and human sources via rainfall, river inflows, runoff from land, and commercial activities in the coastal zone and offshore. Annually, an estimated 55,000 pounds of mercury is deposited from the atmosphere to the surface waters of the entire Gulf of Mexico. An additional 48,000 pounds of mercury enters the northern Gulf of Mexico in the freshwater inflow from the Mississippi River. By comparison, approximately 420 pounds of mercury enters the Gulf in drilling and production discharges under EPA permits. The amount of mercury entering the Gulf from offshore oil and gas operations is less than one-half of one percent of the amount entering the Gulf from the air and in Mississippi River water.

Drilling muds, used to drill wells, contain a natural mineral called barite that contains traces of mercury. During drilling, rock chips, called cuttings, are produced by the drill bit as it penetrates the earth. Drill cuttings may contain traces of mercury. Water may come to the surface with the oil and gas from a production well. This produced water also may contain traces of mercury. The Environmental Protection Agency permits drilling muds to be discharged to the ocean more than 3 miles from shore if the barite in the mud contains less than 1 part per million (ppm) mercury. Drilling muds used offshore in the Gulf of Mexico since imposition of the EPA limit on mercury in barite in 1993 have contained an average of about 0.5 ppm mercury, well below EPA's standard. Drilling muds and cuttings discharged to offshore waters of the Gulf during drilling of approximately 900 wells in 2001 contained about 340 pounds of mercury. An additional 80 pounds of mercury was discharged to the Gulf in treated produced water discharges from offshore platforms.

Some of the mercury entering the Gulf from all sources binds to suspended particles in the water column and settles with them to the bottom. Most nearshore sediments in the Gulf contain less than 0.1 ppm total mercury. Deep water, offshore sediments usually contain less than 0.05 ppm mercury. Most concentrations of mercury in sediments near offshore platforms are 0.2 ppm or less. Sediments at only one platform site, of more than 30 platforms surveyed, contained more than 1 ppm mercury, and elevated mercury concentrations were restricted to sediments within about 100 ft of the platform. In this atypical drilling operation, drilling muds and cuttings were discharged directly to the sea floor to prevent any possibility of damage to nearby coral reefs. With one exception, this was the only site, among the more than 30 surveyed, where surface sediments near the discharge contained more than the sediment quality guideline, the Effects Range Median (ERM), of 0.7 ppm mercury. A few sediment samples collected near a platform off Galveston, TX in 1978-79 contained slightly more than the ERM concentration of mercury. These samples were analyzed by less reliable methods than those used today and reported concentrations may be higher than true values. These results show that ocean discharge of drilling muds and cuttings does not result in environmentally significant mercury contamination of sediments near platforms.

Most of the mercury entering the Gulf of Mexico is inorganic mercury and metallic mercury (the silvery liquid in a mercury thermometer). Inorganic and metallic mercury are only moderately toxic and they are not passed efficiently through the marine food chain to man. However, some species of bacteria that live in sediments and ocean water containing very low concentrations of oxygen are able to convert some of the inorganic mercury dissolved in water to toxic methylmercury through a process called methylation. Mercury methylation also occurs in the organic-rich sediments of coastal salt marshes and wetlands, such as the Everglades in south Florida.

The mercury in drilling mud is in a solid, insoluble form. Bacteria have only a very limited ability to absorb the mercury found in barite; therefore, it is not likely to be methylated. Marine plants and animals cannot accumulate the insoluble mercury from barite in in their tissues. Therefore, little or none of the mercury from drilling mud and cuttings is methylated and it does not bioaccumulate in the marine food chain.

Marine animals are not able to convert inorganic mercury to methylmercury. However, they are able to accumulate inorganic mercury and methylmercury directly from the water or by ingestion of food or sediments. Most of the methylmercury in the edible muscle tissues of fish comes from their food. Concentrations of mercury in tissues of marine shellfish and finfish from the Gulf of Mexico vary widely in different species. However, concentrations in edible tissues of fish and shellfish from the Gulf of Mexico are comparable to those in the same or similar species from other marine environments in the U.S. and abroad that do not have offshore oil and gas operations. Mercury concentrations are highest in muscle tissues of large, predatory ocean fish, such as swordfish, sharks, and king mackerel. Mercury concentrations usually are low in soft tissues of shellfish, such as oysters, crabs, and shrimp, and bottom fish, such as flounder, red snapper, and mullet.

There have been several studies to measure the concentrations of mercury in tissues of marine animals in the vicinity of offshore platforms. Fish collected near the Gulf platform where mercury concentrations in sediments were above 1 ppm contained slightly elevated mercury concentrations in their livers. Whole soft tissues of edible shrimp from the vicinity of this platform contained low concentrations of mercury, similar to concentrations in shrimp from throughout the Gulf. In all cases where measurements were made, mercury concentrations in edible muscle tissues of fish near platforms were similar to concentrations in muscle tissue of the same or similar species well away from and out of the influence of the platforms. Shellfish from the vicinity of platforms contained low concentrations of mercury, similar to those in shellfish collected away from offshore platforms. This distribution of mercury in marine fish and shellfish populations throughout the Gulf of Mexico indicates that the mercury in the edible tissues of these seafoods is not derived from offshore platform discharges.

Jerry Michael Neff, Ph.D.
Battelle Memorial Institute

Education
B.A. Biology, Antioch College - 1963
Ph.D. Zoology, Duke University - 1967

Qualifications
Dr. Neff is an internationally recognized authority on the fate and effects of petroleum hydrocarbons, oil well drilling fluids, and produced waters in marine freshwater, and terrestrial environments. During the past 30 years, he has performed more than 100 research and monitoring programs on these and related subjects for government and industrial clients worldwide. He has written three books dealing with petroleum and aromatic hydrocarbon contamination of aquatic environments and a major literature review on drilling fluids in the marine environment. He has been a member of four review panels of the U.S. National Academy of Sciences, the first dealing with Fate and Effects of Drilling Mud and Cuttings in the Marine Environment, the second dealing with marine oil spills; the third was an assessment of marine environmental monitoring in the Southern California Bight. He currently is serving on the Committee to Review the Oil Spill Recovery Institute's Research Programs.

He also was a member of the Steering Committee and a Technical Review Author for the Federal COPRDM Committee on "Predictive Assessment for Design of Studies of Long-Term Effects of OCS Oil and Gas Development." This assignment resulted in the authorship of two chapters of a book, published by Elsevier Applied Science Publishers, on "Long-Term Environmental Effects of Offshore Oil and Gas Development", the first dealing with development activities potentially causing long-term environmental effects, and the second with the fate and effects of drilling muds, cuttings, and produced water in the marine environment. He also assisted to assemble and edit a book on the effects of offshore oil and gas development on the marine environment of Australia for the Australian Petroleum Exploration Association. This book won the first prize for new technical books from the Australian Minerals and Energy Environmental Foundation. The book is the most current and comprehensive review of the effects of drilling fluids, produced water, oil spills, and industry facilities on the marine environment. He recently completed a book on "Bioaccumulation in Marine Organisms. Effect of Contaminants from Oil Well Produced Water" that will be published by Elsevier Science Publishers in April 2002.

Dr. Neff has performed extensive research for the oil industry, the U.S. Federal government, and foreign governments on the marine environmental fate and effects of heavy metals and petroleum hydrocarbons from offshore drilling and production operations, and clean ballast water discharges from tankers He has participated in Natural

Resource Damage Assessments for several oil spills, including the *Amoco Cadiz* crude oil spill in France, the *Exxon Valdez* crude oil spill in Alaska, the *Haven* oil spill off Genoa, Italy, the Trecate oil spill in rice fields north of Milan, Italy, and the *Seki* oil spilll in the United Arab Emirates. The focus of much of this research was on the bioavailability and bioaccumulation by aquatic organisms of chemicals, particularly PAHs from permitted and accidental oil industry discharges. The work also included studies of the sources (including natural seeps) and weathering of crude and refined oil in the environment. Several of the oil spill studies included assessments of injury and compensatory damages to commercial and recreational fisheries resources in areas affected by the spills.

Dr. Neff has published more than 150 scientific articles and two books. A few publications related to environmental fates and effects of drilling fluids and produced water are listed here.

Books

Neff, J.M. 2002. Bioaccumulation in Marine Organisms: Effect of Contaminants from Oil Well Produced Water. Elsevier Science Publishers, Amsterdam. 452 pp.

Swan, J.M., J.M. Neff, and P.C. Young (Eds.). 1994. Environmental Implications of Offshore Oil and Gas Development in Australia - The Findings of an Independent Scientific Review. Australian Petroleum Exploration Association, Sydney, Australia. 696 pp.

Recent Peer-Reviewed Publications

Neff, J.M., T.C. Sauer, and A. Hart. 2001. Monitoring Polycyclic Aromatic Hydrocarbon (PAH) Bioavailability Near Offshore Oil Wells. Pages 160-180 In: B.M. Greenberg, R.N. Hull, M.H. Roberts, Jr., and R.W. Gensemer, Eds., Environmental Toxicology and Risk Assessment: Science, Policy and Standardization – Implications for Environmental Decisions (Tenth Volume), ASTM STP 1403, American Society for Testing and Materials, Philadelphia.

Neff, J.M., S. McKelvie, and R.C. Ayers, Jr. 2000. Environmental Impacts of Synthetic Based Drilling Fluids. MMS OCS Study 2000-064. U.S. Dept. of the Interior, Minerals Management Service, Gulf of Mexico OCS Region, New Orleans, LA. 119 pp.

Neff, J.M. and T.C. Sauer, Jr. 1996. An ecological risk assessment for polycyclic aromatic hydrocarbons in produced water discharges to the western Gulf of Mexico. Pages 163-176 In: M. Reed and S. Johnson, Eds., Produced Water 2: Environmental Issues and Mitigation Technologies. Plenum Press, NY.

Neff, J.M. and T.C. Sauer, Jr. 1996. Aromatic hydrocarbons in produced water: bioaccumulation and trophic transfer in marine food webs. Pages 355-366 In: M. Reed and S. Johnson, Eds., Produced Water 2: Environmental Issues and Mitigation Technologies. Plenum Press, NY.

Current and Proposed Mercury Science and Education Projects

Fish Advisories in Alabama
Dr. Neil Sass
Alabama Department of Public Health

A Healthy, Informed Choice: Contaminants in Fish

There is variability in the health value of eating some fish. Part of this is due to the chemical composition of the fish itself, the protein, fat, vitamin, and/or mineral content of the fish. There is another aspect of fish composition that should be examined to determine its value as a healthy or unhealthy source of nutrition. This other aspect is the level of contaminants that may be present in the fish.

If fish live in a clean body of water, their health should be good and they are good to eat. If fish live in a body of water that has higher than normal contaminants, their health might not be good and they should not be eaten. The longer fish live in contaminated water, the more likely it is that they are contaminated. Since fish cannot get rid of all the poisons or toxins from their bodies, their bodies end up storing them. The highest concentrations of toxins, like some pesticides, mercury and polychorinated biphenyls (PCBs), are found in the fat and liver of fish.

Contaminants in the environment can be natural or man-made. Some of the materials we call contaminants are actually beneficial chemicals that have been distri buted in the environment intentionally, like pesticides that have been applied on crops. Others are by-products of processes which have escaped into the environment, e.g., dioxins and furans from the production of bleach kraft paper. A substance like mercury, although a natural product, escapes into the environment in large amounts through the burning of coal in commercial power plants and factories. Other materials, like polychlorinated biphenyls (PCBs) enter the environment as waste materials put into discard piles at factories. The PCBs then are subject to the effects of wind and rain in becoming disbursed throughout the environment. These contaminants can find their way into waters in the State. Contaminants can be taken up by plants or fish or other organisms (e.g., crayfish) in the water. Fish develop measurable levels of contaminants depending on the content of contaminant in the materials on which the fish feed. Some of these can accumulate in the fish over time.

As a rule, eating fish is a healthy choice. Fish should be included in every balanced diet. However, while most Americans need to include more fish in their diet, care must be taken in in choosing and preparing fish. Some fish may actually cause harm. Alabama Department of Public Health, in conjunction with the Alabama Department of Environmental Management, surveys fish in varying watertbodies across the state to determine whether or not conditions in the waters of the state are changing. Increases of specific contaminants in specific waterbodies will result in the issuance of consumption advisories to inform the public of dangers involved in consuming certain species of fish from specific waterbodies. To make sure you and your family get all the health benefits from fish, you should know:

- Which type(s) of fish you're eating
- Where these fish were caught
- How the fish is prepared and cooked
- How much fish is safe to eat

Through awareness of these elements, individuals can increase the health content of their diet without significantly increasing their exposure to potentially hazardous contaminants in fish.

Neil L. Sass, Ph.D.

State Toxicologist and State Counterterrorism Coordinator
Alabama Department of Public Health, Montgomery, AL

Dr. Sass received a B.S. in Biology (Wake Forest College, Winston-Salem, NC), a M.S. in Physiology and a Ph.D. in Biochemistry (West Virginia University, Morgantown, WV), and was a Fellow in Applied Behavioral Science and received a M.S. in this field from The Johns Hopkins University, Baltimore, MD, a department in which he also held a teaching appointment. Dr. Sass' first career was in Federal service, initially as a research toxicologist in the U.S. Army conducting programs on development and testing of military peculiar chemicals (Edgewood Arsenal, MD) and later as Chief of Clinical Investigations, Wm. Beaumont Army Medical Center, El Paso, TX. He transferred his commission from the U.S. Army to the U.S. Public Health Service and was assigned to various positions in the Food and Drug Administration as well as being detailed to various other organizations within the Department of Health and Human Services.

His assignments at the Food and Drug Administration (FDA) included serving as a regulatory toxicologist specializing in the evaluation of toxicity of food and/or color additives and cosmetics, and directing Federal nutrition programs for Public Health Service agencies. Dr. Sass spent 19 years assigned as the Special Assistant to the Director, Center for Food Safety and Applied Nutrition (CFSAN). In this role, he was responsible for developing and implementing food safety policy, including testifying before various Congressional committees to further the implementation of these policies.

He was also Chairperson, CFSAN IACUC, and was primarily responsible for redesigning the protocol development and review process to render it user and administration friendly and efficient. Dr. Sass was also Director, Division of Toxicological Research, CFSAN/FDA. In this role, he redesigned the program with primary emphasis on the development of alternatives to animal studies. He was also a member of the federal Interagency Coordinating Committee for the Validation of Alternative Methods (ICCVAM) and the Assistant Secretary for Health's Environmental Health Policy Committee (EHPC). The charter for the ICCVAM group is to achieve acceptance of national standardization of test methods for alternatives to animal test procedures. This goal is extended to achievement of international standardization of methods between nations and industry organizations.

Dr. Sass also represented the United States on a working group assembled by the OECD to develop guidance on the humane use of animals in safety testing. As a member of EHPC, Dr. Sass served with the heads of Public Health Service Agencies to negotiate and address issues which spanned the jurisdiction of multiple PHS agencies. This role involved the determination of research needs to answer questions critical to the decision making processes in each agency.

Dr. Sass was active in Applied Research Ethics National Association (ARENA) activities and affairs. He served as the alternate member of the ARENA Council for two years, and as Arena Council representative for the Mid-Atlantic Region for six years, as well as serving as the program chair and co-chair for the annual ARENA Meeting for four years.

In 1999, Dr. Sass retired from active federal service and accepted a position as State Toxicologist and State Counterterrorism Coordinator, operating out of the Alabama Department of Public Health, in Montgomery. In this capacity, Dr. Sass has the responsibility of overseeing the possible impact on the health of Alabama citizens from assorted sources of contaminants, e.g., from inhalation of materials due to a leak from an industrial or transportation source, or from ingestion through contaminants entering the food/water supply.

As Counterterrorism Coordinator, Dr. Sass directs activities within the Alabama Department of Public Health designed to increase the level of preparedness of the medical assets within the state should a catastrophic event occur, be it of natural, accidental, or terrorist origin. In this role, Dr. Sass works closely with the Alabama Emergency Management Agency, the Alabama Department of Public Safety, and the Federal Emergency Management Agency to make certain possible contingencies surrounding an event have been considered and appropriately addressed with minimum duplication of efforts.

Current and Proposed Mercury Science and Education Projects

Fish Monitoring Programs in Alabama

Mr. Fred Leslie
Alabama Department of Environmental Management

Regular monitoring of fish in Alabama for mercury contamination was initiated in 1970 by ADEM's predecessor, the Alabama Water Improvement Commission (AWIC). The monitoring was conducted in Cold Creek Swamp and adjacent Mobile River in response to concerns of mercury contamination from area industries. Monitoring of fish by the AWIC/ADEM continued in this area from 1970 to the present.

In 1991, the ADEM expanded its Fish Tissue Monitoring Program to provide statewide monitoring. The Program expansion was in response to concerns regarding mercury and other bioaccumulative contaminants in fish, and national emphasis by the U.S. Environmental Protection Agency (USEPA). The expanded program exists as a cooperative arrangement between the ADEM, the Alabama Department of Public Health (ADPH), the Alabama Department of Conservation and Natural Resources (ADCNR), and the Tennessee Valley Authority (TVA). With increasing awareness during the 1990's of mercury contamination in fish, the ADEM also joined other states in this region as a member of the Southern States Mercury Task Force to share information and expertise in determining necessary action.

Through the Program, fish are collected from all major reservoirs and streams in Alabama over five (5) year periods in a basin rotation cycle. Each year, sites in waterbodies outside the scheduled basin are also included for monitoring as needed and as resources allow. Since 1991, 596 composite samples comprised of several thousand fish have been collected from over 230 sites and analyzed for mercury. In addition, individual analyses of many fish have also been conducted. All samples are analyzed by the ADEM Environmental Laboratory for mercury and other contaminants with the potential to bioaccumulate. Analytical data is provided to the Alabama Department of Public Health (ADPH) for review, with the ADPH using FDA action levels of 1.0 parts per million (ppm) mercury in fish tissue for issuance of advisories.

Recently, analyses of mercury concentrations in fish samples from 30 Alabama coastal estuary sites was conducted by ADEM, USEPA, and ADCNR for the Coastal 2000 Program. No fish collected for this Program exceeded FDA levels for mercury. During 2001, fish tissue samples were collected by ADEM from 34 sites in the state and analyzed for mercury concentrations, with a total of 407 fish collected. Eighteen of these sites were in the Mobile Bay area, where the greatest number of ADPH consumption advisories in Alabama currently exist. Bass collected from the Escatawpa River and Styx River in 2001 exceeded FDA guideline levels, though bass collected from these locations in 1995 did not. As in previous years, bass collected during 2001 from Fowl River and Fish River exceeded FDA guidelines for mercury. In addition, two fish in a sample of six largemouth bass from Chickasaw Creek and one fish in a sample of six largemouth bass from the Tensaw River exceeded the guideline levels. Bass collected from Bay Minette Creek in 2001 did not exceed FDA levels, while those collected in 1997 and in 1998 did.

Future monitoring activities include sample collection in the Warrior and Cahaba River basins in 2002, resampling locations where fish exceeded FDA levels for the first time in 2001, resampling of south Alabama locations not sampled in several years, as well as targeting the remaining areas in south Alabama where fish have not been collected for the ADEM Program. Future sampling of marine and estuarine species depends in part on the outcome of ongoing negotiations at the state and federal level.

Fred Leslie

Chief of the Aquatic Assessment Unit, Field Operations Division
Alabama Department of Environmental Management

Fred was born in Macon, GA, on September 14, 1958 and grew up in rural Twiggs County, GA. He graduated with honors from Georgia College in June 1983, receiving a B.S. in Biology and Environmental Sciences. In January 1984, he enrolled in Auburn University as a graduate student in the Department of Fisheries and Allied Aquacultures under Dr. David Bayne. He worked as a graduate research assistant in the Rivers and Reservoirs Laboratory until graduating with an M.S. in Fisheries Science in June 1989. In October 1989, he began work for the Alabama Department of Environmental Management and was primarily involved in the initiation and development of the Department's Reservoir Water Quality Monitoring Program, Fish Tissue Monitoring Program, and in various stream water quality studies. Currently, he is Chief of the Aquatic Assessment Unit of the Field Operations Division, which is primarily responsible for all surface water quality monitoring in the state. Fred currently resides in Montgomery, AL and is married to the former Lucy Quina of Pensacola, FL. Most of his free time is spent fishing, hunting, canoeing/kayaking, scuba diving, reading, and backpacking.

Current and Proposed Mercury Science and Education Projects

Survey of the Occurrence of Mercury in Fishery Resources of the Gulf of Mexico
Dr. Frederick Kopfler
EPA Gulf of Mexico Program

Survey of the Occurrence of Mercury in Fishery Resources of the Gulf of Mexico

An understanding of methylmercury concentrations in edible fish and shellfish tissues is the foundation for public health risk assessments. A regional database - the Gulfwide Mercury in Tissue Database - was created with recent GIS-based tissue monitoring data from the five Gulf States (Florida, Alabama, Mississippi, Louisiana, Texas), the USEPA EMAP program, the NOAA NOS National Status and Trends program, and the NMFS GulfChem study. The study area for database analysis included waters within the 94 USGS 8-digit hydrologic unit code watersheds that comprise the major estuarine drainage areas of the Gulf of Mexico, and the nearshore and blue waters of the Gulf of Mexico. We present the occurrence of mercury in 24 estuarine/marine species and species groups (and 3 size classes of king mackerel) commonly harvested in the study area. Species-specific maps show relative mercury concentrations at tissue sample sites across the Gulf of Mexico. Based on input from the Gulf of Mexico Program's Mercury Project Advisory/Review Committee, we present recommendations for Gulf-region tissue monitoring program enhancements.

Fred Kopfler
EPA-Gulf of Mexico Program

Fred Kopfler is a native of Louisiana. He received a BS in chemistry from Southeastern Louisiana University in Hammond and a Masters and PhD in Biochemistry and Food Science and Technology from Louisiana State University.

After a two year post-doctoral at the US Department of Agriculture's Protein Pioneering Laboratory in Philadelphia, PA, he worked at the US Public Health Laboratory at Dauphin Island, Alabama investigating the pesticide and trace metal contaminants in shellfish.

When the US EPA was formed, he became one of the charter employees and moved to Cincinnati, OH where he worked until 1989 on the health effects of chemical contaminants and disinfection by-products in drinking water.

In 1989 he joined the newly formed Gulf of Mexico Program with offices at Stennis Space Center in Mississippi. At the Gulf of Mexico Program he has worked on public health issues associated with the use of the Gulf's waters and its seafood products including chemical contaminants of seafood; sewage pollution of shellfish growing waters and recreational waters; and harmful algal blooms.

Current and Proposed Mercury Science and Education Projects

Methylmercury in Marine Fish: A Gulf-Wide Initiative
Mr. Ron Lukens
Gulf States marine Fisheries Commission

The Gulf States Marine Fisheries Commission, during its Annual Fall Meeting in October 2001, began an initiative to investigate the need for a Gulf-wide survey to collect fish tissue for analysis of mercury content, and to determine the need for convening appropriate federal and state agency and industry representatives to discuss developing compatible fish consumption advisory levels and advisory language.

A report was provided to the Commission during its Annual Spring Meeting in March 2002. That report provide the Commissioners with background information on sources of mercury, biological processes and implications, public health concerns, and federal and state actions regarding mercury in fish. That report also contains seven recommendations, listed below, which were presented to the Commissioners.

1) The Gulf States Marine Fisheries Commission, in cooperation with the appropriate state and federal agencies, should encourage and facilitate the development of a Gulf-wide survey to collect fish tissue for mercury analysis. The survey should collect tissue from species commonly consumed by the public from commercial sources and caught and consumed by recreational anglers, and
2) The Gulf States Marine Fisheries Commission, in cooperation with the appropriate state and federal agencies, should encourage and facilitate the establishment of sufficient capacity for timely analysis of mercury tissue samples collected by the Gulf-wide survey,
3) The Gulf States Marine Fisheries Commission should work with the Gulf of Mexico Program, administered by the U.S. Environmental Protection Agency, to facilitate convening appropriate state and federal agency representatives to consider establishing consistent seafood consumption advisories and establishing common advisory levels for mercury in fish tissue, and
4) The Gulf States Marine Fisheries Commission, in cooperation with the appropriate state and federal agencies, should encourage and facilitate the development of an education and outreach strategy, including the development of new, and more effective education and outreach materials, to educate the general public about the risks associated with consumption of seafood that may be contaminated with mercury,
5) The Gulf States Marine Fisheries Commission, in cooperation with the appropriate state and federal agencies, should encourage and facilitate the development of a fish consumption survey of recreational anglers,
6) The Gulf States Marine Fisheries Commission, in cooperation with the appropriate state and federal agencies, should encourage and facilitate the establishment of a common, centralized database on mercury in marine fish tissue, and
7) Recognizing that methylmercury contamination of fish tissue is not confined to the Gulf of Mexico region, the Gulf States Marine Fisheries Commission should encourage similar initiatives as embodied in this report for the Atlantic and Pacific Coasts.

The Commissioners tabled definitive action on the report, pending additional staff work to develop more detail for the recommendations. Included will be appropriate agency roles, effort involved, and associated costs if the recommendations were to be implemented. The Commission staff and the Steering Committee are currently compiling the necessary information regarding each recommendation and will be providing the information to the Commissioners at the Annual Fall Meeting in October 2002.

Ron Lukens
Assistant Director,
Gulf States Marine Fisheries Commission, Ocean Springs, MS

Ron Lukens is a marine fisheries biologist and has worked in the fields of research, aquaculture, and policy for marine fisheries in the Gulf of Mexico region since 1975. Since 1990, Ron has served as Assistant Director of the Gulf States Marine Fisheries Commission (GSMFC). The GSMFC coordinates interstate and state-federal research, data collection and management, regulation, legislation, and policy among the five states in the Gulf of Mexico and the federal government.

The following describes Ron's work experience:

- fisheries science and policy, working as a marine bio-technician, an intensive culture striped bass aquaculture scientist, a field technician collecting biological samples, and finally a fishery administrator helping to develop regional and national fisheries policy,

- biodiversity and genetics issues, involved as a writer and planner in the development of the Gulf Sturgeon Recovery/Management Plan, and setting protocol for the collection and analysis for Gulf sturgeon and striped bass genetics samples,

- marine environmental education, working as a marine fisheries specialist for the Mississippi Sea Grant Advisory Service, developing and presenting adult education programs regarding commercial and recreational fishing activities,

- ecosystems monitoring and limited GIS experience, assisting in the development of a GIS data base for the Pascagoula, Leaf, Chickasawhay River System,

- fisheries data base design and integration of data bases, through the development of the Fisheries Information Network (FIN), which is a state-federal cooperative fishery dependent data collection and management program. Through the FIN, various state and federal data bases are being integrated to form a large diverse fisheries data base, and standard protocols and formats have been developed to guide future data collection and management efforts,

- and the use of the Internet for environmental communications and research, through the implementation of a communications network of fisheries scientists, primarily associated with management of the FIN.

- directly involved in invasive species work, currently serving on the Gulf of Mexico Regional Panel of the Aquatic Nuisance Species (ANS) Task Force and as an Ex-Officio member of the ANS Task Force. In addition, Ron serves on the National Invasive Species Advisory Committee.

Current and Proposed Mercury Science and Education Projects

Gulf-Wide Fish Monitoring Program
Dr. Spencer Garrett
National Marine Fisheries Service

Synoptic Survey of Total Mercury in Recreational Finfish in the Gulf of Mexico

The public health ramifications of mercury (Hg) in fish is a complex issue of numerous dimensions. There are professional differences of opinion on what the allowable tolerance (guideline) in fishery products should be. Methylmercury is an ecotoxicant that bio-accumulates in marine seafood species. There are natural sources and anthropogenic sources of mercury released into the marine environment that through bacterial processes becomes the bio-accumulating ecotoxic Methylmercury. Methylmercury binds to proteins in living organisms and is passed up the food chain where the Methylmercury can reach dangerous levels in certain seafood species.

The strategy to protect the public against ingesting unsafe amounts of Methylmercury has been premised on the following:

1. the average consumer eats approximately 15 pounds of seafood per year;
2. the primary source of seafood for the average consumer is from commercial harvested species; and
3. the amount of seafood consumed can be used to back calculate an acceptable level of methylmercury ingestion,
4. therefore, seafood below 1.0 part per million (ppm) Methylmercury is generally acceptable.

Various consumption advisories have been issued by FDA and EPA encouraging the public, especially women of child bearing age and children to consume seafood species that are low in Methylmercury, and to avoid eating Swordfish, Shark, Tile Fish, and King Mackerel. As Methylmercury can adversely affect the neurological development of fetuses and small children at low doses, women of child bearing age and children are of particular concern. EPA and FDA recognize and have so indicated in 1996, that "...FDA's action levels ensure a safe food supply for consumers of commercial fish, they may not be appropriate levels for ensuring the safety of those who consume locally caught fish...." and therefore various fish consumption advisories have been issued by states to take into account local conditions and local consumption patterns.

There may be an unrecognized portion of the public that consumes seafood in excess of 15 pounds per year, and they also consume large quantities of seafood that are harvested for personal consumption. In particular, subsistence, commercial, and marine recreational fishermen and their families may be at increased risk of exceeding the FDA Methylmercury consumption guidelines as they may be consuming seafood well in excess of 15 pounds per year, and they may be consuming non-commercially harvested seafood that exceeds the FDA's 1.0 ppm Methylmercury monitoring and restrictions. Therefore, subsistence, commercial, and marine recreational fishermen and their families represent a new sub-population of the seafood consuming public that could likely require additional informational safeguards in order to protect them against excessive Methylmercury ingestion via seafood.

The Methylmercury levels in commonly available commercially harvested seafood species are fairly well known. However, the Methylmercury levels in seafood species not commonly available through commercial sources are less well known. Since the development of consumption advisories for the subsistence, commercial, and marine recreational fishermen and their families should be based on sound science, data on the mercury levels in the seafood species this sub-populations consumes will be collected. NMFS' National Seafood Inspection Laboratory and EPA's Gulf of Mexico Program plan to carry out a synoptic survey analyzing 2,500 samples in 2002-2003 to collect preliminary data on the mercury level for selected popular marine recreational seafood finfish, and to provide data for later more extensive Gulf-wide mercury in seafood survey designs if needed.

The synoptic survey will be carried out in three parts:

1. Estuarine Sampling and Modeling: Selected estuarine finfish will be collected from estuaries with varying degrees of mercury contamination. Previously collected mercury data in oysters from these estuaries will be modeled against the finfishes mercury levels. If the modeling finds that the low oyster's mercury levels can be used as a surrogate for the finfishes mercury levels, then the 31 estuaries of the Gulf Coast could be modeled for their finfishes mercury levels using NOAA's Mussel Watch's previously collected oyster mercury data for the 31 Gulf estuaries.

2. Reef and Rig Sampling: Selected reef finfishes will be collected from oil and gas drilling rigs and non-oil and gas drilling rig reefs. The samples will be tested to determine if a statistical difference exists in the mercury level in the reef finfish caught near the drilling rigs versus those caught near the non-rig reefs. If no difference is observed, then a generic Gulf-wide modeling of the mercury levels in reef fish could be possible. Conversely, if the mercury levels in the reef finfish taken from the vicinity of the rigs are statistically higher than those taken at non-rig reefs, then additional surveys will be required.

Migratory Species Sampling: Selected highly migratory finfish species will be collected from off the Florida Gulf and Texas Coasts. The samples will be tested to determine if a statistical difference exists between the fishes taken from these geographic regions of the Gulf. If no difference can be determined, then a generic Gulf-wide modeling of the mercury levels in these species could be possible. Conversely, if a difference is observed, then additional surveys would be required.

NMFS anticipates that the synoptic survey will, at a minimum, provide valuable data that will allow for an assessment of the scope of sampling required to adequately cover the marine recreational finfishes of the Gulf of Mexico. Such data is needed to support the development of consumption advisories for the general public, and especially for the subsistence, commercial, and marine recreational fishermen sub-population that is believed to be at the highest risk presently.

It should be understood that this NMFS Synoptic Survey deals, in a limited manner, with only one-half of the information needs that address the exposure component of a mercury risk assessment. The other necessary component of the exposure risk assessment is the need for consumption studies for the recreational fisheries and/or commercial fishers (who consume portions of their catch) relative to the species that may be identified as containing elevated mercury levels.

E. Spencer Garrett
Director, National Seafood Inspection Laboratory
Pascagoula, MS

- E. Spencer Garrett serves as Director of the National Seafood Inspection Laboratory in Pascagoula, Mississippi which is recognized as a premier seafood public health and information transfer center and provides scientific services to the National Oceanic and Atmospheric Administration/National Marine Fisheries Service's Seafood Inspection and Certification Program. He has received national and international recognition for his strong scientific, technical and administrative abilities in developing, executing, and evaluating complicated food safety, quality, and food hygiene programs. He serves as his Agency's representative on the Interagency Mercury Working Group of the Executive Office of the President's Office of Science and Technology.

- He received his under graduate and graduate degrees in microbiology from the University of Southern Mississippi and currently serves as the North American Representative on the Board of Directors of the International Association of Fish Inspectors, has served as the USA Delegate to the Codex International Food Standards Programme Committee on Food Hygiene sponsored by the Foreign Agriculture Organization and the World Health Organization of the United Nations and currently serves on the prestigious National Advisory Committee on Microbiological Criteria for Foods.

- Author or coauthor of more than 200 scientific and technical papers and/or feasibility studies and presentations dealing with measures to improve consumer protection in the consumption of fishery products. Spencer is among the most contemporarily published authors of the HACCP concept. He presently serves as his agency's representative to the National Academy of Science "Food Forum" and the Interagency Committee on Human Nutrition and is his Agency's principle spokesperson on seafood safety issues.

Selected Mercury Related Research

Current Research into Mercury Control from Coal-Fired Power Plants

Dr. Larry Monroe
Southern Company Services, Inc.

In December 2000, EPA announced their intention to require coal-fired utility boilers to control mercury emissions. Since mercury concentrations in flue gas are one million times lower than that for the other pollutants of interest, control of these emissions will be quite a technical challenge. Various approaches to controlling mercury emissions will be discussed, and the commercial potential of each technology will be noted. Several exciting new processes have been proposed and these will also be discussed. Finally, the probable time table and several proposed emission levels will be presented.

Dr. Larry Monroe
Southern Company Services, Inc.

Dr. Larry Monroe is the Program Manager for Pollution Control Research for Southern Company Generation and Energy Marketing. Based in Birmingham, Dr. Monroe supervises a team which develops new pollution control technologies, evaluates new processes, and solves problems for existing controls for Southern's fossil-fired generation.

Before joining Southern Company, Dr. Monroe managed the Combustion Research Facility for Southern Research Institute, also in Birmingham. This facility is a 1/1000 scale pilot coal furnace, and is owned by Southern Company.

Dr. Monroe holds a B.S. in Chemical Engineering from Auburn University and a Ph.D., also in Chemical Engineering, from MIT.

Selected Mercury Related Research

Distribution of Mercury in the Mobile River Basin in Relation to Land Use

Dr. Kimberley Warner
University of Alabama Department of Biological Sciences
Co-authors: Jean-Claude Bonzongo, Eric Roden, W. Berry Lyons, Milt Ward, Indrajeet Chaubey, and Hobson Bryan.

In the past decade, mercury (Hg) concentrations above levels that could pose human health risks have been measured in predatory fish from many rivers and reservoirs in the southeastern region of the United States. This region, and mainly its Coastal Plain portion, may be particularly vulnerable to Hg contamination in aquatic food chains, due to the coexistence of both natural and human-imposed conditions which favor the production and accumulation of methyl-Hg. Several specific factors are hypothesized to contribute to the development of such conditions, including: (1) nutrient loading from certain land-use activities and increased sedimentation above water impoundments develop conditions favorable for methyl-Hg production. (2) Increased sulfate loading from energy resource extraction operations result in increased methyl-Hg production. (3) Abundant wetlands within the Mobile-Alabama River Basin (MARB) contribute to methyl-Hg loads downstream and in fish. (4) Fish tissue levels of methyl-Hg are related to levels of Hg in river waters and to net rates of methyl-Hg production in sediments. The objectives of this study were to: (1) to determine levels and speciation of Hg in different compartments of various aquatic systems in the MARB; (2) to investigate the linkage between land use types or the presence of wetlands and microbial processes associated with methyl-Hg production; (3) to use GIS to represent spatially arranged data and ultimately to predict Hg levels in fish; and (4) to use a participatory approach to environmental decision-making to ameliorate conflict, and achieve an effective public understanding and support for Hg policy. This report will address preliminary results and progress on the science aspects of this interdisciplinary research project conducted by the University of Alabama Center for Freshwater Studies.

The first phase of the project involved a wide survey of Hg distributions in largemouth bass, water and sediment at 52 sites representing various hypothesized impact factors (e.g. wetlands, agriculture, dams). Various sediment chemistry and water quality parameters were measured in conjunction. A total of 96 fish samples were taken from 51 out of 52 sites. While we attempted to collect 2 fish at each site, at least two fish were sampled at 43 sites and only one fish at the remaining 8 sites. The concentrations of Hg in these 96 fish spanned over 2 orders of magnitude, from 0.02 to 2.8 ppm (mg kg^{-1} wet tissue). Mean and median concentrations were 0.45 and 0.32 ppm, respectively, with the mean close to that of the National Mercury Survey (0.39 ppm) reported for AL largemouth bass. Twelve percent (12%) of fish had Hg concentrations $\geq$ 1 ppm, the level at which consumption advisories are posted in Alabama. Average fish Hg concentrations were $\geq$ 0.5 ppm at 21 (41%) of the sites. Coefficients of variation in fish Hg concentrations from any one site were usually high, averaging 68%. In many cases, Hg concentration was higher in the smaller of two fish. Total-Hg and methyl-Hg concentrations in water were low and ranged from 0.2-3.8 and 0.01-1.5ng L^{-1}, respectively. This results in an average -log bioconcentration factor of 5.6 for Hg between fish and water. Hg speciation determinations in sediments are still underway. The annual flux of aqueous total-Hg from the MARB to Mobile Bay is estimated at 138Kg yr^{-1}.

No obvious trends were apparent between fish Hg concentrations and projected impact factors, likely due to the large within-site variability noted above. However, some variability within certain impact factors could be explained by other factors. For example, fish Hg concentrations in wetland sites were positively related to watershed area and inversely related to water depth above dams, for dam impacts. Fish Hg concentrations were also found to be a weak negative function of water pH. Aqueous total-Hg and many sediment and water quality parameters were found to be a significant function of land use.

The second phase of the project involved in-depth investigations examining controls on Hg transformation and bioaccumulation. We focused on one pool (Demopolis) containing 4 sites with different impact factors: (i) dam, (ii) agricultural, (iii) wetland, and (iv) open river. Potential rates of microbial Hg methylation and methyl-Hg demethylation were determined in sediments. The ratio of methylation to demethylation rates was positively related to the percent of methyl-Hg formed in native sediments. Concentrations of Hg (127-393ng g dry sediment^{-1}) and methyl-Hg (0.13-2ng g^{-1}) in native sediments increased in the following order of impacts: open river < dam < agriculture < wetland. The percentage of methyl-Hg produced in the sediments was a positive function of sediment iron, organic matter, and porosity. Aqueous total-Hg increased with aqueous total suspended solids and iron concentrations. Aqueous methyl-Hg was a positive function of dissolved organic carbon in water and of methyl-Hg and total iron concentration in sediments.

Despite differences in net production of methyl-Hg in sediments from the different sites, the average concentration of Hg in fish tissues among the 4 sites was consistently rather high. The average concentrations of Hg (ppm±1SD) in 6 fish from each site were: open river: 0.77±0.45; dam: 0.85±0.35; agriculture: 0.88±0.35; wetland: 1.7±0.80. Comparing mean fish Hg concentrations among sites, only the wetland site was found to be significantly different from the other three sites.

These preliminary results suggest net methyl-Hg production in sediments and flux to water is greater in environments with organic-rich, slower moving turbid waters and fine-grained sediments. These, in turn, are related to land use and hydrological variables, consistent with our hypotheses. However, we found that fish collected from rivers/stream sites with differing impacts have highly variable Hg burdens, which may be explained, at least in part, by their mobility or the mobility of their prey. Therefore, it may not be possible to draw direct quantitative links between land use types and Hg concentration in fish in physically dynamic riverine ecosystems.

Kimberly A. Warner
Research Scientist, Center for Freshwater Studies,
University of Alabama Department of Biological Sciences, Tuscaloosa, AL

Professional Experience

- Specialty: Microbial Ecology and Biogeochemistry
- Current position (Oct. 1999-Present): Research Scientist, Center for Freshwater Studies, The University of Alabama, Department of Biological Sciences, Tuscaloosa, AL 35487-0206. Coordinating an interdisciplinary project on watershed and hydrological controls on mercury transformation and accumulation in predatory fish in Alabama rivers. Emphasis on microbial and biogeochemical aspects and explanation of science to stakeholders.
- Previous position (Feb.-Sept. 1999): Assistant Research Scientist, UMCES Chesapeake Biological Laboratory, Solomons, MD. Completed project on environmental controls on microbial transformation and degradation of chlorinated organic pollutants in Chesapeake Bay.

Education

Ph.D., Marine, Estuarine and Environmental Sciences, February 1999. University of Maryland, College Park. Dissertation title: "Reductive dechlorination of the model compound 2,4-dichlorophenol in Chesapeake Bay sediments: Effects of sulfur biogeochemistry."
B.S., Environmental Science, *Summa cum laude,* December 1988. University of the District of Columbia, Washington, D.C.
A.A.S., Marine Science, December 1986. University of the District of Columbia, Washington, D.C.

Research Interests

Anaerobic microbial ecology and biogeochemistry; sulfur cycling; microbial-pollutant interactions; mercury transformation and behavior on watershed scales; estuarine and watershed ecology, health and integrity.

Selected Mercury Related Research

Social Impact Assessment of Mercury Contamination in Mobile River Basin

Dr. Hobson Bryan
University of Alabama Department of Geography
Coauthors: Misty Samya, Hendrik Snow, Kimberly Warner, Jean-Claude Bonzongo, Eric Roden, W. Berry Lyons, Milton Ward, and Indrajeet Chaubey

This reports the social assessment and public involvement dimension of a three-year research project concerning mercury in largemouth bass. An interdisciplinary research team in the Departments of Biology, Geology, and Geography at the University of Alabama is currently just completing an investigation of factors that control the movement of methyl mercury through the aquatic system of the primary rivers of the Mobile-Alabama River Basin. These factors include inputs from agricultural and urban land uses, impoundment of rivers, and wetland abundance. Findings of high mercury levels in some largemouth bass prompt concerns about human exposure to mercury, primarily through consuming contaminated fish.

The social assessment phase of the research focuses on identifying a full range of stakeholder groups to solicit prominent issues and suggest public policy approaches to controlling methyl mercury exposure. This project took a dual approach to public involvement through the social assessment process. As stakeholders, recreational fishermen (i.e., tournament bass anglers) participated in the data collection, a resident expert fisherman, knowledgeable about access sites and area land use along the waterways, advised project management. In addition, meetings on the topic were conducted with representatives of the power generation industry, environmental groups, the coal bed methane industry, and various state government agency officials. Thus, the process informed stakeholders about the research, solicited expert advice, and generated suggestions for policy response through meetings with a number of groups representing a range of interests and concerns about mercury levels.

As final project results are obtained, stakeholder groups will be informed and solicited for their advice and feedback. A particular challenge will be to reach such at-risk groups as minorities and the poor--who may not be in the information mainstream, yet who may be heavy consumers of fish from public waters--women of child-bearing age, those who are pregnant, and children. Information strategies will have to be addressed that inform the public on reasonable actions they can take to avoid harm from eating fish, while being cognizant of the concerns of various segments of the recreational fishing industry and other areas of the economy dependent on a healthy fishery.

Hobson Bryan
Professor, Department of Geography
Urban and Regional Planning, University of Alabama

Hobson Bryan is a professor in the Department of Geography, Urban and Regional Planning, at the University of Alabama. He is Immediate Past President of the International Association for Impact Assessment, a 2,500-member organization of consultants, academics, and decision officials from 112 countries. His teaching, research, and consulting interests center on environmental policy, natural resource issues, environmental and social impact assessment. Dr. Bryan served as Program Leader for Social Impact Assessment for the U.S. Forest Service, developed a national training program for that agency, and aided in the development of social impact assessment guidelines and policy for the New Zealand Commission for the Environment as a Senior Fulbright Research Fellow. His international training courses in social impact assessment, organizational analysis, and strategic planning span over twenty years of work. Dr. Bryan is a co-author of the text, Social Assessment: Theory, Process & Techniques (Taylor Baines, 1995) and has authored dozens of articles on this and related topics.

Economic Realities of Mercury in the Environment

Seafood Industry Perspective
Mr. Bob Collette
National Fisheries Institute

There is not much about mercury that is simple. The science is complicated and the issues surrounding public policy are complex and multifaceted. It goes without saying that fishermen, processors and purveyors of seafood do not add mercury to fish or to the environment. Nevertheless, we find ourselves in the middle of a debate that intermingles environmental and public health concerns. We certainly believe that protecting people, such as pregnant women, who are especially vulnerable to the potential effects of mercury is a paramount concern. Because fish is such an important part of a healthy diet for many consumers, government agencies must have sound scientific justification when they tell people to limit consumption of fish or place limits on which fish can be sold. Decisions about protecting consumers, therefore, must be based on a thorough assessment of scientific data and the public health impacts, both positive and negative, associated with various risk management approaches.

I will focus my comments on how various approaches to managing and communicating about the relative risks and benefits associated with fish consumption may impact consumer habits, the nutrition and health status of consumers and the viability of the seafood industry.

Robert L. Collette
National Fisheries Institute

Mr. Collette received a Master of Science degree in Food Science and Technology from the University of Rhode Island where his graduate studies included work on the shelf life extension of seafood products. He was a seafood technology specialist for the Alabama Sea Grant Extension Program before joining the National Fisheries Institute as the Director of Food Regulatory Affairs in 1984. In 1999, he was promoted to his current position of V.P of Science and Technology for the Institute. Primary responsibilities include at NFI include planning and execution of NFI's programs to facilitate the development and transfer of scientific information and technological innovations to association members. Mr. Collette is a member of the Seafood HACCP Alliance Steering Committee and one of the original trainers for the Alliance's three-day training course.

Economic Realities of Mercury in the Environment

Recreational Fishing Perspective
Dr. Bob Shipp
University of South Alabama

Recreational fishermen do not share quite the level of concern over mercury in finfish as one might expect from commercial harvesters. This is because there has been much more sentiment toward tag and release in the recreational sector in recent years, and much less toward "meat fishing." Nevertheless, recreational fishermen are anxious to learn the methyl mercury levels at various life stages of popular species.
This is because many species are still consumed by family members, and regulations regarding slot limits, bag limits, seasonal closures, etc. may be impacted by this information.

Additionally, recreational tournaments often donate catches to local charitable groups, orphanages, and other worthwhile recipients. This practice may be sharply curtailed or modified should mercury levels dictate.

Of additional concern and interest to recreationals is the suggestion that oil platforms may contribute to mercury levels in reef species. Should this prove true, fishing preferences would likely be greatly modified.

Dr. Bob Shipp
University of South Alabama

Bob was born in Tallahassee, but grew up with his time split between New Orleans and Ft. Walton Beach, FL. He graduated from Spring Hill College in 1964, and received his MS (1966) and Ph D (1970) degrees from Florida State. During his graduate days he was also an instructor of Biology at Florida A&M.

He has taught at the University of South Alabama since 1972, where he is presently chairman of the Department of Marine Sciences and director of the Alabama Center for Estuarine Studies. He was associate director of the Dauphin Island Sea Lab for ten years. He edited the marine journal Northeast Gulf Science (now Gulf of Mexico Science) for twenty years, and for four years was editor of Systematic Zoology, a premier international journal devoted to evolutionary theory.

He served on the Board of Governors of the American Society of Ichthyologists and Herpetologists, and was president of its southeastern division. He was appointed to the Gulf of Mexico Fishery Management Council in 1991, served as its chairman during 1996-97 and again from 1999-2000. The Councils, created by Congress, are charged with management of the nation's marine fishery resources. He was director of the Alabama CCA and continues to serve as their senior scientist.

He has judged many fishing tournaments, including, since 1982, the Alabama Deep Sea Fishing Rodeo, the nation's oldest and largest tournament, which was recently featured along with Bob in the New Yorker magazine. He is a staff writer for Sport Fishing magazine, and authored the July 1999 issue cover story on Mako sharks. His semi-popular/semi-technical "Dr. Bob Shipp's Guide to Fishes of the Gulf of Mexico" is currently in its fifth printing, and is used by the U.S. Coast Guard and National Marine Fisheries Service for field identification of fishes. He has also authored scores of scientific as well as popular papers and articles.

His research interests are fish systematics and zoogeography, and ecology of artificial reef systems. On the latter topics he has presented numerous papers at conferences including two at the recent international reef conference in San Remo, Italy. He was also an invited presenter on artificial reefs at the NOAA sponsored Marine Recreational Fisheries Symposium in San Diego, Senate hearings on fisheries management, and the Minerals Management Services symposium in New Orleans.

His wife, Linda, instructed biology at Spring Hill College for many years, and chaired the Biology Department at the University of Mobile until she went to work at their son Matt's Mobile restaurants (Justine's and the Downtown Octopus). He also has an older daughter, Karla, in Tampa, and a younger one, Erin, at Loyola University of New Orleans and the American University of Paris.

Economic Realities of Mercury in the Environment

Environmental Perspective
Ms. Felice Stadler
National Wildlife Federation

To date, the U.S. EPA has not completed a thorough analysis of the cost of mercury contamination, and the economic benefits that will be gained from cleaning up the contamination. Nevertheless, one can make some general assumptions on the potential costs associated with mercury pollution by looking at the intrinsic value of those resources; additionally one can examine the economic costs to those industries that are dependent on healthy fisheries, like the recreational fishing industry. Perhaps more important, though, is evaluating whether there are feasible, cost-effective alternatives to mercury containing products and processes, and if there are, what steps can be taken to eliminate the sources of mercury pollution.

The National Wildlife Federation has been leading a campaign in Michigan calling on the state to adopt a mercury phase-out plan. Advocacy on the federal level has focused primarily on control strategies for coal-fired power plants and consumer products. Through this work, NWF is completing an economic analysis to better understand the cost of implementing a phase-out plan. Information also is being compiled on the cost of mercury control for power plants, and the cost of mercury-free products. Preliminary results of this research will be presented.

Felice Stadler
National Wildlife Federation

Felice Stadler is the national policy coordinator for NWF's Clean the Rain campaign, the goal of which is to eliminate the release of mercury and other persistent toxic chemicals through policy changes both locally and nationally. In this capacity, she serves as a technical resource to Congressional staff, U.S. EPA, environmental advocates, and the general public on mercury control strategies and policy. Ms. Stadler has been working in the field of clean air policy (with a focus on air toxics) for 10 years in a variety of capacities.

Prior to joining NWF in the fall of 2000, Ms. Stadler was the policy director at the Clean Air Network, a project of the Natural Resources Defense Council, for over three years. Before that, she managed the Small Business Assistance Program in Kansas for two years, providing technical assistance to companies being targeted by new federal clean air rules. Ms. Stadler began her work on air toxics issues in Missoula, Montana where she volunteered with the city-county health department developing clean air programs while completing her master's degree at the University of Montana's Environmental Studies program.

Economic Realities of Mercury in the Environment

Minamata Plus 50: Where Are We?

Dr. Leonard Levin
Electric Power Research Institute

Some 50 years after the direct discharge of methylmercury into Minamata Bay, and the "cats of Minamata," our understanding of environmental mercury has increased substantially, but mercury management methods remain less developed. Inventories of atmospheric sources show that not only do Asian industrial sources contribute half of the global burden, but that they contribute substantially to mercury additions to U.S. waterways by atmospheric deposition. Background sources, both natural sources such as hot springs and legacy sources such as abandoned mill sites, are roughly equal to industrial sources as emitters, but may play a lesser role in local and regional deposition.

Coal-fired electric power plants make up about one-third of current U.S. industrial emissions. These plants already remove about 40% of the mercury in the coal fuel before it is released from the stack, due to coal cleaning and current emissions controls. Studies show that additional levels of current controls, such as sulfur scrubbers or precipitators, become substantially more expensive when dedicated to mercury removal, and that advanced systems such as activated carbon are essentially unproven. This leaves unresolved the basic management question: will there be a substantial drop in fish mercury if there is a substantial cut in utility mercury emissions? This source-receptor relationship is not only unresolved, but is faced with increasing questions as scientific issues continue to be addressed. The possibility that emissions plumes chemically reduce the soluble divalent form of mercury to the globally-cycling elemental form has now been demonstrated initially in both field trials and laboratory measurements, but remains to be clarified. Modeling studies by EPA and others have recently shown a small contribution of utility emissions to deposition patterns at U.S. locations. These and other issues remain to be clarified.

Dr. Leonard Levin
Electric Power Research Institute

Dr. Leonard Levin is Program Manager for Air Toxics Health and Risk Assessment at EPRI, in Palo Alto, California. He is responsible for program management in environmental mercury, human exposure and health effects, atmospheric chemistry and physics of trace substances, and risk assessment. Dr. Levin has degrees from M.I.T., the University of Washington, and the University of Maryland. He has served as advisor to the U.S. EPA, the U.S. Department of Energy, the University of California at Berkeley, and the Tulane University School of Medicine.

Research and Educational Recommendations

The following pages contain research and educational recommendations from three sources: the National Research Council's *Toxicological Effects of Methymercury*, EPA's *Mercury Study Report to Congress* and the Gulf State's Marine Fisheries Commission's *Methylmercury in Marine Fish: A Gulf-Wide Initiative*. These recommendations are provided as a reference for needs as identified by panels of experts in the field of mercury.

NRC's *Toxicological Effects of Methylmercury*

Chemistry, Exposure, Toxicokinetics, and Toxicodynamics (Page 60)

1. As data become available, exposure to elemental Hg from dental amalgams should be considered in risk assessment of MeHg. Exposure to other chemical forms of Hg should also be considered.
2. Retention of inorganic Hg in the brain for years following early MeHg intake is possibly related to the latent or long-term neurotoxic effects reported. The long half-life of inorganic Hg in the brain following MeHg intake should be considered in risk assessment of MeHg.
3. The mechanisms, including any enzymes, involved in the biotransformation of MeHg to mercuric Hg in human tissues need to be investigated, especially at the subcellular level. The effects of Hg on signaling pathways and the conformation of enzymes and structural proteins should be further elucidated, because the development and function of the brain would be particularly sensitive to such effects.
4. Exposure assessment of the U .S. population-including those with high fish consumption-is needed to provide a full picture of the distribution of MeHg and total Hg exposure nationally and regionally.

Biological Variability (Page 96-98)

1. Future studies of MeHg exposures in humans should include a thorough assessment of the diet during the periods of vulnerability and exposure. They should involve assessment of the nutritional adequacy of the group, including the assessment of nutritional and environmental factors that might attenuate or exacerbate the effect of MeHg on the health end points measured.
2. Dietary assessment should be conducted concurrently with the exposures, because retrospective assessment is influenced by many factors, including memory , changes in eating behavior, food fortification, and use of prenatal and postnatal vitamin and mineral supplementation. Dietary assessment should be conducted on a person-specific basis, with particular effort to estimate quantitatively individual consumption and consumption patterns of fish and pilot whale.
3. For all the studies, the estimates of consumption of fish (and whale meat as appropriate) should be used with information on MeHg concentrations in the food to estimate possible MeHg intake by pregnant women, young children, and adults. Attempts should be made to validate estimates of intake by using experimental data on the relationship between hair Hg concentration and diet intake.
4. Future studies should include a standardized measure of the duration of breast-feeding and the quantity of breast milk ingested by infants. The dose of MeHg is dependant on the amount of milk ingested and the MeHg content of the milk. Historical recording of duration of breast-feeding is likely to be biased; therefore, a prospective diary of breast-feeding and weaning should be considered.
5. Studies using animal models should examine changes in the dose response characteristics of Hg effects associated with nutritional or genetic factors.
6. Any biomarker-based Rill for MeHg should specifically address interindividual toxicokinetic variability in the estimation of dose corresponding to a given biomarker concentration.

a. The starting point for addressing interindividual toxicokinetic variability should be a central-tendency estimate of the ingested dose corresponding to a critical biomarker concentration (e.g., a benchmark hair concentration).
b. The central-tendency estimate of the ingested dose should be based on careful consideration of the several possible and sometimes contradictory data sets for each parameter. A starting point for such consideration is the discussion of parameter distributions presented in the analyses of Stern (1997), Swartout and Rice (2000), and Clewell et al. (1999).

7. An uncertainty-factor adjustment should be applied to any central-tendency estimate of the ingested dose corresponding to the critical biomarker concentration.
8. For an RfD based on maternal-hair Hg concentration, an uncertainty-factor adjustment of 2 should be applied to the central-tendency estimate of dose to be inclusive of 95% of the toxicokinetic variability in the population. An uncertainty-factor adjustment of 2-3 should be applied to be inclusive of 99% of the toxicokinetic variability.
9. For an RfD based on blood Hg concentration, an uncertainty factor adjustment of about 2 should be applied to the central-tendency estimate of dose to be inclusive of 95-99% of the toxicokinetic variability in the population.
10. Because of the recognized nutritional benefits of diets rich in fish, the best method of maintaining fish consumption and minimizing Hg exposure is the consumption of fish known to have lower MeHg concentrations.

Dose Estimation (Pages 139-140)

1. Quantitative dietary intake data on patterns of consumption of the primary sources of MeHg including all marine food sources, should be collected in all prospective studies of MeHg exposure. Estimates of exposures will improve dose-response analyses that have implications for regulatory purposes.
2. In future studies, data on maternal fish intake by species and by meal should be collected along with Hg biomarker data. Those data should be used to provide estimates of temporal variability in MeHg intake during pregnancy.
3. Future studies should collect data on maternal-hair, blood, and cord-blood Hg concentrations. All three dose metrics should be considered in attempting to identify dose-response relationships.
4. Data are needed that reliably measure both Hg intake and biomarkers of Hg exposure to clarify the relationship between the different dose metrics. NHANES IV data should be examined when it becomes available to determine if it satisfies those needs.
5. To detect exposure variability, archived hair strands from both the Seychelles and the Faroe Islands studies should be analyzed by continuous single-strand XRF analysis. The possible dose metrics that can be derived from XRF analysis should be examined in the dose-response assessment. Such considerations should also be addressed in future studies.

Health Effects of Methylmercury (Pages 231-232)

1. Epidemiological research is needed to evaluate the prevalence of chromosomal aberrations and cancer, especially leukemia and renal tumors, among populations that have chronic exposure to MeHg through ingestion of contaminated fish.
2. The ability of MeHg to cause chromosomal damage and promote tumor growth should be considered in the establishment of exposure guidelines.
3. Research is needed to determine the effects of MeHg exposure on the immune system, including the effects on the developing immune system, resistance to microbial pathogens, and autoimmunity. Mechanisms by which the immune system is involved in the target-organ toxicity of Hg should also be examined.

4. Research is needed to assess the effects of MeHg on reproduction, including the effects on fertility indicators, such as sperm production, conception rates, and pregnancy outcomes.
5. Research is needed to evaluate the impact of dietary exposure to MeHg on the prevalence of hypertension and cardiovascular disease in the United States. The risk of fatal and nonfatal heart disease must be considered in the development of a reference dose for this contaminant.
6. Research is needed to determine the long-term implications of the neuropsychological and neurophysiological effects of low-level prenatal MeHg exposure detected in children, specifically whether they are associated with an increased risk for later neurological diseases.
7. Research using animal models is needed to better define the immediate and long-term effects of early chronic low-level MeHg exposure. Studies should focus on several important issues:
    a. Critical periods for MeHg effects (in utero or postnatal).
    b. Low-level dose-response relationships (ppb range).
    c. MeHg demethylation in the brain following early MeHg exposure.
    d. Synergistic effects of early MeHg and Hg vapor exposure.
    e. Neurodegenerative disorders related to early MeHg exposure.
8. Animal studies should be conducted to examine the neurodevelopmental effects of continuous versus peak MeHg exposures.

Comparision of Studies for Use in Risk Assessment (Page 269)

1. It would be helpful to obtain more comprehensive nutritional data from all three populations as well as single-strand hair analyses to address more effectively the issue of spiking or bolus dose. A reanalysis of the 5.5-year SCDS data controlling statistically for examiner might also be useful.
2. Most of the MeHg exposure standards currently in effect are based on extrapolations from the Iraqi MeHg poisoning episode, in which exposure was due to the consumption of highly contaminated and resulted in body burdens that greatly exceeded those found in the general population of fish consumers. Given the availability of data from three well-designed epidemiological studies in which prenatal MeHg exposures were in the range of general-population exposures, exposure standards should be based on data from these newer studies.

Dose-Response Assessment (Pages 300-301)

1. Until better statistical methods become available, risk assessment for MeHg should be based on benchmark dose calculations rather than NOAELs or LOAELs.
2. Given the available data, risk assessment should be based on the Boston Naming Test from the Faroe Islands study using MeHg measured in cord blood.
3. Despite some potential for PCB exposures to bias BMD estimates based on the Faroe Islands study, the committee recommends using estimates based on the full cohort and not adjusting for PCB exposure, mostly because the larger sample size is believed to result in more reliable estimates.
4. Benchmark doses should be based on the K-power model with K constrained to take a value of 1 or greater.
5. Because the integrative analysis is exploratory, it would be premature to recommend it for use now. However, the approach should be considered in context of a weight-of-evidence argument. Further research on the use of integrative models for risk assessment would be useful.
6. Further research is generally needed on statistical issues related to risk assessment that is based on epidemiological data. In particular, further research to develop more appropriate methods for handling model uncertainty (e.g., the Bayesian technique of *model averaging* (Carlin and Louis 1998)) would be useful. Further work is also needed to develop risk assessment methods for a setting like MeHg where the study population contains no true controls.

Risk Characterization and Public Health (Pages 326-328)

1. Hg is pervasive and persistent in the environment. Its use in products and emission from industrial processes and combustion have resulted in global circulation and atmospheric deposition. There have been well-documented instances of population poisonings, highly exposed occupational groups, and worldwide chronic low-level environmental exposures. The bioaccumulation of MeHg can lead to high concentrations in many species of fish and result in unacceptable levels of exposure and risk to highly exposed or susceptible subpopulations.
2. The weight of the evidence of developmental neurotoxic effects from exposure to MeHg is strong. There is a strong data base, which includes multiple human studies and experimental evidence in animals and in vitro tests. Human studies include both high-exposure scenarios and evaluations of effects of chronic low-level exposure. The epidemiological studies also include well-established biomarkers to evaluate exposure levels in study populations.
3. The weight of evidence from multiple epidemiological studies supports the selection of neurotoxicity in children exposed in utero as the most sensitive well-documented effect and a suitable end point for the derivation of the BMD. However, emerging evidence of other potential effects should also be considered in the calculation and the implementation of the EP A RfD.
4. Given the availability of results from large prospective epidemiological studies, the Iraq study results should no longer be considered the critical study for the EPA RfD. The exposure scenarios in Iraq are not comparable to the low-level chronic exposures in North America. In addition, there are wel l-recognized uncertainties concerning exposure and response classification in the Iraq study.
5. The New Zealand, Faroe Islands, and Seychelles studies are well designed epidemiological investigations in which prenatal MeHg exposures were within the range of at least some U.S. population exposures. Any revision of the RID or other exposure standards should consider the findings of these studies. After considering the weight of evidence and range of results from the three major epidemiological studies, the committee concludes that a positive study will provide the strongest public-health basis for the Rill and recommends the Faroe Islands study as the critical study. Within that study, the lowest BMD for a neurobehavioral end point considered to be sufficiently reliable is the Boston Naming Test. The BMDL estimated from that test is 58 ppb Hg in cord blood (approximately corresponding to 12 ppm Hg in hair). That value should be considered a reasonable point of departure for the development of the revised RfD.
6. An MOE analysis using available estimates of population exposure levels indicates that average U.S. population risks from MeHg exposure are low. However, those with high exposures from frequent fish consumption might have little or no margin of safety.
7. The population at highest risk is the offspring of women of childbearing age who consume large amounts of fish and seafood. The committee estimates that over 60,000 children are born each year at risk for adverse neurodevelopmental effects due to in utero exposure to MeHg,
8. There is a critical need for improved characterization of population exposure levels to improve estimates of current exposure, track trends, and identify high-risk subpopulations. Characterization should include improved nutritional and dietary exposure assessment and improved biomonitoring for all population groups. Exposure to other chemical forms of Hg, including exposure to elemental Hg from dental amalgams, should also be investigated.
9. The application of uncertainty factors in the revision of the Rill should be based on a thorough quantitative and qualitative evaluation of the full range of uncertainties and limitations of the critical studies. Uncertainty factors applied in the development of a revised Rill should include data-base insufficiency and interindividual toxicokinetic variability in dose reconstruction. As a starting point, an uncertainty factor of 2-3 should be applied to a central tendency estimate of dose derived from maternal hair, or a factor of about 2 should be applied to a central tendency estimate of dose derived from cord blood to account for interindividual pharmacokinetic variability in dose reconstruction, The choice of an uncertainty factor for data-base insufficiency

is, in part, a policy decision; however, given the data indicating possible low-dose sequelae and latent effects and immunotoxicity and cardiovascular effects, the committee concludes that an overall composite uncertainty factor of no less than 10 is needed.

10. Concurrent with the revision of the RfD, harmonization efforts should be undertaken to establish a common scientific basis for the establishment of exposure guidance and reduce current differences among agencies. Harmonization efforts should address the risk-assessment process and recognize that risk-management efforts reflect the differing mandates and responsibilities of these agencies.
11. Recent studies have found associations between exposure to MeHg and impairments of the immune, reproductive, and cardiovascular systems. Immune and cardiovascular effects have been observed following both prenatal and adult exposures. MeHg exposure levels associated with those effects are comparable to and in some cases lower than those known to cause neurodevelopmental problems. Additional research should be done using animal models and human populations that have chronic, low-dose exposure to MeHg. Effects of exposure during fetal development through the entire life span is needed. Further research is also needed to evaluate MeHg-induced chromosomal aberrations and cancer.
12. The committee recommends that results from the Boston Naming Test in the Faroe Islands study be used in the calculation of the RfD. For that study, dose- response data based on Hg concentrations in cord blood should be modeled using the K-power model ($K \sim 1$). On the basis of that study, that test, and that model, the committee's preferred estimate of the BMDL is 58 parts per billion (ppb)l of Hg in cord blood (approximately corresponding to 12 ppm Hg in hair). To estimate this BMDL, the committee's calculations involved a series of steps, each involving one or more assumptions and related uncertainties. Alternative assumptions could have an impact on the estimated BMDL value. In selecting a single point of departure, the committee followed established public-health practice of using the lowest value for the most sensitive, relevant end point.

Mercury Study Report to Congress – Volume I: Executive Summary

(EPA-452/R-97-003 December 1997)
Research Needs from Pages 5-1 Through 5-7

Anthropogenic Mercury Emissions in the United States

An effort has been made to characterize the uncertainties (at least qualitatively) in the emissions estimates for the various source categories described. There are inherent uncertainties in estimating emissions using emission factors. To reduce these uncertainties, a number of research needs remain, including the following.

1. Source test data are needed from a number of source categories that have been identified as having insufficient data to estimate emissions. Notable among these are mobile sources, landfills, agricultural burning, sludge application, coke ovens, petroleum refining, residential woodstoves, mercury compounds production and zinc mining. A number of manufacturing sources were also identified as having highly uncertain emissions estimates. Notable among this category are secondary mercury production, commercial and industrial boilers, electric lamp breakage, primary metal smelting operations and iron and steel manufacturing. The possibility of using emissions data from other countries could be further investigated.
2. Development and validation of a stack test protocol for speciated mercury emissions is needed.
3. More data are needed on the efficacy of coal cleaning and the potential for slurries from the cleaning process to be a mercury emission source.
4. More data are needed on the mercury content of various coals and petroleum and the trends in the mercury content of coal burned at utilities and petroleum refined in the U.S.
5. Additional research is needed to address the potential for methylmercury to be emitted (or formed) in the flue gas of combustion sources.
6. The importance (quantitatively) of re-emission of mercury from previously deposited anthropogenic emissions and mercury-bearing mining waste needs to be investigated. This would include both terrestrial and water environments. Measuring the flux of mercury from various environments would allow a determination to be made of the relative importance of re-emitted mercury to the overall emissions of current anthropogenic sources.
7. Determination of the mercury flux from natural sources would help determine the impact of U.S. anthropogenic sources on the global mercury cycle as well as the impact of all mercury emissions in the United States.
8. The use of more sophisticated fate and transport models for mercury will require more detailed emissions data, particularly more information on the chemical species of mercury being emitted (including whether these species are particle-bound) and the temporal variability of the emissions.

Mercury Fate and Transport Modeling

During the development of the mercury fate and transport assessment, many areas of uncertainty and significant data gaps were identified. Many of these have been identified in the document, and several are presented in the following list.

1. Improved analytical techniques for measuring speciated mercury air emissions are needed as well as total mercury emissions from point sources. Laboratory evidence suggests that divalent mercury gas emissions will wet and dry deposit much more readily than elemental mercury gas. Particle-bound mercury is also likely to deposit relatively quickly. Current stack sampling methods do not provide sound information about the fraction of mercury emissions that are in oxidized form. While filters are used to determine particulate mercury fractions, high temperature stack samples may not be indicative of the fraction of mercury that is bound to particles after dilution and cooling in the first few seconds after emission to the atmosphere. Methods for

determination of the chemical and physical forms of mercury air emissions after dilution and cooling need to be developed and used to characterize significant point sources.

2. Evaluated local and regional atmospheric fate and transport models are needed. These models should treat all important chemical and physical transformations which take place in the atmosphere. The development of these models will require comprehensive field investigations to determine the important atmospheric transformation pathways (e.g., aqueous cloud chemistry, gas-phase chemistry, particle attachment, photolytic reduction) for various climatic regions.
3. The evaluation of these models will require long-term national (possibly international) monitoring networks to quantify the actual air concentrations and surface deposition rates for the various chemical and physical forms of mercury.
4. Better understanding of mercury transport from watershed to water body including the soil chemistry of mercury, the temporal aspects of the soil equilibrium and the impact of low levels of volatile mercury species in surface soils and water bodies on total mercury concentrations and equilibrium.
5. Better understanding of foliar uptake of mercury and plant/mercury chemistry. (The most important questions: do plants convert elemental or divalent mercury into forms of mercury that are more readily bioaccumulated? Do plants then emit these different forms to the air?) A better understanding of the condensation point for mercury is needed.
6. Better understanding of mercury movement from plant into soil (detritus). May need to refine the models used to account for movement of mercury in leaf litter to soil.
7. The impact of anthropogenic mercury on the "natural," existing mercury levels and species formed in soil, water, and sediments needs better understanding. How does the addition of anthropogenic mercury affect "natural" soil and water mercury cycles? Natural emission sources need to be studied better and their impacts better evaluated.
8. Improved understanding of mercury flux in water bodies and impact of plant and animal biomass are needed. Unlike many other pollutants, most of the methylmercury in a water body appears to be in the biological compartment. The sedimentation rate as well as benthic sediment:water partition coefficient require field evaluation. Important to consider rivers and other larger water bodies in these flux analyses.

Exposure from Anthropogenic Mercury Emissions in the United States

1. To improve the quantitative exposure assessment modeling component of the risk assessment for mercury and mercury compounds, U.S. EPA would need more and better mercury emissions data and measured data near sources of concern, as well as a better quantitative understanding mor mercury chemistry I the emission plume, the atmosphere, soils, water bodies, and biota.
2. To improve the exposure estimated based on surveys of fish consumption, more study in needed among potentially high-end fish consumers, which examines specific biomarkers indicating mercury exposure (e.g., blood mercury concentrations and hair mercury concentrations).
3. A pharmacokinetic-based understanding of mercury partitioning in children is needed. Additional studies of fish intake and methylmercury exposure among children are needed.

Health Effects of Mercury and Mercury Compounds

1. In addition to the ongoing studies identified in the health effects review, further research is necessary for refinement of the U.S. EPA's risk assessments for mercury and mercury compounds. In order to reduce uncertainties in the current estimates of the oral reference doses (RfDs) and inhalation reference concentrations (RfCs), longer-term studies with low-dose exposures are necessary. In particular, epidemiological studies should emphasize comprehensive exposure data with respect to both dose and duration of exposure. Some

studies should be targeted to populations identified in this Report as likely to experience methylmercury exposure in fish (e.g., subsistence fishers).

2. The current RfD and RfC values have been determined for the most sensitive toxicity endpoint for each compound; that is, the neurological effects observed following exposure to elemental or methylmercury, and the renal autoimmune glomerulonephritis following exposure to inorganic mercury. For each of these compounds, experiments conducted at increasingly lower doses with more sensitive measures of effect will improve understanding of the respective dose-response relationships at lower exposure levels and the anticipated thresholds for the respective effects in humans. Similar information from developmental toxicity studies would allow determination of RfDs for developmental toxicity (RfD) for elemental and inorganic mercury. dt
3. Research needs include studies which will delineate the most appropriate indicators of neurotoxic effects for exposed adults, children and individuals exposed to methylmercury *in utero*. Well conducted studies are also needed to clarify critical levels at which other toxic effects could occur in humans.
4. Well-conducted studies are also needed to clarify exposure levels at which toxic effects other than those defined as"critical" could occur in humans. For all three forms of mercury, data are inadequate, conflicting, or absent for the following: adverse reproductive effects (effects on function or outcome, including multigeneration exposure); impairment of immune function; and genotoxic effects on human somatic or germinal cells (elemental and inorganic mercury).
5. Investigations that relate the toxic effects to biomonitoring data will be invaluable in quantifying the risks posed by these mercury compounds. In addition, work should focus on subpopulations that have elevated risk because they are exposed to higher levels of mercury at home or in the workplace, because they are also simultaneously exposed to other hazardous chemicals, or because they have an increased sensitivity to mercury toxicity.
6. There are data gaps in the carcinogenicity assessments for each of the mercury compounds. The U.S. EPA's weight-of-evidence classification of elemental mercury (Group D) is based on studies in workers who were also potentially exposed to other hazardous compounds including radioactive isotopes, asbestos, or arsenic. There were no appropriate animal studies available for this compound. Studies providing information on the mode of action of inorganic mercury and methylmercury in producing tumors will be of particular use in defining the nature of the dose response relationship.
7. The assessment of both noncarcinogenic effects and carcinogenic effects will be improved by an increased understanding of the toxicokinetics of these mercury compounds. In particular, quantitative studies that compare the three forms of mercury across species and/or across routes of exposure are vital for the extrapolation of animal data when assessing human risk. For elemental mercury there is a need for quantitative assessment of the relationship between inhaled concentration and delivery to the brain or fetus; in particular the rate of elemental to mercuric conversion mediated by catalase and the effect of blood flow. Such assessment is needed for evaluation of the impact of mercury exposure from dental amalgam.
8. Work has been done on development of physiologically-based pharmacokinetic models. While one of these has developed a fetal submodel, data on fetal pharmacokinetics are generally lacking. The toxicokinetics of mercury as a function of various developmental stages should be explored. Elemental mercury and methylmercury appear to have the same site of action in adults; research is, therefore, needed on the potential for neurotoxicity in newborns when the mother is exposed. This work should be accompanied by pharmacokinetic studies and model development.

1. *Process-based Research.* Mechanistic information is needed to understand the variability that presently typifies the mercury literature. This research includes laboratory and field studies to identify the determinants of mercury accumulation in aquatic food chains and kinetic information that would allow researchers to describe the dynamics of these systems. Areas of uncertainty include: (1) translocation of mercury from watersheds to waterbodies; (2) factors that determine net rates of methylation and demethylation; (3) dietary absorption efficiency from natural food sources; (4) effect of dietary choice; and (5) bioavailability of methylmercury in the presence of dissolved organic material and other potential ligands. In time, it is anticipated that this information can be used to develop process-based models for mercury bioaccumulation in fish and other aquatic biota. Significant progress in this direction is represented by the Mercury Cycling Model (MCM) (Hudson et al., 1994) and by the ISC3M model described in Volume III of this Report and employed in the wildlife exposure characterization.
2. *Wildlife Toxicity Data*. There is a need to reduce the present reliance on a relatively few toxicity studies for WC development. Additional data are needed for wildlife that constitute the most exposed organisms in various parts of the country, and in particular there is need to evaluate whether dietary selenium and endogenous demethylating pathways confer protection to piscivorous birds and mammals. Toxicity studies should examine endpoints relevant to the mode of action of methylmercury, including assessments of both reproductive and behavioral effects. There is also a critical requirement for toxicity data (e.g., growth and fecundity) that can be related to effects on populations, including effects on organisms that comprise the lower trophic levels.
3. *Improved Analytical Methods*. Efforts to develop and standardize methods for analysis of total mercury and methylmercury in environmental samples should be continued. Such methods must recognize the importance of contamination, both during the collection of such samples and during their analysis. It is particularly important that mercury measurements, which at present tend to be operationally defined (e.g., "soluble" or "adsorbed to organic material"), be made in such a way that mercury residues in fish can be correlated with the bioavailable mercury pool. Whenever possible, water samples should be filtered to obtain a measure of dissolved mercury species. As validated methods become available, it is important to analyze for both total and methylmercury so that differences between aquatic systems can be definitively linked to differences in methylmercury levels. Analyzing the two mercury species together will contribute to an understanding of existing data, much of which is reported as total mercury.
4. *Complexity of Aquatic Food Webs*. Present efforts to develop WC values for mercury are based on linear, four-tiered food chain models. Research is needed to determine the appropriateness of this simple paradigm and to develop alternatives if field data suggest otherwise. Of particular interest is whether zooplankton and phytoplankton should be modeled as two different trophic levels. Current information for detritivores and benthic invertebrates is extremely limited, even though their importance in mobilizing hydrophobic organic contaminants has been demonstrated.
5. *Accumulation in Trophic Levels 1 and* 2. Ongoing efforts to understand mercury bioaccumulation in aquatic systems continue to be focused on trophic levels 3 and 4, despite the fact that uncertainties in PPFs are relatively small. Additional emphasis should be placed on research at the lower trophic levels. In particular, there is a need to understand the determinants of mercury accumulation in phytoplankton and zooplankton and how rapid changes in plankton biomass impact these values.
6. *Field Residue Data*. High-quality field data are needed to support process-based research efforts and to determine residue concentrations in the fish and other aquatic biota that wildlife

eat. Whenever possible, it is desirable to collect residue data at all trophic levels and to analyze mercury levels in the abiotic compartments of a system (e.g., water and sediments). It is particularly important that such measurements be made in a broader array of aquatic ecosystem types (including both lakes and rivers) so that a better understanding of mercury cycling and accumulation can be obtained. Residue data from wildlife are needed to identify populations that are potentially at risk. Feathers and fur hold considerable promise in this regard due to the potential for "non-invasive" determination of mercury residues. Laboratory research is required, however, to allow interpretation of these data. Factors such as age, sex, and time to last molt are likely to result in variability among individuals of a single population and need to be understood. Whenever possible, tissue samples should be analyzed for both total and methylmercury, as well as selenium. This is especially true of the liver. More attention should be given to analysis of mercury levels in brain tissue, since this is the primary site of toxic action. Sampling efforts with wildlife should be accompanied by analyses of likely food items.

7. *Natural History Data*. The development of WC values requires knowledge of what wildlife eat. Fish sampling efforts are frequently focused on species that are relevant to human consumers but that may be of little significance to wildlife. There is an additional need to collect information for macroinvertebrates and amphibians. Seasonal and spatial effects on predation should be explored and methods developed to describe this information adequately. Additional life history data is needed to characterize fully the nature and extent of exposure to mercury. Complicating factors must be considered, including migratory behaviors and sex-specific differences in distribution and resource allocation. It is particularly important that information be collected to support the development of predictive population models for sensitive species. Such models must account for immigration and emigration, density dependent factors, and the observation that mercury often bioaccumulates as animals age resulting in variable residues in breeding animals from a single population.

Risk Characterization

1. A monitoring program is needed to assess either blood mercury or feather/hair mercury of piscivorous wildlife; particularly those in highly impacted areas. This program should include assessment of health endpoints including neurotoxicity and reproductive effects.
2. There is a need to collect additional monitoring data on hair or blood mercury and assess health endpoints among women of child-bearing age and children. This study should focus on high-end fish consumers and on consumption of fish from contaminated water bodies.
3. There is a need for improved data on effects that influence survival of the wildlife species as well as on individual members of the species.
4. There is a need for controlled studies on mercury effects in intact ecosystems.
5. Monitoring data sufficient to validate or improve the local impact exposure models are needed.

Mercury Control Technologies

1. Data from full-scale testing of activated carbon injection at a coal-fired utility boiler.
2. Additional data on the efficiency of various sorbents including fly ash-based sorbents, activated carbon, impregnated carbons and other types of sorbents, in reducing the different chemical species of mercury present in flue gas
3. Information on the cost-effectiveness and commercialization costs of other technologies for mercury control that are currently in the research stage. These include impregnated activated carbon, sodium sulfide injection, activated carbon fluidized bed and other types of sorbents.
4. Additional data on the ability and cost of conventional or advanced coal cleaning techniques to remove mercury from raw coal. The potential for mercury emissions from coal-cleaning slurries needs to be characterized.

5. Additional data on the fundamental mechanisms responsible for conversion of mercury to other chemical species as a result of combustion of certain coals or post-combustion conditions.
6. Additional information on improving the capture of mercury in wet FGD systems.
7. Additional analyses are required on the feasibility, cost-effectiveness of other mercury emission prevention measures such as emissions trading, emissions averaging, energy conservation, renewable energy, and fuel switching.

Methylmercury in Marine Fish: A Gulf-Wide Initiative

Gulf States Marine Fisheries Commission

March 13, 2002

1. The Gulf States Marine Fisheries Commission, in cooperation with the appropriate state and federal agencies, should encourage and facilitate the development of a Gulf-wide survey to collect fish tissue for mercury analysis. The survey should collect tissue from species commonly consumed by the public from commercial sources and caught and consumed by recreational anglers, and
2. The Gulf States Marine Fisheries Commission, in cooperation with the appropriate state and federal agencies, should encourage and facilitate the establishment of sufficient capacity for timely analysis of mercury tissue samples collected by the Gulf-wide survey,
3. The Gulf States Marine Fisheries Commission should work with the Gulf of Mexico Program, administered by the U.S. Environmental Protection Agency, to facilitate convening appropriate state and federal agency representatives to consider establishing consistent seafood consumption advisories and establishing common advisory levels for mercury in fish tissue, and
4. The Gulf States Marine Fisheries Commission, in cooperation with the appropriate state and federal agencies, should encourage and facilitate the development of an education and outreach strategy, including the development of new, and more effective education and outreach materials, to educate the general public about the risks associated with consumption of seafood that may be contaminated with mercury,
5. The Gulf States Marine Fisheries Commission, in cooperation with the appropriate state and federal agencies, should encourage and facilitate the development of a fish consumption survey of recreational anglers,
6. The Gulf States Marine Fisheries Commission, in cooperation with the appropriate state and federal agencies, should encourage and facilitate the establishment of a common, centralized database on mercury in marine fish tissue, and
7. Recognizing that methylmercury contamination of fish tissue is not confined to the Gulf of Mexico region, the Gulf States Marine Fisheries Commission should encourage similar initiatives as embodied in this report for the Atlantic and Pacific coasts.

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The Mercury Forum

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The Mercury Forum

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