MERCURY, HUMAN HEALTH EFFECTS, AN OVERVIEW

MERCURY WORKING GROUP, HARRISBURG, PA
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DONALD J. McGRAW, M.D., M.P.H.

• WHAT IS MERCURY?

Mercury (Hg), from the Greek Hydrargyros, meaning “water silver,” is a naturally occurring metal, mined largely as mercuric sulfate (HgS) from cinnabar ore. There are three primary forms of mercury, each with its own toxicology:

1. Elemental or metallic mercury (Hg^{1+}).
2. Inorganic mercury salts (Hg^{2+}).
3. Organic (methyl, ethyl, phenyl or alkyl) mercury.

• ELEMENTAL MERCURY

Elemental mercury is the only metal which is a liquid at room temperature. It is commonly used in thermometers, barometers, blood pressure cuffs, batteries, electrical switches and fluorescent light bulbs. Some dental fillings are composed of about 50% metallic mercury. Exposure may occur if these instruments are broken. When metallic mercury is released into the environment it begins to vaporize as a colorless, odorless gas, a process which increases with rising temperature. These vapors may pose a significant potential health risk.

• INORGANIC MERCURY SALTS

When mercury combines with other elements, such as chlorine, sulfur or oxygen, inorganic mercuric salts occur, generally in the form of white powders or crystals. These compounds are most familiar to the older generation as mercurochrome, formerly a common antibacterial containing about 2% mercury, and even now at present, predominantly an ophthalmic product, such as eye drops and contact lens solutions, and vaginal contraceptives gels and suppositories: mercuric chloride, a topical antiseptic once widely used in laxatives, teething powders and worming medications; or mercuric iodide, used in creams to lighten the skin. These formulations are still used in some prescription and over-the-counter medications, as well as pharmaceutical preservatives such as Thimerosal, the subject of considerable public concern in its vaccine application. Mercuric sulfide and oxide are sometimes used as colorants in paint and tattoo dyes.

- ## ORGANIC MERCURY

Organic mercury may occur as methyl mercury, alkyl mercury or phenyl mercury. Methyl mercury is the most common form of organic mercury, and is produced primarily by microorganisms, bacteria and fungi in the environment. When the adverse health effects of methyl mercury were recognized in the 1970's, its use in fungicides, was banned in the U.S. In 1990, phenyl mercuric compounds were also prohibited from use as anti-fungal agents in both interior and exterior paints due to their release of mercury vapors. The greatest concern for methyl mercury derived from its uptake by fresh and saltwater fish and shellfish. Fish at the top of the food chain will have the largest amount, and the oldest fish will have accumulated the most. The FDA estimates that the average individual is exposed to about 50 nanograms of mercury per kilogram of body weight, or approximately 3.5 mcg of mercury per day. This level is not believed to have any harmful effects. The range of consumption is estimated to be from 50 to 100 ng of mercury per kilogram per day.

- ## MERCURY IN THE ENVIRONMENT

Elemental mercury is part of the earth's natural crust and is released into the human environment by the erosive forces of wind and water, as well as through volcanic activity. Approximately eighty (80) percent of mercury generated by human activities (est. 2,000 tons) is metallic mercury released to the air from mining and smelting of ore, fossil fuel combustion and solid waste incineration. Fifteen (15) percent derives from fertilizers, fungicides and municipal solid waste. And, about five (5) percent is released through industrial waste water. The WHO reported in 1991 that the major source of atmospheric mercury is global degassing of mineral mercury from the hydrosphere at an estimated rate of 3,000 to 6,000 tons per year, or about 1-3 times the rate of release from anthropogenic sources. The variable overall contributions by human versus "natural" activities is not known, due to the significant and diverse background levels present for ions. **The atmospheric levels of mercury breathed in the air of our general environment are very low and do not represent an adverse human health risk.**

Surface soils have been shown to contain 25 to 625 ng/gram of mercury. Ocean water may contain from 3 ng per liter in the open sea to 5 to 6 ng in coastal waters. Surface waters have less than 50 ng per liter.

Inorganic mercury compounds represent a relatively minor exposure source.

The natural production of methyl mercury by oceanic plankton, bacteria and fungi generate the disproportionately largest human exposure source through the consumption of marine life which have bioaccumulated this form of mercury. Methyl mercury released from these microorganisms also can enter the water or soil and remain there for a long time. It usually stays on soil surfaces and does not move through into the ground water. In water, mercury generally settles to the bottom.

- WHAT HAPPENS WHEN MERCURY ENTERS THE BODY?

The oral consumption of metallic mercury is not associated with ill effects, even in large amounts, unless the exposed individual has a seriously diseased stomach or gastrointestinal tract. Most, if not all of ingested elemental mercury will be excreted via the feces and urine. When exposure is high, the urinary route will dominate. Similarly, inorganic mercury will be eliminated through the same routes. The half-life of elemental mercury is about 50 days when it is inhaled, but may disseminate to the brain and kidneys, where retention may be longer. In the case of inorganic mercury, entry in the body is also easiest via inhalation. Most inorganic mercury is excreted through the urine and kidneys over weeks to months. A small amount may be converted to metallic mercury and is exhaled through the breath as mercury vapors. Only a minimal amount of inorganic mercury is absorbable through the skin. However, even though only up to 15% of an oral dose is absorbed, large amounts are corrosive to the mucosal lining of the GI tract. Most exposure to inorganic mercury is a result of accidental or intention ingestion. Since some of the mercurial salts are used as folk remedies, in religious and/or cult practices in some cultures, chronic ingestion may lead to chronic toxicological effects similar to those of elemental mercury.

In citing his contemporary and friend, Michael Etmuller (1644-1683), Professor of Botany and Surgery at Leipzig, Dr. Bernardo Ramazzini, who subsequently became known as the grandfather of occupational medicine, described a common miner's malady of his day, "within four months they become subject to palsy of the limbs, paralytic, and suffer from vertigo, and that this is caused by the mercurial spirits which are particularly injurious to the nerves." Ramazzini (1633-1714), a medical pioneer, subsequently wrote what has become a famous tract in the annals of occupational medicine, De Morbis Artificum, or Diseases of Workers, in 1713. In this work, Ramazzini first identified the importance of evaluating patients from a perspective of their occupations and admonished his medical successors to inquire as to the potential work exposures of their patients. Obviously, he was cognizant of the bizarre and deadly illnesses that appeared to have a causal association from exposure to mercury, among the miners of his day.

Ramazzini also observed the presence of other serious illnesses resulting from the fumes of metals, such as lead. Among those who worked as potters, he noted "first their hands become palsied, then they become paralytic, splenetic, lethargic, cachectic and toothless, so that one rarely sees a potter whose face is cadaverous and the color of lead." Ramazzini also recognized the potential for harmful exposures among painters, "painters too are attacked by various ailments such as palsy of the limbs, cachexy, blacked teeth, unhealthy complexions, melancholia and loss of the sense of smell. Their sedentary life and melancholic temperament may be partly to blame, for they are almost entirely cut off from intercourse with other men and constantly absorbed in the creations of their imagination. But, for the liability to disease, there is a more immediate cause. I mean the materials of the colors that they handle and smell constantly, such as red lead, cinnabar, white lead, varnish, nut oil and linseed oil, which they used for mixing colors; and the numerous pigments made of various mineral substances. The odors of varnish and the above-mentioned oils make their work rooms smell like a latrine; this is very bad for the head and perhaps accounts for the loss of sense of smell."

“We all know that cinnabar is a product of mercury, that cerissa is made from lead, verdigris from copper, and ultramarine from silver. In fact the mineral world supplies materials to almost any color in use, and this accounts for the really serious ailment that ensue.” An admired predecessor of Ramazzini’s, Jean Fernel of Paris (1497-1558), Chief Physician to Henri II and author of a number of tracts including Concealed Causes, is thought to have described the first case of acute appendicitis with perforation, among others. However, he also anecdotally noted a curious case of a painter of Anjou. This individual was “seized first with palsy of the fingers and hands, later with spasms in these parts, and the arm too was similarly affected; this disorder next attacked his feet; finally he began to be tormented by pain in the stomach and both hypochondria, so violent that it could not be relieved by clysters, fomentations, baths or any other remedy. When the pain came on, the only thing that gave him any relief was for 3 or 4 men to press with their whole weight on his abdomen; this compression of the abdomen lessened the torture. At last, after about three years of this cruel suffering, he died consumptive.” Fernel stated that the most imminent physicians of his day disagreed violently as to the true cause of this terrible disorder. And when Ramazzini read Fernel’s case study, he stated, “I admired the frank confession of Fernel; ‘we were all beside the mark and completely off the track.” However, Fernel went on to say that since this painter was “in the habit of squeezing the color from his brush with his fingers and worse still was imprudent and rash enough to suck it, it is probable that the cinnabar was carried from the fingers to the brain by direct communication and so to the whole nervous system; while that which he took in by the mouth infected the stomach and intestines with the mysterious and malignant qualities, and was the occult cause of those violent pains.”

Unlike in the eras of Fernel and Ramazzini, or even in the height of the industrial revolution in the 19th century UK when mad hatters’ disease was described (“Alice in Wonderland”), occupational mercury toxicity has fortunately become an uncommon entity, at least in the United States and most of the western world. Though approximately 70,000 United States workers annually are potentially exposed to mercury, modern mining and production techniques together with workplace control and regulatory standards representing awareness of the risks involved, have vastly reduced the exposures. The highest remaining work-related risks appear to occur in health services and dental medicine (more about that later).

As noted, release of mercury into the world environment from human activities had been estimated at about 2,000 tons per year, mainly from mining and ore smelting.

Contemporary concerns about the potential for human mercury toxicity, however, are focused on the consumption of organic mercury in foodstuffs, mainly fish and shellfish. Historically, the antifungal and antibacterial properties of organic mercurials lead to their use initially as antisyphilitics and diuretics, and later as seed dressings. It was in this later capacity that several major epidemics occurred in Iraq in 1956 and in 1960, when people ate wheat grain treated with a mercuric fungicide. The largest of these epidemics occurred in Iraq in 1971 and 1972, when some 6500 poisoning cases and 450 recorded deaths resulted from the ingestion of homemade bread made from treated wheat seed. Perhaps the most well-known case of methyl mercury poisoning took place in Minnemata, Japan, in 1956, when inorganic mercury affluent from factories was methylated by microbiota of the Bay and concentrated in local fish. Residents of this fishing village consumed these fish, which caused devastating

developmental anomalies in some 25 infants born to mothers eating large quantities. The mothers themselves were affected little, or none at all, but the infants developed cerebral palsy-like syndromes with severe mental retardation.

Methyl mercury, the most common and potentially injurious form of organic mercury, is more than 90% absorbed through the gastrointestinal tract. Once it enters the blood stream, organic mercury crosses into most tissues easily, including the brain and placenta. In the body, methyl mercury can be converted into inorganic mercury, which renders it less readily eliminated. It generally leaves the body slowly, equilibrating, like all ions, with the external environment gradually over a several-month period, mostly as inorganic mercury in the feces.

- ## WHAT ARE THE POTENTIAL ADVERSE HEALTH EFFECTS OF MERCURY POISONING?

Many factors determine the potential effects of exposure to any substance. Namely:

A. Dose (how much)
B. Duration (how long)
C. Route (how it was contacted)
D. Personal characteristics including your age, sex, diet, family/genetic traits, lifestyle and general state of health, may also have an influence.

Acute inhalation exposure to elemental mercury can irritate the mucosal linings of the mouth and GI tract and range from mild gastritis to severe ulceration. Dependent upon duration and intensity of the exposure, there may be nausea, vomiting, diarrhea, eye irritation, skin rashes or alterations in blood pressure or heart rate. Chronic exposure to airborne vapors may lead to the classic triad of gingivostomatitis, tremor and a collection of neuropsychiatric symptoms known as erythism, which include fatigue, insomnia, mood changes, anorexia, nervousness, irritability and memory dysfunction.

The target organs of inorganic mercury poisoning are the GI tract and the kidneys. On an acute basis, many of the same effects as noted with metallic mercury poisoning may be seen, including nausea, vomiting and diarrhea. If these symptoms are severe enough, they may be accompanied by signs of acute renal failure. Chronic effects are similar to those of elemental mercury exposure. Children who breathe metallic mercury regularly or are long-term uses of inorganic mercury salts or mercuric skin ointments may develop a condition known as acrodynia, or pink disease. In addition to redness and peeling of the skin, they may have leg cramps, irritability, excess salivation, sweating, fever, insomnia or weakness. This syndrome is believed to be due to a sensitivity reaction. Cases in teenagers and adults also have been seen.

There is no scientific evidence to suggest an increased incidence of cancer of any type with exposure to elemental or inorganic mercury.

The effects of organic mercury toxicity tend to be developmental and/or insidious. Little information exists regarding any acute lethal dose, and the effects seen are largely chronic in nature. The central nervous system is especially vulnerable to the toxic effects of organic mercury. Neurological disorders, as described by Fernel and Ramazzini, include impaired vision and hearing, slurred speech, gait disturbances, muscle weakness, memory loss, irritability and insomnia. Methyl mercury is not usually associated with GI or renal toxicity.

Increased cancer incidence as a consequence of exposure to methyl mercury has not been observed in experimental animal studies.

- WHAT ARE THE BEST TESTS TO DETERMINE WHETHER EXPOSURE HAS OCCURRED?

The best test for inorganic mercury is urine measurement. For occupationally exposed individuals, semi-annual to annual 24-hour urine mercury determinations should be performed, along with periodic physical examinations. The recommended threshold limit value (TLV) for an 8-hour time weighted average (TWA) working day and 40-hour work week for mercury vapor and inorganic and non-alkyl organic mercurials is 0.05 mg per cubic meter in the U.S. and EEC. This air concentration of mercury corresponds to a urinary concentration of about 50 mcg per liter and a blood concentration of 30 to 35 mcg per liter. The U.S. and Environmental Protection Administration (EPA) recommends that ambient air level for population exposure of less than 10 to 20 ng per cubic meter. There also are atmospheric discharge limits for industrial facilities and sludge incineration and drying processes. Non-occupationally exposed persons generally have less than 5 mcg per gram of creatinine, or less than 10 mcg per liter of urine and less than 2 mcg per deciliter in whole blood. Adults excreting less than 50 mcg of mercury per gram of creatinine are not likely to experience renal dysfunction. Again, for diagnosing acute mercury exposure, a quantitative 24-hour urinary mercury excretion measurement is the best available means of assessment.

- WHAT ABOUT HAIR TESTING?

Many people call or come in to clinics with “elevated” hair tests results. First of all, studies of commercial laboratories have demonstrated poor reliability and consistency. Their use may be more applicable to epidemiologic studies than for individual clinical testing. Secondly, hair samples themselves are not all that useful. The half-life of hair is less than 50 days, especially in some people with short hair. The best response to an allegedly elevated hair level is to perform blood and urine mercury tests. These will most likely prove to be normal.

Blood mercury levels and absent urine mercury levels are, by definition, indicative of organic mercury exposure. In the absence of detectable urine mercury, blood mercury levels most often reflect dietary intake of seafood, which may be even higher with large fish. On average, the following rule of thumb is helpful:

1. Less than two fish meals per week leads to an average of 0.50 mcg per ml of blood mercury level.
2. Greater than or equal to four fish meals per week may lead to 0.85 mcg Hg per ml.
3. Greater than four fish meals per week may correlate to approximately 4.5 mcg Hg per ml.

Most often, even individuals consuming large quantities of fish containing mercury are asymptomatic, even in the face of elevated mercury blood levels. For example, a middle aged man ate fish 2 to 3 times per week, including sword fish and shark. He had no other work or environmental exposure. His blood mercury level was 8 mcg per liter, and his urine mercury was non-detectable. He was asymptomatic. Another man, 54 years old, had a history of having consumed a can of tuna fish daily for five years. His blood mercury level was 52 mcg per liter with none in the urine. He also was asymptomatic. He discontinued his tuna fish, and the level was reduced to about ½ in 80 days and to 7 mcg per liter after seven months.

The Take-Home Message: Methyl mercury is the most common population exposure (through fish consumption), 90% bound to red blood cells and mainly excreted in feces, not urine. The threshold for early signs of neurotoxicity in adults hypothetically ranges from 50 to 200 mcg per liter in the blood. Early symptoms of intoxication might include paresthesias in the fingers, tongue, face and decreased visual fields, impaired hearing, gait and speech disturbance. Obviously, by the examples, you can readily see that it takes a fair amount of mercury in the blood to lead to a symptomatic, adverse effect level, and there is a fairly rapid drop-off when the source is discontinued. The EPA is currently considering a recommendation of keeping your blood lead level at less than 5 mcg per liter if you are a female attempting to become pregnant. “The best advice is to avoid fish species with the highest average amounts of methyl mercury, e.g., King Mackerel,tilefish, shark, sword fish and tuna. According to the latest EPA advisory for pregnant women, keep your daily exposure below 0.1 mcg per kilogram body weight per day. You can find mercury levels in various fish species on the EPA web site. Remember, this recommendation is arbitrarily fabricated for the protection of the developing fetus, not the adult.

- <u>WHAT ABOUT DENTAL AMALGAMS</u>?

The short answer is that there is no evidence to show any adverse health effects associated with dental amalgam fillings and mercury exposure. Moreover, the treatment is a potentially hazardous proposition. That is, drilling out mercury aerosolizes more mercury than eating saliva containing small amounts of mercury that may have leached out of dental fillings. There is an enormous amount of misinformation circulating in various venues on this topic. Dentists, dental hygienists and other medical professionals may, however, be at risk from the continuous inhalation of aerosolized mercury accidentally dropped in an office setting and residing in carpets and other textiles and fabrics.

Another subject fraught with extraordinarily bad information relates to the popular allegation of an association between autism and exposures to Thimerosal, a mercuric

preservative used in some vaccines in the U.S. until about 2001. According to the CDC, the American Academy of Pediatrics, the Institute of Medicine and the National Academy of Sciences, there is no evidence to support any such association, and overwhelming evidence to the contrary. Further, no U.S. manufactured pediatric vaccines currently contain any Thimerosal. Unfortunately, for the developing world, where refrigeration is largely nonexistent, this is an especially costly trade-off.

- SUMMARY

Mercury exposure through ambient air and water does not represent a significant toxicologic risk to the general population. Accidental poisoning cases in the United States are now rare. Two boys died in New Mexico in 1969 after consuming meat from a hog fed seed grain treated with a methyl mercury compound. And, in 1998, a university chemistry professor and researcher accidentally exposed to dimethyl mercury experienced the rapid onset of neurologic symptoms and subsequently died. Consequently, fish consumption, practically speaking, represents the primary source of mercury exposure to the U.S. population.

In 1978, the World Health Organization established a standard of allowable daily intake of 0.5 mcg of mercury per kilogram body weight per day. A recent recommendation by the EPA reduced the safe daily allowance to 0.1 mcg mercury per kilogram. This level translates into a weekly consumption level of one 7 ounce can of tuna for an adult. Since canned tuna is both the cheapest and most widely consumed fish in the U.S. and approved by the American Heart Association as part of a diet low in saturated fat and cholesterol, this discussion will most likely continue. Fish is generally considered an excellent source of dietary protein and the associated health benefits, including reduction of the incidence of coronary artery disease, are well recognized. The FDA has recommended that pregnant women, nursing mothers and young children avoid eating fish with a high mercury content (greater than 1 PPM), such as shark, sword fish, tile fish and King Mackerel, and also whale meat (up to 3 PPM). For the general population, the FDA advises limiting the regular consumption of shark and swordfish (which typically contain about 1ppm of methylmercury)to about 7 oz. /week (one serving). No consumption advice is felt necessary by the FDA for the top ten seafood species, representing about 80% of the seafood market: canned tuna, shrimp, Pollock, salmon, cod, catfish, clams, flatfish, crabs and scallops.The methylmercury in these fish is generally less than 0.2ppm, and few people eat more than the suggested weekly limit of 2.2 pounds. However, a major study in the Seychelles Islands showed an average weekly fish consumption of about 12 meals per week, and mercury concentrations in the hair of the Seychellois are 10 to 20 times those found in the U.S. This child development study covered a time frame of about nine years and demonstrated no independent adverse health effects from fish consumption. As previously noted, the FDA has estimated that most Americans ingest about 50 ng per kilogram per day of mercury with a range of 50 to 100 ng, or 3.5 to 7.0 mcg of mercury per day. This level has not been to be associated with any adverse health effects. There is, no current medical information to support any changes in dietary recommendations or environmental health practices other than relying upon those standards in place as reasonable guidelines.

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DONALD J. McGRAW, M.D., MPH, FACPM, FACOEM, FRSM

Occupational and Environmental Medicine, LLC
Pittsburgh, Pennsylvania

EDUCATION: M.P.H. (Occupational and Environmental Medicine) 1980 - The Johns Hopkins University
Residency and Fellowship 1978-1980 - The Johns Hopkins University
Internship and Residency 1976-1978 - McGill University
M.D. 1976 - State University of New York at Stony Brook, New York

PROFESSIONAL CERTIFICATION AND LICENSURE: Pennsylvania, Formerly, Ohio, Maryland, Georgia and West Virginia.

PROFESSIONAL AFFILIATIONS:

Diplomat, American Board of Preventive Medicine
Fellow, American College of Occupational and Environmental Medicine
Fellow, American College of Preventive Medicine
Fellow, Royal Society of Medicine
Member, American College of Physicians
Certified Medical Review Officer (MRO)
Adjunct Assoc. Prof. of Occ. Medicine - University of Pittsburgh Schools of Medicine and Public Health
Clinical Instructor in Medicine/University of Pittsburgh School of Medicine
Allegheny County Medical Society
Pennsylvania Medical Society
American Board of Independent Medical Examiners
American College of Forensic Examiners
American College of Occupational and Environmental Medicine
Pittsburgh Association of Occupational Medicine
Tri-state Occupational Medicine Association
Member of Faculty of the Johns Hopkins University School of Medicine and Deputy Director, Dept. of Occupational Medicine, 1980-1982.
Member of Adjunct Faculty of University of Pittsburgh Schools of Medicine and Public Health, 1981-Present.
Director of Occupational Medicine Clinic for Dept. of Medicine and later GSPH, 1990-1992.
Director of Occupational/Environmental Medicine, Shadyside Hospital, 1992-1998.
Attending Physician, Presbyterian University Hospital, Shadyside Hospital and the Rehabilitation Institute (Children's Institute), 1990-Present.
Attending Physician Western Pennsylvania Hospital, Jan. 2005 - present.
Taught annual sections for GSPH, Occ. Med. Residents Dept. of Occupational Medicine annually since 1982 to present. These have included sections on pulmonary medicine, dermatology and musculoskeletal/rheumatology.
Has taught seminars on Multiple Chemical Sensitivity, Workers Compensation and Independent Medical Evaluations, Dermatology and Occupational Medicine to GSPH Occ. Med. Residents and other allied health students and graduate students for 10-15 years.
Medical students and residents have completed elective medical rotations in office.
Taught medical students section on Occupational Medicine and General Internal Medicine intermittently since 1982.
Taught summer term courses to Occupational Medicine Residents on various topics.
Participated in Occupational Medicine Resident Journal Club for many years.
Taught section on clinical skills to 3rd year Medical Students (Introduction to Patient Care) intermittently in 1990's and earlier.
Long standing Member of Residing Advisory Committee GSPH, since 1980's.
Member Occupational Med. Committee (Alleheny County Medical Society) since 1982.
Member Pittsburgh Association of Occ. Medical Directors since 1982.

DONALD J. McGRAW, M.D., MPH, FACPM, FACOEM, FRSM

Occupational and Environmental Medicine, LLC
Pittsburgh, Pennsylvania

FIELDS OF SPECIALIZATION:
Occupational Medicine, Environmental Medicine, Physical Medicine/Rehabilitation/Workers Compensation, Disability Evaluation, Toxicology Consultation, Epidemiology.

EXPERIENCE SUMMARY

Dr. McGraw's experience in occupational and environmental medicine has encompassed the health and safety oversight of thousands of employees for a Fortune 500 company as Corporate Medical Director and Vice-President of Science and Technology. His responsibilities included management of scientists, technicians, toxicologists, industrial hygienists, safety specialists, nurses and physicians. Medical surveillance, product safety and regulatory compliance also were among his administrative responsibilities. The range of product lines and facilities included in his purview and inspections covered coke ovens, coal tar distillation plants, wood treatment operations (including creosote, CCA, and pentachlorophenol), paints and coatings lines, resins and industrial coatings, road material quarries, crushing and distribution operations and metal products manufacturing.

Dr. McGraw was a faculty member at The Johns Hopkins University for several years and at the University of Pittsburgh for more than 20 years.

At the latter, he has taught medical students and residents, as well as allied health sciences students for many years. He has been Director of Occupational and Environmental Medicine at both Presbyterian University Hospital and Shadyside Hospital where he has evaluated and treated patients regularly for the past twenty-five years.

In addition, Dr. McGraw has provided expert testimony for corporations in product liability cases and on behalf of businesses and insurance companies in evaluating countless workers compensation claimants over many years. This has included depositions, record reviews, patient evaluations and trial testimony.

As an attending physician for several major regional hospitals, Dr. McGraw has served on numerous hospital and foundation boards and committees and been active in all aspects of hospital affairs.

PRESENTATIONS

10 Nov 1994	Western Pennsylvania Association of Healthcare Risk Management Fall Symposium – "Managed Care"
Mar 2000	Western Pennsylvania Safety Council "At Ease with IME's"
9 Feb 1996	Fifth Interdisciplinary Grand Rounds, Naples, FL. Shadyside Hospital "Alcoholism: An Update"
30 Jun, 1981	"The Impact of Poly-Chlorinated Biphenyls on the Human Environment", U.S. Office of Technology Assessment.
Apr 1982	"Preventive Medicine in the Geriatric Patient", Levindale Hebrew Geriatric Center and Hospital, Baltimore, MD.
10-11 May 1982	Symposium "Reproductive Health Policies in the Workplace", University of Pittsburgh, Graduate School of Public Health. "Corporate Policies and Practices Concerning Reproductive Health, An Overview".
18 May 1982	"Keeping the Frail Elderly in the House", City of Baltimore. "Commission on Aging and Retirement Education", Baltimore, MD.
Oct 1982	"Evaluation of Occupational and Environmental Health Hazards", Franklin Square Hospital, Baltimore, MD.

PRESENTATIONS (Continued)

Apr 1983	"Occupational Medicine – Career Planning and Opportunities", University of Pittsburgh, School of Medicine, Pittsburgh, PA.
20 May 1983	Teaching Panelist, clinical skills case presentation for second year medical students, University of Pittsburgh, School of Medicine, Pittsburgh, PA.
1983-87	Teaching Faculty, University of Pittsburgh, School of Medicine, Course in Occupational Medicine Annual Seminar in Occupational Dermatology 1983 – 1988. Program for students in Schools of Medicine, Nursing, Industrial Hygiene (GSPH).
Apr 1984	Special presentation to 2nd and 3rd year medical students at the University of Pittsburgh, School of Medicine, "Occupational Medicine".
Sep-Oct 1985	Preservative Review Workshops, "The Toxicological Status of Arsenic, Pentachlorophenol and CCA", San Francisco, CA; Houston, TX; Atlanta, GA; Chicago, IL; Pittsburgh, PA.
Jan 1992	"Neurotoxicity", Department of Neurosurgery, University of Pittsburgh, Presbyterian University Hospital, Pittsburgh, PA.
17 Mar 1992	"Multiple Chemical Sensitivities". Department of Outpatient Psychiatry, WPIC, University of Pittsburgh, Pittsburgh, PA.
Apr 1992	"Panel Member, MIT Enterprise Forum of Pittsburgh, Cardiac Telecom Corporation Case Assessment. Engineers Club, Pittsburgh, PA.
Apr 1992	"Hospital Involvement with Community Programs", Hospital Trustee Association of Pennsylvania, Sheraton, Pittsburgh, PA.
5 Nov 1992	Presentation to Shadyside Hospital Medical Staff and Administration, "Managed Care Workers' Compensation".
13 Feb 1993	"Medical Evaluations and Managed Care Services: A Multidisciplinary Approach to Treatment", Interdisciplinary Grand Rounds, Longboat Key, FL.
28 Apr 1993	Presentation to: Pittsburgh Society of Public Accountants, "Changing Environment of Health Care Delivery in South Western Pennsylvania, Pittsburgh, PA.
Feb 1994	"Multiple Chemical Sensitivities", Interdisciplinary Grand Rounds, Boca Raton, FL.
25 Feb 1995	"New Opportunities in Managed Care", Lite-Care Conference, Pittsburgh, PA.
26 May 2004	"How to Choose a Medical Specialist/Consultant", Western Pennsylvania Safety Council, Annual Safety, Health, Environmental & Security Conference.
30 Jan 2005	"Historical Medical Uses of Benzene, Tar and Tar Derivatives", Natural Gas Technologies (GTI) Conference, Orlando, Fla.

DONALD J. McGRAW, M.D., MPH, FACPM, FACOEM, FRSM

Occupational and Environmental Medicine, LLC
Pittsburgh, Pennsylvania

PUBLICATIONS

Editor and Writer (Silent): Working with Older People, Clinical Aspects of Aging), A Guide to Practice, William Reichel 1976, USPHS, HSS.

Clinical Aspects of Aging, William Reichel, 1978, Williams and Wilkins Company.

The Geriatric Patient, William Reichel, 1978, Hospital Practice and Publishing Company.

Editor, "Post-Graduate Medicine", 1983-1990, Reviewed and critiqued dozens of submitted articles on topics of general medicine.

"Effects of Somatostatin on 14C – Glucose Metabolism in Rat Adipose Tissue", submitted but unpublished, D.J. McGraw, G.G. Murthy, 1976, Stony Brook, NY.

"The Medical Bookshelf", A review of Procedures and Techniques in Emergency Medicine", Post-Graduate Medicine, September 1983, pp. 52, 55.

"Utilization Review Strategies and Outlook", Shadyside Hospital Physician's Newsletter, Summer 1993, Vol. 7, No. 3.

"Utilization Review: Update on Pre-Certification", Shadyside Hospital Physician's Newsletter, September 1995, Vol. 5, No. 4.

"Low Back Pain", Shadyside Hospital Physician's Newsletter, November-December 1996, Vol. 10, No. 9.

TABLE 72-1. Inorganic and organic mercurial compounds and their uses

Inorganic	Ammoniated mercury ($HgNH_2Cl$)—antiseptic
	Mercuric acetate [$Hg(OOC_2H_3)_2$]—catalyst in organic synthesis, pharmaceuticals
	Mercuric arsenate ($HgNH_3O_4$)—waterproofing and antifouling paints
	Mercuric benzoate [$Hg(C_7H_5O_2)_2$]—antisyphilitic
	Mercuric bromide ($HgBr_2$)—medicinal use
	Merbromin (Mercurochrome; 25% mercury + 20% bromine)—antiseptic cream
	Mercurous chloride (calomel, mercury monochloride, Hg_2Cl_2)—a laxative
	Mercuric chloride (corrosive sublimate, mercury bichloride)—antiseptic solution
	Mercury cyanate [fulminate of mercury, $Hg(CNO)_2$]—explosive
	Mercuric cyanide [$Hg(CN)_2$]—antiseptic, photography
	Mercuric oxide, red (red or yellow precipitate Hg^0)—pigment, dry batteries
	Mercuric potassium cyanide—silvering glass, in mirrors
	Mercuric sulfide (cinnabar, red vermilion, Chinese red)—used in tattoos, combined with cadmium sulfide
	Mercuric salicylate (salicylate mercury)—topical antiseptic
	Mercuric acetate [$Hg(OOC_2H_3)_2$]
	Sublimate ($HgCl_2$)
Organic	Thimerosal (Merthiolate; 49% mercury)
	Alkyl mercury fungicides: dialkyl mercury, ethyl mercury
	Phenyl mercury fungicides ($PhHg^+$): phenyl mercury
	Alkoxyalkyl mercury fungicides: methoxyethyl mercury
	Mercurial diuretics (Mersalyl, Chlormerodrin)

TABLE 72-2. Products and industries associated with potential mercury exposure

Elemental mercurials
- Dental medicine
- Batteries
- Barometers
- Boiler makers
- Calibration instruments
- Caustic soda production
- Carbon brush production
- Ceramics
- Chloralkali production
- Ultrasonic amplifiers
- Direct current meters
- Infrared detectors
- Electrical apparatus
- Electroplating
- Fingerprint detectors
- Silver and gold extraction
- Jewelry
- Fluorescent, neon, and mercury arc lamps
- Manometers
- Paints
- Paper pulp manufacturing
- Photography
- Pressure gauges
- Thermometers
- Semiconductor solar cells

Inorganic mercurials
- Disinfectants
- Paints and dyes
- Explosives
- Fireworks manufacturing
- Fur processing
- Ink manufacturing
- Chemical laboratory workers
- Percussion caps and detonators
- Spermicidal jellies
- Tannery workers
- Wood preservatives
- Tattooing materials
- Taxidermists
- Vinyl chloride production
- Mercury vapor lamps
- Antisyphilitic agents
- Thermoscopy
- Silvering in mirrors
- Photography
- Perfumery and cosmetics
- Acetaldehyde production

Organic mercurials
- Bactericides
- Embalming preparations
- Paper manufacturing
- Farmers
- Laundry and diaper services
- External antiseptics
- Fungicides
- Insecticide manufacture
- Seed handling
- Wood preservatives
- Germicides

TABLE 72-3. Mercury regulations and guidelines

Agency	Description	Concentration
International: WHO		
Guidelines	Drinking-water guideline values (applies to all forms of mercury)	0.001 mg/L
Regulations	Permissible tolerable weekly intake	5 μg/kg total 3.3 g/kg CH_3Hg
United States		
Regulations: Air		
OSHA	Alkyl compounds—PEL, TWA	0.01 mg/m³
	Inorganic mercury (skin)	0.05 mg/m³—TWA
	Alkyl compounds	0.03 mg/m³ (skin)—STEL
Guidelines		
Air		
ACGIH	Ceiling-alkyl compounds—STEL	0.03 mg/m³
	Alkyl compound—TWA	0.01 mg/m³
	Aryl compounds	0.1 mg/m³
	Metallic mercury and inorganic compounds	0.025 mg/m³
NIOSH	Aryl or inorganic mercury as mercury—REL for occupational exposure (8-h TWA)	0.1 mg/m³ ceiling (skin)
	Mercury (organic) alkyl compounds	0.01 mg/m³—TWA
	Mercury vapor as mercury	0.03 mg/m³ (skin)—STEL
		0.05 mg/m³ (skin)—TWA
Water		
EPA	Inorganic mercury—Lifetime Health Advisory (adult)	0.002 mg/L
	Inorganic mercury—Longer-Term Health Advisory (adult)	0.002 mg/L
	Drinking water equivalent level	0.002 mg/L
	Mercury and phenylmercuric acetate—Ambient Water Quality Criteria for Human Health	
	Water and fish	0.05 μg/L
	Fish only	0.051 μg/L
	Mercury and phenylmercuric acetate as mercury—Ambient Water Quality Criteria for Aquatic Organisms	
	Acute (1-h average)	Marine: 1.8 μg/L; freshwater: 1.4 μg/L
	Chronic (4-d average)	Marine: 0.94 μg/L; freshwater: 1.77 μg/L
	National Primary Drinking Water Regulations	
	MCLGs for inorganic compounds	0.002 mg/L
	MCL for inorganic compounds	0.002 mg/L
Food		
FDA	Action level for poisonous or deleterious substances in human food and animal feed—fish, shellfish, crustaceans, other aquatic animals (fresh, frozen, or processed)	1 ppm
	Bottled water	0.002 mg/L

ACGIH, American Conference of Governmental Industrial Hygienists; EPA, U.S. Environmental Protection Agency; FDA, U.S. Food and Drug Administration; IRIS, integrated risk information system; MCL, maximum contamination level; MCLG, maximum contamination level goal; NIOSH, National Institute for Occupational Safety and Health; OSHA, U.S. Occupational Safety and Health Administration; PEL, permissible exposure limit; ppm, parts per million; REL, recommended exposure limit; STEL, short-term exposure limit; TWA, time-weighted average; WHO, World Health Organization.
From ref. 2, with permission.

TABLE 72-4. Events and regulatory decisions associated with methyl mercury

1953	In Minamata, Japan, 111 people die or suffer nervous system damage from consuming fish from waters severely polluted by mercury from industrial discharges.
1965	In Niigata, Japan, 120 people are poisoned by consuming fish polluted by methyl mercury.
1969	The EPA sets a 0.5-ppm limit for total mercury in fish.
1969	Swedish researchers discover that methyl mercury accumulates in fish.
1969	The FDA sets a 0.5-ppm action level as the maximum safe limit for total mercury in fish. Action levels are the limit at or above which the FDA will act to remove a product from the market.
1971–1972	A methyl mercury poisoning outbreak occurs in Iraq when seed grain treated with a methyl mercury fungicide is ingested. Children born to mothers who were pregnant at the time they ate the grain were found to experience neurologic effects, delayed development, and delayed motor skills.
1979	The FDA raises the mercury action level to 1 ppm based, in part, on a National Marine Fisheries Service study that showed this level would adequately protect consumers.
1980	The WHO publishes a study on methyl mercury toxicity that states that "the general population does not face a significant health risk from methyl mercury."
1984	The FDA changes the basis for enforcement of the mercury action level from total mercury to methyl mercury.
1984	The NIEHS and Rochester University begin a study in the Seychelles Islands, where fish is a major source of protein, to track prenatal exposure to methyl mercury and effects on the fetus.
1991	Under mandate of the Clean Air Act amendments, the EPA begins an assessment of an acceptable level of methyl mercury in fish to be completed by 15 December 1993.
1992	The NIEHS and Odense University begin a study of methyl mercury effects on a fish-eating population in the Faroe Islands.
1993	The Sierra Club and the Natural Resources Defense Council sue the EPA to complete and release its methyl mercury report. The EPA is granted a 1-yr extension.
1994	The EPA delays release of its report. The environmental groups sue again. The EPA is ordered to submit its report by 15 April 1993.
1995	The EPA misses the deadline for release of its report. EPA officials say the delay is due to waiting for additional data from the Seychelles studies.
1995–1996	Initial results of the Seychelles studies show no major health problems. Initial results of the Faroe Islands study, released in 1994, however, indicate neuropsychological dysfunction in children with increased methyl mercury exposure levels.
1996	The EPA has yet to release its final report, although the agency has called for a stricter standard for methyl mercury in fish of 0.1 μg/kg body wt/d.

EPA, U.S. Environmental Protection Agency; FDA, U.S. Food and Drug Administration; NIEHS, National Institute of Environmental Health Science; WHO, World Health Organization.

Adapted from Wheeler M. Focus: measuring mercury. *Environ Health Perspect* 1996;104:826–831.

Table 5-3. Atmospheric Mercury Emission Inventory for the United States by Anthropogenic Source Type[a]

Source type	Mg/yr[-1]	% of total emissions	% mercury species		
			Hg^0	Hg^{2+}	Hg_p
Medical waste incineration	58.6	26	20	60	20
Municipal waste collection	49.8	22	20	60	2
Electric utility boilers (coal, gas, oil)	48.5	22	50	30	20
Non-utility power and heat generation	28.5	13	50	30	20
Non-ferrous metal smelting	8.7	4	85	10	5
Chloralkali factories	6.5	3	70	30	0
Other point sources	16.2	7	80	10	10
Area sources (e.g., dental amalgams, fluorescent lighting fixtures)	6.9	3	100	0	0
Total	223.7	100%	41%	41%	18%

[a] Emission rates are specified in units of megagrams per year ($Mg\ yr^{-1}$)

Hg^0 = elemental mercury vapor; Hg^{2+} = mercuric form; Hg_p = mercury associated with particulates

Source: Bullock 1997

Table 5-13. Mercury Concentrations in the Top 10 Types of Fish Consumed by the U.S. Population

Fish	Mercury concentration (ppm)[a]	Comments
Tuna	0.206	Mercury content is the average of the mean concentrations in 3 tuna species: Albacore tuna (0.264 ppm) Skipjack tuna (0.136 ppm) Yellowfin tuna (0.218 ppm) The FDA measured the methylmercury concentration in 220 samples of canned tuna in 1991; the average amount of methylmercury measured was 0.17 µg/g and the range was <1–0.75 µg/g) (Yess 1993).
Shrimp	0.047	Mercury content is the average of the mean concentrations in 7 shrimp species: Royal red (0.074 ppm) White (0.054 ppm) Brown (0.048 ppm) Ocean (0.053 ppm) Pink (0.031 ppm) Pink northern (0.024 ppm) Alaska (sidestripe) (0.042 ppm)
Pollack	0.150	The Pesticide and Chemical Contaminant Data Base for the FDA (1991/1992) reports the methylmercury concentration in pollack in commerce as 0.04 ppm
Salmon	0.035	Mercury content is the average of the mean concentrations in 5 salmon species: Pink (0.019 ppm) Chum (0.030 ppm) Coho (0.038 ppm) Sockeye (0.027 ppm) Chinook (0.063 ppm)
Cod	0.121	Mercury content is the average of the mean concentrations in 2 cod species: Atlantic (0.114 ppm) Pacific (0.127 ppm)
Catfish	0.088 0.160	Two data sets were collected from U.S. freshwater sources: Bahnick et al (1994): channel, largemouth, rock, striped, and white Lowe et al. (1985): channel and flathead. Neither survey included farm-raised catfish, which is the type predominantly consumed in the U.S. Mercury content of farm-raised catfish may be significantly different from feral catfish.
	0.020	The Pesticide and Chemical Contaminant Data Base for USFDA (1991/1992) reports the methylmercury concentration in catfish as 0.02 ppm.
Clam	0.023	Mercury content is the average of the mean concentrations in 4 clam species: Hard (quahog) (0.034 ppm) Pacific littleneck (0 ppm) Soft (0.027 ppm) Geoduck (0.032 ppm)

Table 5-13. Mercury Concentrations in the Top 10 Types of Fish Consumed by the U.S. Population (continued)

Fish	Mercury concentration (ppm)[a]	Comments
Flounder (flatfish)	0.092	Mercury content is the average of the mean concentrations in 9 flounder species: Gulf (0.1487 ppm) Summer (0.127 ppm) Southern (0.076 ppm) Four-spot (0.090 ppm) Windowpane (0.151 ppm) Arrowtooth (0.020 ppm) Witch (0.083 ppm) Yellowtail (0.067 ppm) Winter (0.066 ppm)
Crab	0.117	Mercury content is the average of the mean concentrations in 5 crab species: Blue (0.140 ppm) Dungeness (0.183 ppm) King (0.070 ppm) Tanner *(C. opilio)* (0.088 ppm) Tanner *(C. bairdi)* (0.102 ppm)
Scallop	0.042	Mercury content is the average of the mean concentrations in 4 scallop species: Sea (smooth) (0.101 ppm) Atlantic bay (0.038 ppm) Calico (0.026 ppm) Pink (0.004 ppm)

[a] All concentrations determined on a wet weight basis

Source: EPA 1996e

Table 2-4. Half-lives of Inorganic Mercury in Humans

Tissue	Half-life	Phase	Reference
Lung	2 days	Early phase	Berlin et al. 1969a
Brain	20 days	Biphasic	Hursh et al. 1976
Blood	3.3 days	Early phase	Cherian et al. 1978
Plasma	3.3 days	Early phase	Cherian et al. 1978
Blood	2.4 days	Early phase	Clarkson 1978
Blood	15 days	Late phase	Clarkson 1978
Blood	28 days	Late phase	Rahola et al. 1973
Whole body	60 days		Rahola et al. 1973
Whole body	60 days		Hursh et al. 1976
Kidney	60 days		Hursh et al. 1976

Compiled from: Bakir et al. 1973; Cox et al. 1989; Kershaw et al. 1980; Miettenen et al. 1971; Sherlock et al. 1984

Table 5-12. Estimated Average Daily Intake and Retention of Total Mercury and Mercury Compounds in the General Population

Source of exposure	Elemental mercury vapor	Inorganic mercury compounds	Methylmercury
Air	0.030 (0.024)	0.002 (0.001)	0.008 (0.0064)
Food			
Fish	0	0.600 (0.042)	2.4 (2.3)
Non-fish	0	3.6 (0.25)	0
Drinking water	0	0.050 (0.0035)	0
Dental amalgams	3.8–21 (3–17)	0	0
Total	3.9–21 (3–17)	4.3 (0.3)	2.41 (2.31)

Note: Values given are the estimated average daily intake (in µg/day) for adults in the general population who are not occupationally exposed to mercury; the figures in parentheses represent the estimated amount retained in the body of an adult.

Source: WHO 1990, 1991