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Toxicity of mercury inhalation

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ABSTRACT

Mercury is a silver-white volatile liquid metal at room temperature. In its free metallic state, mercury is most poisonous as a vapor. All mercury compounds are poisonous to various extents, with organic mercury compounds much more poisonous than inorganic compounds. The accumulation of mercury in the body causes brain damage and affects the nervous system. Mercury has a high affinity for sulfur and thus interferes with physiological sulfur containing enzymes. Mercury poisoning has been associated with acrodynia, Minamata disease, and Hunter-Russell syndrome. Exposure to mercury fumes has notably been associated with Mad hatter disease, a vocational hazard of hat making, which resulted in vision and speech impairment, hallucinations, tremors, and lack of coordination. Mercury vapors are readily absorbed and spread quickly throughout the body, including the central nervous system and placenta. Inhalation of high concentrations of mercury vapor will lead to symptoms up to a few hours later that include: chills, fever, cough, shortness of breath, nausea, vomiting, and diarrhea. For children, for chronic or acute exposure of mercury can enable adverse effects during any period of development. In an ideal scenario there is absolutely no mercury in a child's body. Adverse health affects can be a result of mercury exposure from workplace or residential habitat. Studies of occupational mercury vapor exposure produces long term effects on psychomotor function, increased depression, increased anxiety, and affects on the information processing of nervous system. Mercury inhalation will induce pneumonitis. When broken indoors, light bulbs containing mercury can emit sufficient vapor to present health concerns with multiple bulbs causing even a greater concern. Accordingly, the breakage of larger and numerous bulbs will increase the danger to health. The central nervous system is considered to be the most sensitive of targets from mercury vapor incursion.

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KEYWORDS

Mercury poisoning;
Mad hatter disease;
Mercury toxicity;
Mercury.

GENERAL CONSIDERATIONS

Mercury is one of five metallic chemical elements

that are liquid near or at room temperature and pressure (the others are cesium, francium, gallium, and rubidium)^[1]. Mercury has a freezing point of -38.83 °C

and a boiling point of 356.7 °C, which is one of the broadest ranges of liquid state for any metal^[1]. Of the anthropogenic associated level of mercury in the atmosphere: 65 % come from stationary combustion, 11 % from gold extract in mines, 6.8 % from non-ferrous metal production, 6.4 % from cement production, 3.0 % from caustic soda production, 3.0 % from waste disposal, and remaining from various sources (see Figure 1)^[2].

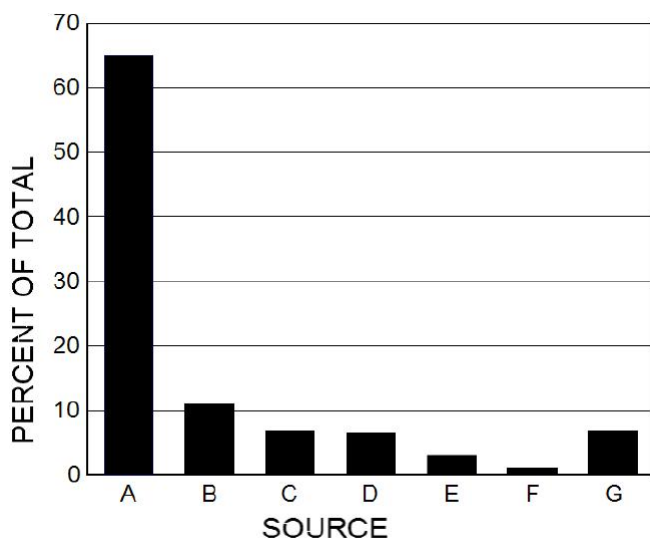


Figure 1 : Sources of atmospheric mercury and their relative percent of total amount of mercury emissions: A) Stationary combustion (power plants fueled by coal, oil, and natural gas); B) Gold production emission; C) Non-ferrous metal production; D) Cement production; E) Waste disposal; F) Mercury production; G) Other sources.

Mercury is most poisonous as a vapor and the metal interferes with sulfur containing enzymes due to its high affinity for sulfur^[1]. All mercury compounds are poisonous to specific degrees with organic mercury compounds much more poisonous than inorganic mercury compounds^[1]. At room temperature, mercury is a silver-white liquid metal that is volatile^[3]. Vapors of elemental mercury are odorless, heavier than air (thus resting in low lying locations), readily absorbed after inhalation (75 % to 80 %), and rapidly distribute throughout the body^[4]. Potential sources of exposure include from household items of broken light bulbs, broken thermostats, electrical switches, and broken thermometers^[4]. Inhalation of high concentrations of elemental mercury will result in fever, cough, chills, shortness of breath, nausea, vomiting, and diarrhea, followed by damage to lungs, kidneys, and the central

nervous system^[4].

Acute high doses of mercury inhalation affects the kidneys and has been associated with nephrotic syndrome, proteinuria, temporary tubular dysfunction, and oliguric renal failure^[4]. In addition, effects on the cardiovascular system include tachycardia and hypertension^[4]. In workers exposed to mercury vapors up to 5.5 years investigators have identified the presence of anti-laminin antibodies which is an indicator of immune dysfunction. In those same workers there occurred no unusual renal parameters^[5]. In chloroalkali workers there was no detectable damage of glomerular or tubular damage after 8.7 to 18.7 years of work^[6]. However workers of the same background had increased number of EEG abnormalities, slower and attenuated brain activity, with a statistically significant increase in memory disturbances, and sleep disorders^[7]. For workers involved with fluorescent bulb manufacture and exposed to low level mercury vapors, it was found that there were increased tremors^[8]. Other investigators found that in laboratory workers having 35 microgram/liter levels of mercury in urine had increased proteinuria^[9].

CASE STUDIES OF INHALED MERCURY PATHOLOGY

Mercury is a highly toxic element that has no known safe level of human exposure. Because mercury does not present any physiological benefits there should be no mercury in the bodies of children or adults^[10]. Mercury is nephrotoxic, neurotoxic, immunotoxic and will put at risk in utero and early life child development^[10] in part because mercury is ubiquitous and pre- & post-natal exposure can occur in many ways^[10]. Mercury is bio-accumulating, which results in a serious health hazard for children^[10].

Elemental mercury vapor poisoning is known to cause hemorrhagic colitis^[11]. Mercury is an ubiquitous environmental toxin that is associated with a broad range of human adverse health effects^[12]. Wherein elemental mercury exposure is acquired generally by inhalation and ingestion. Mercury readily volatilizes at standard temperature and pressure (The current version of IUPAC's standard is a temperature of 0 °C (273.15 K, 32 °F) and an absolute pressure of 100 kPa (14.504

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psi, 0.986 atm)^[12]. Its presence in an open container can induce biological significant aerated concentrations in poorly ventilated locations. Adverse health effects can occur in individuals that are exposed in residential as well as industry^[13].

Increasing evidence is mounting that the health effects from toxic metals differ in prevalence and/or manifestation in females and males^[14]. Mercury, in the form of elemental mercury vapor (and methylmercury), are easily transferred from the pregnant women to the fetus^[14]. Studies have determined that young males are more susceptible to neurotoxic effects of lead and methylmercury following an exposure event occurring early in life^[14], while actual experimental data suggests that females are more susceptible to immunotoxic effects of the heavy metal lead. Additional studies on the gender-related differences in health effects that are caused by heavy metals requires considerably more attention^[14].

Anthropogenic activities have frequently resulted in mercury release into the biosphere with diminished consideration of the adverse consequences to human health^[15]. Humans can take in toxic doses of mercury through inhalation of elevated concentrations of gaseous elemental mercury^[15].

Industrial workers have been diagnosed with chronic occupational mercurialism located at a fluorescent lamp factory^[16]. It was found that after exposure to elemental mercury for an average of 10.2 ± 3.8 years and even after being away from this work for 6 ± 4.7 years, there was a mean urinary mercury concentrations (following 1 year cessation of work) were at 1.8 ± 0.9 microgram/g creatinine^[16]. In this particular study the neuropsychological assessment included attention, inhibitory control, verbal/visual memory, verbal fluency, manual dexterity, visual-spatial function, executive function, and semantic knowledge evaluation. Beck Depression Inventory and the State and Trait Inventory were applied to assess levels of depression and anxiety symptoms, respectively^[16]. Raw scores outcome for this particular group exposed to mercury indicated slower information processing speed, inferior performance in psychomotor speed, verbal spontaneous recall memory, and manual dexterity of the dominant hand and non-dominant hand^[16]. These individuals also demonstrated increased depression and anxiety symptoms, whereas

a statistically significant correlation (Pearson r) was observed between mean urinary mercury and anxiety trait ($r = 0.75$, $P = 0.03$)^[16]. The neuropsychological performances of this group also suggests that exposure to elemental mercury has long-term effects on psychomotor function and information processing, in addition to increased depression and anxiety^[16].

Other investigators have determined that performance on the grooved pegboard and the Benton visual retention test was poorer among mercury vapor exposed chloralkali workers when compared with suitable referents^[17]. For this study the subjects who had experienced the highest intensity of exposure [a cumulative urinary mercury index ≥ 550 nmol/(1 x year)] did have a poorer performance on a trailmaking test (inclusive of part A and B), on a digit symbol test, and on a word pairs test (a test of retention errors)^[17]. This same investigation did identify a slight persistent effect of mercury vapor exposure on the central nervous system that chiefly involved the motor functions and attention, nonetheless possibly with relation to the visual system^[17]. Previous exposure did not seem to have affected the workers' general intellectual level or their ability to reason logically^[17].

Various sources of elemental mercury (Hg^0) will include old natural gas regulators, manometers, sphygmomanometers, thermometers, as well as thermostats^[18]. Spillage of Hg^0 will include improper storage of vessels, as well as container breakage, children playing with Hg^0 , and abusive use of Hg ^[18]. Inhalation is the primary exposure route for elemental mercury^[18]. Chronic exposure to elemental mercury vapors could damage the kidneys and neurologic system whereas short-term exposure to high levels of vapors could cause lung damage, nausea, vomiting, diarrhea, increases in blood pressure or heart rate, skin rashes, and eye irritation^[18]. Minimization of elemental mercury dispersal is crucial^[18] but spreading by contaminated shoes/apparel or vacuuming can spread elemental mercury thus increasing airborne concentrations and costs of cleansing^[18]. Hard surfaces can generally be decontaminated however contaminated porous items must be appropriately cast aside. Even marginally contaminated items outdoors for a month or more during a warm weather spell could dissipate the Hg^0 ^[18]. Reduction of use is the the optimal way to prevent spills

of elemental mercury^[18].

The volatility and biotransformation of inhaled mercury causes Hg⁰ to be a unique toxicant. The heating of liquid elemental mercury can lead to multiple pathological symptoms such as dyspnea, chest pain, cough, malaise, and metallic taste^[19]. This mechanism of exposure can result in plasma levels of Hg⁰ over toxic levels and the need of hospitalization that includes oxygen therapy^[19]. Mercury is excreted in the urine^[19]. Vapors of elemental mercury are well absorbed via the lungs (80 %) but has significantly poorer absorption through the gastrointestinal tract^[20]. A significant subcutaneous or intravenous intromission of elemental mercury results directly in chest pain, fever, kidney damage, respiratory failure, liver damage, neurologic symptoms, and potentially death^[20]. Mercury is toxic in all forms. Mercury is primarily excreted from the human body through the kidneys albeit the rate of excretion is generally slow with traces of mercury observed in the urine even as long as two years after the index event^[20]. In the case of injected mercury small but discrete metallic distributions can form at the base of the lungs and throughout the parenchyma bilaterally^[20].

Inhalation of mercury vapour is extremely dangerous. A case study of a patient following mercury inhalation produced observations of a nonfatal pneumonitis^[21]. In this case the pulmonary infiltration was resolved rapidly with antibiotics and supportive care. In this case study gingivostomatitis was found, along with central nervous system manifestations such as tremors and erethism (shyness, withdrawal, depression, insomnia and irritability)^[21]. A chest x-rays displayed diffuse, patchy changes of pulmonary edema, (which can diminish but can progress to interstitial fibrosis, pulmonary granulomas, and bronchiectasis)^[21]. Pulmonary function tests may show a mixture of restrictive and obstructive defects. These investigators treatment management (this an acute mercury vapour inhalation event) centers around the maintenance of respiratory function by oxygen, bronchodilators, mechanical ventilation, and evaluation of systemic absorption^[21]. Levels of mercury in whole blood can be determined accurately in recent exposures, but do not measure total body burden in chronic exposures or exposures that have occurred more than a few days previously. Timed, 24 hour urinary collections can pro-

vide a more efficacious measure of total body burden and are quite useful for determination of chelation therapy^[21]. Significant clinical symptoms are usually associated with 24 h urine content of greater than 250 nanomole/Liter^[21].

Acute mercury inhalation poisoning can be fatal because of progressive pulmonary failure^[22]. Prompt treatment with corticosteroids and penicillamine for acute chemical pneumonitis can be beneficial following a three phase progression that begins with a manifested flu-like illness, then an intermediate phase involving a period in which severe multi-organ symptoms is observed then a late phase consisting of a period when the central nervous symptoms persist^[22]. With cases of very high acute exposure to mercury vapor, then severe respiratory symptoms dominate the clinical picture^[22]. The cause of death in lethal cases is progressive respiratory failure which at autopsy will show pathologic findings in the lungs revealing various stages of acute lung injury, analogous to those found in the acute respiratory distress syndrome (ARDS)^[22]. Penicillamine has been generally accepted as an efficacious chelating agent for mercury poisoning. Dimercaptopropanol (of BAL) is also effective, but penicillamine has the added advantage of oral administration and is possibly more potent^[22]. D-penicillamine given orally may be useful in the less severe symptomatic inorganic and elemental mercury inhalation exposures. D- penicillamine reverses sulfhydryl binding in the blood and chelates both mercury and lead. N- acetyl- d,L- penicillamine has been administered successfully to patients with inorganic mercury- induced neuropathies (tremor, ataxia) and chronic elemental mercury toxicity^[22]. Although chelation therapy has been shown to decrease serum mercury levels, studies show that this has no effect on progression of acute lung injury. Previous studies have shown that lung tissue damage is complete and that the treatment of serum levels with chelating agents has no effect on the reversal of lung damage^[22]. Despite the reduction of serum mercury levels with dimercaprol, there was also no reversal in the progression of lung injury and respiratory dysfunction^[22].

SPECIFIC ILLUSTRATIVE TOXICITY

Although it is most toxic if inhaled as vapors, mer-

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cury may be absorbed through the skin and ingested^[23]. Any spillage should be immediately attended to as it is very persistent in the atmosphere. Mercury that is absorbed through the lungs is transported to the brain and other parts of the nervous system, as well as other tissues. Elemental mercury is also excreted in the feces. Three factors that influence the development of clinical manifestations as the result of inhalation include: 1) Length of exposure; 2) Individual susceptibility; and 3) Concentration of the exposure^[24]. Symptoms associated with high concentrations of exposure will include chills, malaise, nausea, coughing, chest pain, shortness of breath, and fever^[25]. The risks of mercury exposure are especially increased for children because mercury vapor is dense and will settle in lower areas or of poor ventilation where the child may be active^[25]. Due to lipid solubility mercury crosses the placenta as well as excreted in breast milk therefore posing a health hazard for unborn children and infants still in breast feeding phase^[25].

Because mercury inhibits the formation of myelin, exposure to this element for young children may induce severe neurological outcomes by preventing the nerve sheaths from forming in good order^[26]. Mercury is a toxic agent which is highly reactive and damages the endocrine system, kidneys, central nervous system, other organs, and adversely affects the teeth, mouth, and gums^[26]. At zero oxidation state (Hg^0) of mercury the element exists as a liquid metal and vapor, but at oxidation state of 1+ (Hg^+) it is an inorganic salt and at 2+ it can exist as an inorganic salts or organomercury compounds. In addition to toxic affect on the brain, kidney, and lung, mercury poison can lead to diseases such as Hunter-Russell syndrome, pink disease (acrodynia), and Minamata disease^[27,28]. When broken, fluorescent bulbs release mercury. Mercury can exist as vapor or liquid state (or both) within bulbs which is released after breakage, allowing the liquid portion to evaporate at the ambient temperature to pose a health threat^[29]. The breakage of numerous bulbs is an additive threat that raises great concern as a health issue. For example, a case study showed that a 23-month child playing in a nursery adjacent to a shed housing a container of broken 8-foot fluorescent light bulbs still caused anorexia, weight loss, profuse sweating, irritability, and acrodynia (pink

discoloration of the hands and feet)^[30].

Mercury contamination is an environmental problem worldwide^[31]. Exposure to a dose of elemental mercury vapor (Hg^0) at 1 to 2 milligram/meter³ for several hours causes acute chemical related bronchiolitis and pneumonitis. After this exposure and two hours later the lung injury appears as a hyaline membrane formation, followed by expensive pulmonary fibrosis^[31]. However studies have shown that corticosteroids and chelating agents appear to effectively decrease the inflammation, delay pulmonary fibrosis, and prevent outcome of long-term mercury poisoning^[31]. Chelating therapy will efficaciously reduce the levels of mercury in the serum^[31]. The chelating agents N-acetyl penicillamine and dimercaprol will displace mercury ions from sulfhydryl groups (-C-SH or R-SH) group where R represents an alkene, alkane, or carbon-containing moiety and the -SH functional group is referred to as either a thiol group or a sulfhydryl group^[31].

ADDITIONAL CASE STUDIES OF EXPOSURE

Recorded incidents of short-term exposure to high levels of mercury vapor induce a range of health effects. These effects range from mild proteinuria to pronounced proteinuria, hematuria or oliguria, to actual acute renal failure having necrosis of the proximal convoluted tubules^[32].

Previous clinical observations have identified the threat of inhaling mercury vapors from intentionally heating liquid mercury^[33]. In this case, involving a young child, the detrimental health outcome included hepatocellular consequences^[33].

Fatal outcome can occur from short-term but high level exposure to vapors of elemental mercury (Hg^0)^[34,35]. In this case study, involving a grown adult male, high level exposure to vapor induced hepatomegaly (Hepatomegaly is a condition of having an enlarged liver) and central lobular vacuolation (a condition of having become filled with vacuoles)^[34,35].

CONCLUSIONS

Although it is most toxic if inhaled as vapors, mercury may be absorbed through the skin and ingested. Mercury is a highly toxic element that has no known

safe level of human exposure. Because mercury does not present any physiological benefits there should be no mercury in the bodies of children or adults. Mercury is most poisonous as a vapor and the metal interferes with sulfur containing enzymes due to its high affinity for sulfur. Mercury is primarily excreted from the human body through the kidneys albeit the rate of excretion is generally slow with traces of mercury observed in the urine even as long as two years after the index event.

Mercury is an ubiquitous environmental toxin that is associated with a broad range of human adverse health effects and elemental mercury exposure is acquired generally by inhalation and ingestion. Mercury, in the form of elemental mercury vapor (and methylmercury), are easily transferred from the pregnant women to the fetus. Due to lipid solubility mercury crosses the placenta as well as excreted in breast milk therefore posing a health hazard for unborn children and infants still breast feeding.

Because mercury inhibits the formation of myelin, exposure to this element for young children may induce severe neurological outcomes by preventing the nerve sheaths from forming. Acute mercury inhalation poisoning can be fatal because of progressive pulmonary failure. Prompt treatment with corticosteroids and penicillamine for acute chemical pneumonitis can be beneficial. Mercury is a toxic agent that is highly reactive and damages the endocrine system, kidneys, central nervous system, other organs, and adversely affects the teeth, mouth, and gums.

Inhalation of high concentrations of elemental mercury will result in fever, cough, chills, shortness of breath, nausea, vomiting, and diarrhea, followed by damage to lungs, kidneys, and the central nervous system. Avoidance of mercury exposure is crucial for pregnant women, infants, young children, as well as adults. The potential health threat due to breakage of mercury containing light bulbs cannot be minimized and must be considered in residential and industrial environments. The potential danger to health that mercury containing light bulbs render must be a vital consideration if expansion of these types of lighting apparatus is pursued.

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