Mercury poisoning

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Classification and external resources

Elemental mercury

ICD-10	T56.1 (http://apps.who.int/classifications/apps/icd/icd10online/?gt51.htm+t561)
ICD-9	985.0 (http://www.icd9data.com/getICD9Code.ashx?icd9=985.0)
DiseasesDB	8057 (http://www.diseasesdatabase.com/ddb8057.htm)
MedlinePlus	002476 (http://www.nlm.nih.gov/medlineplus/ency/article/002476.htm)
eMedicine	emerg/813 (http://www.emedicine.com/emerg/topic813.htm)

Mercury poisoning (also known as **hydrargyria** or **mercurialism**) is a disease caused by exposure to mercury or its compounds. Mercury (chemical symbol Hg) is a heavy metal that occurs in several forms, all of which can produce toxic effects in high enough doses. Its zero oxidation state Hg^0 exists as vapor or as liquid metal, its mercurous state Hg^+ exists as inorganic salts, and its mercuric state Hg^{2+} may form either inorganic salts or organomercury compounds; the three groups vary in effects. Toxic effects include damage to the brain, kidney, and lungs.[1] Mercury poisoning can result in several diseases, including acrodynia (pink disease), Hunter-Russell syndrome, and Minamata disease.[2]

Symptoms typically include sensory impairment (vision, hearing, speech), disturbed sensation and a lack of coordination. The type and degree of symptoms exhibited depend upon the individual toxin, the dose, and the method and duration of exposure.

Contents

Signs and symptoms

Common symptoms of mercury poisoning include peripheral neuropathy (presenting as paresthesia or itching, burning or pain), skin discoloration (pink cheeks, fingertips and toes), swelling, and desquamation (shedding of skin).

Because mercury blocks the degradation pathway of catecholamines, epinephrine excess causes profuse sweating, tachycardia (persistently faster-than-normal heart beat), increased salivation, and hypertension (high blood pressure). Mercury is thought to inactivate S-adenosyl-methionine, which is necessary for catecholamine catabolism by catechol-o-methyl transferase.

Affected children may show red cheeks, nose and lips, loss of hair, teeth, and nails, transient rashes, hypotonia (muscle weakness), and increased sensitivity to light. Other symptoms may include kidney disfunction (e.g. Fanconi syndrome) or neuropsychiatric symptoms such as emotional lability, memory impairment, or insomnia.

Thus, the clinical presentation may resemble pheochromocytoma or Kawasaki disease.

An example of desquamation of the hand of a child with severe mercury poisoning acquired by handling elemental mercury is this photograph (http://adc.bmj.com/content/vol86/issue6/images/large/90015199.f1.jpeg) in Horowitz, *et al.* (2002).[3]

Causes

The consumption of fish is by far the most significant source of ingestion-related mercury exposure in humans and animals, although plants and livestock also contain mercury due to bioaccumulation of mercury from soil, water and atmosphere, and due to biomagnification by ingesting other mercury-containing organisms.[4] Exposure to mercury can occur from breathing contaminated air;[5] from eating foods containing mercury residues from processing, such as can occur with high-fructose corn syrup;[6] from exposure to mercury vapor in mercury amalgam dental restorations;[7] and from improper use or disposal of mercury and mercury-containing objects, for example, after spills of elemental mercury or improper disposal of fluorescent lamps.[8]

Consumption of whale and dolphin meat, as is the practice in Japan, is a source of high-levels of mercury poisoning. Tetsuya Endo, a professor at the Health Sciences University of Hokkaido, has tested whale meat purchased in the whaling town of Taiji and found mercury levels that are more than 20 times acceptable Japanese standards.[9]

Human-generated sources such as coal plants emit approximately half of atmospheric mercury, with natural sources such as volcanoes responsible for the remainder. An estimated two-thirds of human-generated mercury comes from stationary combustion, mostly of coal. Other important human-generated sources include gold production, non-ferrous metal production, cement production, waste disposal, human crematoria, caustic soda production, pig iron and steel production, mercury production (mostly for batteries), and biomass burning.[10]

Small independent gold mining operations employ workers who are exposed to more risk to mercury poisoning because of crude processing methods. Such is the danger for the galamsey in Ghana and similar workers known as *orpailleurs* in neighboring francophone countries. While there are no official government estimates of the labor force, observers believe twenty thousand to fifty thousand work as galamseys in Ghana, a figure that includes many women, who work as porters.

Mercury and many of its chemical compounds, especially organomercury compounds, can also be readily absorbed through direct contact with bare, or in some cases (such as dimethylmercury) insufficiently protected, skin. Mercury and its compounds are commonly used in chemical laboratories, hospitals, dental clinics, and facilities involved in the production of items such as fluorescent light bulbs, batteries, and explosives.[11]

Mechanism

Mercury is such a highly reactive toxic agent that it is difficult to identify its specific mechanism of damage, and much remains unknown about the mechanism.[12] It damages the central nervous system, endocrine system, kidneys, and other organs, and adversely affects the mouth, gums, and teeth. Exposure over long periods of time or heavy exposure to mercury vapor can result in brain damage and ultimately death. Mercury and its compounds are particularly toxic to fetuses and infants. Women who have been exposed to mercury in pregnancy have sometimes given birth to children with serious birth defects (see *Minamata disease*).

Mercury exposure in young children can have severe neurological consequences, preventing nerve sheaths from forming properly. Mercury inhibits the formation of myelin.

There is some evidence that mercury poisoning may predispose to Young's syndrome (men with bronchiectasis and low sperm count).[13]

Mercury poisoning's effects partially depend on whether it has been caused by exposure to elemental mercury, inorganic mercury compounds (as salts), or organomercury compounds.

Elemental mercury

Quicksilver (liquid metallic mercury) is poorly absorbed by ingestion and skin contact. It is hazardous due to its potential to release mercury vapour. Animal data indicate that less than 0.01% of ingested mercury is absorbed through the intact gastrointestinal tract; though it may not be true for individuals suffering from ileus. Cases of systemic toxicity from accidental swallowing are rare, and attempted suicide via intravenous injection does not appear to result in systemic toxicity.[12] Though not studied

quantitatively, the physical properties of liquid elemental mercury limit its absorption through intact skin and in light of its very low absorption rate from the gastrointestinal tract, skin absorption would not be high.[14] Some mercury vapour is absorbed dermally but uptake by this route is only approximately 1% of that by inhalation.[15]

In humans, approximately 80% of inhaled mercury vapor is absorbed via the respiratory tract where it enters the circulatory system and is distributed throughout the body.[16] Chronic exposure by inhalation, even at low concentrations in the range 0.7–42 $\mu g/m^3$, has been shown in case control studies to cause effects such as tremors, impaired cognitive skills, and sleep disturbance in workers.[17][18] Acute inhalation of high concentrations causes a wide variety of cognitive, personality, sensory, and motor disturbances. The most prominent symptoms include tremors (initially affecting the hands and sometimes spreading to other parts of the body), emotional lability (characterized by irritability, excessive shyness, confidence loss, and nervousness), insomnia, memory loss, neuromuscular changes (weakness, muscle atrophy, muscle twitching), headaches, polyneuropathy (paresthesia, stocking-glove sensory loss, hyperactive tendon reflexes, slowed sensory and motor nerve conduction velocities), and performance deficits in tests of cognitive function.[14]

Inorganic mercury compounds

Mercury occurs inorganically as salts such as mercury(II) chloride. Mercury salts primarily affect the gastro-intestinal tract and the kidneys, and can cause severe kidney damage; however, as they can not cross the blood-brain barrier easily, mercury salts inflict little neurological damage without continuous or heavy exposure.[19] As two oxidation states of mercury form salts (Hg^+ and Hg^{2+}), mercury salts occur in both mercury(I) (or mercurous) and mercury(II) (mercuric) forms. Mercury(II) salts are usually more toxic than their mercury(I) counterparts because their solubility in water is greater; thus, they are more readily absorbed from the gastrointestinal tract.[19]

Mercuric Cyanide, $Hg(CN)_2$ is a particularly toxic mercury compound. If ingested, both life-threatening mercury and cyanide poisoning can occur. $Hg(CN)_2$ can enter the body via inhalation, ingestion, or passage through the skin. Inhalation of mercuric cyanide irritates the throat and air passages. Heating or contact of $Hg(CN)_2$ with acid or acid mist releases toxic mercury and cyanide vapors that can cause bronchitis with cough and phlegm and/or lung tissue irritation. Contact with eyes can cause burns and brown stains in the eyes, and long time exposure can affect the peripheral vision. Contact with skin can cause skin allergy, irritation, and gray skin color.[20]

Chronic exposure to trace amounts of the compound can lead to mercury buildup in the body over time; it may take months or even years for the body to eliminate excess mercury. Overexposure to mercuric cyanide can lead to kidney damage and/or mercury poisoning, leading to 'shakes' (ex: shaky handwriting), irritability, sore gums, increased saliva, metallic taste, loss of appetite, memory loss, personality changes, and brain damage. Exposure to large doses at one time can lead to sudden death.[20]

Mercuric cyanide has not been tested on its ability to cause reproductive damage. Although inorganic mercury compounds (such as $Hg(CN)_2$) have not been shown to be human teratogens, they should be handled with care as they are known to damage developing embryos and decrease fertility in men and women.[20]

According to one study, two people exhibited symptoms of cyanide poisoning within hours after ingesting mercuric cyanide or mercury oxycyanide, $Hg(CN)_2 \cdot HgO$, in suicide attempts. The toxicity of $Hg(CN)_2$ is commonly assumed to arise almost exclusively from mercury poisoning; however, the

patient who ingested mercury oxycyanide died after 5 hours of cyanide poisoning before any mercury poisoning symptoms were observed. The patient who ingested $Hg(CN)_2$ initially showed symptoms of acute cyanide poisoning which were brought under control, and later showed signs of mercury poisoning before recovering. It is thought that the degree to which cyanide poisoning occurs is related to whether cyanide ions are released in the stomach, which depends on factors such as the amount ingested, stomach acidity, and volume of stomach contents.[21] Given that $Hg(CN)_2$ molecules remain undissociated in pure water and in basic solutions,[22] it makes sense that dissociation would increase with increasing acidity. High stomach acidity thus helps cyanide ions to become more bioavailable, increasing the likelihood of cyanide poisoning.

Mercury cyanide was used in two murders in New York in 1898. The perpetrator, Roland B. Molineux, sent poisoned medicines to his victims through the US mail. The first victim, Henry Barnett, died of mercury poisoning twelve days after taking the poison. The second victim, Catherine Adams, died of cyanide poisoning within 30 minutes of taking the poison. As in the suicide cases, the difference between the two cases may be attributed to differences in the acidities of the solutions containing the poison, or to differences in the acidities of the victims' stomachs.[23]

The drug NAP (n-acetyl penicillamine) has been used to treat mercury poisoning with limited success.[20]

Organic mercury compounds

Compounds of mercury tend to be much more toxic than the element itself, and organic compounds of mercury are often extremely toxic and have been implicated in causing brain and liver damage. The most dangerous mercury compound, dimethylmercury, is so toxic that even a few microliters spilled on the skin, or even a latex glove, can cause death.[24][25]

Methylmercury is the major source of organic mercury for all individuals.[1] It works its way up the food chain through bioaccumulation in the environment, reaching high concentrations among populations of some species. Larger species of fish, such as tuna or swordfish, are usually of greater concern than smaller species. The U.S. Food and Drug Administration (FDA) and the U.S. Environmental Protection Agency (EPA) advise women of child-bearing age, nursing mothers, and young children to completely avoid swordfish, shark, king mackerel and tilefish from the Gulf of Mexico, (Golden Tilefish from the Mid- and North-Atlantic present no risk), to limit consumption of albacore ("white") tuna to no more than 6 oz (170 g) per week, and of all other fish and shellfish to no more than 12 oz (340 g) per week.[26] A 2006 review, conducted by Dr. Dariush Mozaffarian and Dr. Eric B. Rimm, of the risks and benefits of fish consumption found that for adults the benefits of one to two servings of fish per week outweigh the risks, even (except for a few fish species) for women of childbearing age, and that avoidance of fish consumption could result in significant excess coronary heart disease deaths and suboptimal neural development in children.[27] (Dr. Rimm has reported in the past that he has received payment or honoraria for presentations about food and diets from both the Culinary Institute of America and the International Chefs Association, among others.)[27]

There is a long latent period between exposure to methylmercury and the appearance of symptoms in adult poisoning cases. The longest recorded latent period is five months after a single exposure, in the Dartmouth case (see History); other latent periods in the range of weeks to months have also been reported. No explanation for this long latent period is known. When the first symptom appears, typically paresthesia (a tingling or numbness in the skin), it is followed rapidly by more severe effects, sometimes ending in coma and death. The toxic damage appears to be determined by the peak value of mercury, not the length of the exposure.[12]

Ethylmercury is a breakdown product of the antibacteriological agent ethylmercurithiosalicylate, which has been used as a topical antiseptic and a vaccine preservative (further discussed under Thiomersal below). Its characteristics have not been studied as extensively as those of methylmercury. It is cleared from the blood much more rapidly, with a half-life of 7 to 10 days, and it is metabolized much more quickly than methylmercury. It probably does not have methylmercury's ability to cross the blood-brain barrier via a transporter, but instead relies on simple diffusion to enter the brain.[1]

Other exposure sources of organic mercury include phenylmercuric acetate and phenylmercuric nitrate. These were used in indoor latex paints for their anti-mildew properties, but were removed in 1990 because of cases of toxicity.[1]

Diagnosis

Diagnosis of elemental or inorganic mercury poisoning involves determining the history of exposure, physical findings, and an elevated body burden of mercury. Although whole blood mercury concentrations are typically less than 6 μg/L, diets rich in fish can result in blood mercury concentrations higher than 200 μg/L; it is not that useful to measure these levels for suspected cases of elemental or inorganic poisoning because of mercury's short half-life in the blood. If the exposure is chronic, urine levels can be obtained; 24-hour collections are more reliable than spot collections. It is difficult or impossible to interpret urine samples of patients undergoing chelation therapy, as the therapy itself increases mercury levels in the samples.[28]

Diagnosis of organic mercury poisoning differs in that whole-blood or hair analysis is more reliable than urinary mercury levels.[28]

Prevention

Mercury poisoning can be prevented (or minimized) by eliminating or reducing exposure to mercury and mercury compounds. To that end, many governments and private groups have made efforts to regulate the use of mercury heavily, or to issue advisories about its use. For example, the export from the European Union of mercury and some mercury compounds has been prohibited since 2010-03-15.[29] The variability among regulations and advisories is at times confusing for the lay person as well as scientists.

[30]

Country	Regulating agency	Regulated activity	Medium	Type of mercury compound	Type of limit	Limit
US	Occupational Safety and Health Administration	occupational exposure	air	elemental mercury	Ceiling (not to exceed)	0.1 mg/m³
US	Occupational Safety and Health Administration	occupational exposure	air	organic mercury	Ceiling (not to exceed)	0.05 mg/m³

US	Food and Drug Administration	drinking	water	inorganic mercury	Maximum allowable concentration	2 ppb (0.002 mg/L)
US	Food and Drug Administration	eating	sea food	methylmercury	Maximum allowable concentration	1 ppm
US	Environmental Protection Agency	drinking	water	inorganic mercury	Maximum contaminant level	2 ppb (0.002 mg/L)

The United States Environmental Protection Agency (EPA) issued recommendations in 2004 regarding exposure to mercury in fish and shellfish.[31] The EPA also developed the "Fish Kids" awareness campaign for children and young adults [32] on account of the greater impact of mercury exposure to that population.

Treatment

Identifying and removing the source of the mercury is crucial. Decontamination requires removal of clothes, washing skin with soap and water, and flushing the eyes with saline solution as needed. Inorganic ingestion such as mercuric chloride should be approached as the ingestion of any other serious caustic. Immediate chelation therapy is the standard of care for a patient showing symptoms of severe mercury poisoning or the laboratory evidence of a large total mercury load.[1]

Chelation therapy for acute inorganic mercury poisoning can be done with DMSA, 2,3-dimercapto-1-propanesulfonic acid (DMPS), D-penicillamine (DPCN), or dimercaprol (BAL).[1] Only DMSA is FDA-approved for use in children for treating mercury poisoning. However, several studies found no clear clinical benefit from DMSA treatment for poisoning due to mercury vapor.[33] No chelator for methylmercury or ethylmercury is approved by the FDA; DMSA is the most frequently used for severe methylmercury poisoning, as it is given orally, has fewer side effects, and has been found to be superior to BAL, DPCN, and DMPS.[1] Alpha-lipoic acid (ALA) has been shown to be protective against acute mercury poisoning in several mammalian species when it is given soon after exposure; correct dosage is required, as inappropriate dosages increase toxicity. Although it has been hypothesized that frequent low dosages of ALA may have potential as a mercury chelator, studies in rats have been contradictory.[34] Glutathione and N-acetylcysteine (NAC) are recommended by some physicians, but have been shown to increase mercury concentrations in the kidneys and the brain.[34] Experimental findings have demonstrated an interaction between selenium and methylmercury, but epidemiological studies have found little evidence that selenium helps to protect against the adverse effects of methylmercury.[35]

Even if the patient has no symptoms or documented history of mercury exposure, a minority of physicians (predominantly those in alternative medicine) use chelation to "rid" the body of mercury, which they believe to cause neurological and other disorders. A common practice is to challenge the patient's body with a chelation agent, collect urine samples, and then use laboratory reports to diagnose the patient with toxic levels of mercury; often no pre-chelation urine sample is collected for comparison. The patient is then advised to undergo further chelation.[33] No scientific data supports the claim that the mercury in vaccines causes autism[36] or its symptoms,[37] and there is no scientific support for chelation therapy as a treatment for autism.[38]

Chelation therapy can be hazardous. In August 2005, an incorrect form of EDTA used for chelation therapy resulted in hypocalcemia, causing cardiac arrest that killed a five-year-old autistic boy.[39]

Prognosis

Many of the toxic effects of mercury are partially or wholly reversible, either through specific therapy or through natural elimination of the metal after exposure has been discontinued.[40] However, heavy or prolonged exposure can do irreversible damage, particularly in fetuses, infants, and young children. Young's syndrome is believed to be a long term consequence of early childhood mercury poisoning.[41] Mercuric Chloride may cause cancer as it has caused increases in several types of tumors in rats and mice, while methyl mercury has caused kidney tumors in male rats. The EPA has classified mercuric chloride and methyl mercury as possible human carcinogens (ATSDR, EPA)

Detection in biological fluids

Mercury may be measured in blood or urine to confirm a diagnosis of poisoning in hospitalized victims or to assist in the forensic investigation in a case of fatal overdosage. Some analytical techniques are capable of distinguishing organic from inorganic forms of the metal. The concentrations in both fluids tend to reach high levels early after exposure to inorganic forms, while lower but very persistent levels are observed following exposure to elemental or organic mercury. Chelation therapy can cause a transient elevation of urine mercury levels.[42]

History

- The first emperor of unified China, Qin Shi Huang, reportedly died of ingesting mercury pills that were intended to give him eternal life.[43]
- The phrase *mad as a hatter* is likely a reference to mercury poisoning, as mercury-based compounds were once used in the manufacture of felt hats in the 18th and 19th century. (The Mad Hatter character of *Alice in Wonderland* was almost certainly inspired by an eccentric furniture dealer, not by a victim of mad hatter disease.)[44]
- In 1810, two British ships, HMS *Triumph* and HMS *Phipps*, salvaged a large load of elemental mercury from a wrecked Spanish vessel near Cadiz, Spain. The bladders containing the mercury soon ruptured. The element spread about the ships in liquid and vapour forms. The sailors presented with neurologic compromises: tremor, paralysis, and excessive salivation as well as tooth loss, skin problems, and pulmonary complaints. In 1823 William Burnet, MD published a report on the effects of Mercurial vapour.[45] The *Triumph*'s surgeon, Henry Plowman, had concluded that the ailments had arisen from inhaling the mercurialized atmosphere. His treatment was to order the lower deck gun ports to be opened, when it was safe to do so; sleeping on the orlop was forbidden; and no men slept in the lower deck if they were at all symptomatic. Windsails were set to channel fresh air into the lower decks day and night.[46]
- For years, including the early part of his presidency, Abraham Lincoln took a common medicine of his time called "blue mass" which contained significant amounts of mercury.
- On September 5, 1920, silent movie actress Olive Thomas ingested mercury capsules dissolved in an alcoholic solution at the Hotel Ritz in Paris. There is still controversy over whether it was suicide, or whether she consumed the external preparation by mistake. Her husband, Jack Pickford (the brother of Mary Pickford), had syphilis, and the mercury was used as a treatment of the venereal disease at the time. She died a few days later at the American Hospital in Neuilly.

- An early scientific study of mercury poisoning was in 1923–6 by the German inorganic chemist, Alfred Stock, who himself became poisoned, together with his colleagues, by breathing mercury vapour that was being released by his laboratory equipment—diffusion pumps, float valves, and manometers—all of which contained mercury, and also from mercury that had been accidentally spilt and remained in cracks in the linoleum floor covering. He published a number of papers on mercury poisoning, founded a committee in Berlin to study cases of possible mercury poisoning, and introduced the term *micromercurialism*.[47]
- The term *Hunter-Russell syndrome* derives from a study of mercury poisoning among workers in a seed packing factory in Norwich, England in the late 1930s who breathed methylmercury that was being used as a seed disinfectant and preservative.[48]
- Outbreaks of methylmercury poisoning occurred in several places in Japan during the 1950s due to industrial discharges of mercury into rivers and coastal waters. The best-known instances were in Minamata and Niigata. In Minamata alone, more than 600 people died due to what became known as Minamata disease. More than 21,000 people filed claims with the Japanese government, of which almost 3000 became certified as having the disease. In 22 documented cases, pregnant women who consumed contaminated fish showed mild or no symptoms but gave birth to infants with severe developmental disabilities.[2]
- Widespread mercury poisoning occurred in rural Iraq in 1971-1972, when grain treated with a methylmercury-based fungicide that was intended for planting only was used by the rural population to make bread, causing at least 6530 cases of mercury poisoning and at least 459 deaths (see Basra poison grain disaster).[49]
- On August 14, 1996, Karen Wetterhahn, a chemistry professor working at Dartmouth College, spilled a small amount of dimethylmercury on her latex glove. She began experiencing the symptoms of mercury poisoning five months later and, despite aggressive chelation therapy, died a few months later from brain malfunction due to mercury intoxication.[24][25]
- In April 2000, Alan Chmurny attempted to kill a former employee, Marta Bradley, by pouring mercury into the ventilation system of her car.[50]
- On March 19, 2008, Tony Winnett, 55, inhaled mercury vapors while trying to extract gold from computer parts, and died ten days later. His Oklahoma residence became so contaminated that it had to be gutted.[51][52]
- In December 2008, actor Jeremy Piven was diagnosed with hydrargyria resulting from eating sushi twice a day for twenty years.[53]

Infantile Acrodynia

Infantile acrodynia (also known as "calomel disease", "erythredemic polyneuropathy", and "pink disease") is a type of mercury poisoning in children characterized by pain and pink discoloration of the hands and feet.[54] The word is derived from the Greek, where άκρο means end (as in: upper extremity) and οδυνη means pain. Also known as pink disease, erythredema, Selter's disease, or Swift-Feer disease, acrodynia was relatively commonplace amongst children in the first half of the 20th century.[55] Initially, the cause of the acrodynia epidemic among infants and young children was unknown;[56] however, mercury poisoning, primarily from calomel in teething powders, began to be widely accepted as its cause in the 1950s and 60s.[55] The prevalence of acrodynia decreased greatly after calomel was excluded from most teething powders in 1954.[55]

Acrodynia is difficult to diagnose, "it is most often postulated that the etiology of this syndrome is an idiosyncratic hypersensitivity reaction to mercury because of the lack of correlation with mercury levels, many of the symptoms resemble recognized mercury poisoning."[57]

Medical procedures

Because elemental mercury often passes through the GI tract without being absorbed, it was used medically for various purposes until the dangers of mercury poisoning became known. For example, elemental mercury was used to mechanically clear intestinal obstructions (due to its great weight and fluidity), and it was a key ingredient in various medicines throughout history, such as blue mass. The toxic effects often were either not noticed at all, or so subtle or generic that they were attributed to other causes and were not recognized as poisoning caused by mercury. While the usage of mercury in medicine has declined, mercury-containing compounds are still used medically in vaccines and dental amalgam, both of which have been the subject of controversy regarding their potential for mercury poisoning.

Thiomersal

For more details on this topic, see Thiomersal controversy.

The mercury-based preservative thiomersal (commonly called *thimerosal* in the U.S.) has been added to vaccines since the 1930s to prevent their deterioration.[12] Its use in vaccines has been hypothesized as a cause of autistic behaviors.[58] This hypothesis is controversial, as much evidence suggests that the cause of autism is about 90% genetic.[59] The hypothesis has not been confirmed by reliable studies.[60] However, organizations such as the American Academy of Pediatrics have recommended that the use of thiomersal be reduced as a precautionary measure. With the exception of some flu vaccines, it is no longer used as a preservative in routinely recommended childhood vaccines in the United States; it is still in limited use as a preservative in multi-dose flu and tetanus vaccines and a few other non-childhood vaccines.[61]

Dental amalgam

For more details on this topic, see Dental amalgam controversy.

Dental amalgam, an alloy of about 50% elemental mercury, was first introduced in France in the early 19th century.[62] Although this amalgam is a source of low-level exposure to mercury, no scientific evidence links it as a cause of clinically significant toxic effects, except for the rare local hypersensitivity reaction. In the United States, the National Institutes of Health has stated that amalgam fillings pose no personal health risk, and that replacement by non-amalgam fillings is not indicated.[1] In Scandinavia amalgam fillings are banned due to concerns about environmental pollution with mercury.[63]

Cosmetics

Some skin whitening products contain the toxic chemical mercury(II) chloride as the active ingredient. When applied, the chemical readily absorbs through the skin into the bloodstream.[64] The use of mercury in cosmetics is illegal in the United States. However, cosmetics containing mercury are often illegally imported. Following a certified case of mercury poisoning resulting from the use of an imported skin whitening product, the United States Food and Drug Administration warned against the use of such products.[65][66] Symptoms of mercury poisoning have resulted from the use of various mercury-containing cosmetic products.[12][67][68] The use of skin whitening products is especially popular amongst

Asian women.[69] In Hong Kong in 2002, two products were discovered to contain between 9,000 to 60,000 times the recommended dose.[70]

Fluorescent lamps

Fluorescent lamps contain mercury which is released when bulbs are broken. Mercury in bulbs is typically present as either elemental mercury liquid, vapor or both since the liquid evaporates at ambient temperature.[71] When broken indoors, bulbs may emit sufficient mercury vapor to present health concerns, and the U.S. Environmental Protection Agency recommends evacuating and airing out a room for at least 15 minutes after breaking a fluorescent light bulb.[72] Breakage of multiple bulbs presents a greater concern. A 1987 report described a 23-month-old toddler who suffered anorexia, weight loss, irritability, profuse sweating, and peeling and redness of fingers and toes. This case of acrodynia was traced to exposure of mercury from a carton of 8-foot fluorescent light bulbs that had broken in a potting shed adjacent to the main nursery. The glass was cleaned up and discarded, but the child often used the area for play.[73]

See also

- Got Mercury?
- Lead poisoning
- Mercury Policy Project
- Mercury vacuum
- Mercury-containing and Rechargeable Battery Management Act
- Erethism
- Diagnosis Mercury: Money, Politics and Poison

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External links

- Hazardous Substances: Mercury (http://www.dmoz.org/Health/Occupational_Health_and_Safety/Hazardous_Substances/Mercury/) at the Open Directory Project
- Toxic Substances: Mercury (http://www.dmoz.org/Science/Environment/Environmental_Health/Toxic_Substances/Mercury/) at the Open Directory Project

- http://www.epa.gov/ttn/oarpg/t3/reports/volume5.pdf|Mercury study report to congres
- http://www.atsdr.cdc.gov/tfacts46.html|Agency for Toxic substances and Disease registry

Categories: Drug eruptions | Element toxicology | Mercury (element) | Toxic effects of substances chiefly nonmedicinal as to source