

Occupational Mercury Exposure among Oil Technicians in Libya

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Abstract :

The aim of this study is to determine blood mercury levels among Libyan technicians who use mercury for analysis of crude oil.

Occupational exposures to mercury can occur where mercury is produced, used in processes, or incorporated in products. The susceptible subpopulations for mercury toxicity include those who are more sensitive to the effects of mercury and those who are exposed to higher levels of mercury.

The mean blood mercury level in the study group is 5.86 μ g/L, which

is 3 times higher than the mean blood mercury level ($1.7\mu\text{g/L}$) in the control group.

Regulations and precautions must be taken by the workers to avoid mercury poisoning.

Key words: mercury, occupational, exposure, toxicity, blood levels.

Introduction :

Mercury toxicity has been recognized since the time of Hippocrates¹. At the end of 18th century, mercury toxicities were caused by the mercury-containing antisyphilitic agents. At the time being, the risk of mercury toxicity is high as it has many uses. Medically, it is still used as dental amalgams and as antiseptic agents. Occupational mercury exposure is another important cause for its toxicity. Environmental pollution by mercury is a major global concern because of increased usage of fuels and agricultural products².

There are three primary categories of mercury and its compounds: elemental mercury; inorganic mercury and organic mercury compounds. Mercury toxicity may occur with all forms. Occupational exposures occur mainly by inhalation of elemental mercury². The toxicokinetics have an important role in determining the toxic effects of elemental mercury; nearly 80% of inhaled elemental mercury is absorbed through the lungs by rapid diffusion. In contrast, only 0.01% of elemental mercury is absorbed through the gastrointestinal tract. Dermal absorption of elemental mercury is limited³. Elemental mercury is highly diffusible and lipid soluble.

Elemental mercury can cross the blood-brain barrier and blood-placenta barrier as well as the lipid bilayers of cellular and intracellular organellar membranes. Though elemental mercury vapor is rapidly oxidized to ionic mercury, it remains as vapor in the blood for a short time, which is long enough for a significant amount of mercury vapor to penetrate the blood-brain barrier before it is oxidized to the mercuric form (Hg^{++}) and accumulate in the brain⁴. The primary organs of mercury deposition following inhalation exposure to elemental mercury vapor are the brain and kidney⁵. Elemental mercury is bound strongly to selenium or SH-groups after oxidation in the brain⁴. With time after exposure, the greater proportion of the body burden of mercury is found in the kidney⁵. Urine and feces are the main pathways of excretion, although a small amount of inhaled mercury can be eliminated in the breath, sweat, and saliva⁵. The biological half-life of mercury is estimated to be approximately 30 to 60 days in the body⁵. The half-life of mercury in the brain is not entirely clear, but is estimated to be as long as approximately 20 years⁵.

Mercury may cause different organ toxicities. Human exposure to toxic levels of mercury vapor in adults causes the classic triad of erethism (bizarre behavior, eg, excessive shyness or aggression), tremor, and gingivitis⁶. The cardinal neurologic sign of toxic vapor exposure is tremor that may be accompanied by a variety of neuropsychological effects ranging from emotional lability at high exposure levels to subtle performance deficits at lower levels^{6,7}.

The neurotoxic effects of organic and inorganic mercury are different. Organic mercury toxicity may cause prominent neuronal loss and gliosis in the calcarine and parietal cortices and cerebellar folia, as seen in cases of classic Minamata disease⁸, while inorganic mercury may lead to cerebral infarctions⁹. Mercury damages the nervous system through several ways; it binds to sulfhydryl groups and incapacitates the enzymes involved in the cellular stress response, protein repair, and oxidative damage prevention¹⁰. It may disrupt the muscarinic cholinergic systems in the brainstem and occipital cortices¹¹. It may inactivate Na-K-ATPase that leads to membrane depolarization, calcium entry, and eventual cell death¹².

Renal effects are variable from mild transient proteinuria to severe proteinuria, hematuria, and/or oliguria to acute renal failure, with degeneration or necrosis of the proximal convoluted tubules¹³.

Dermal changes may appear after inhalation, oral, or dermal exposure to elemental mercury vapors or inorganic mercury. Erythematous and pruritic rashes are the result of irritation or sensitization reactions. Heavy perspiration and reddened and/or peeling skin on the palms of the hands and soles of the feet typically associate the acrodynia¹⁴.

Respiratory symptoms are a prominent effect of short-term, high-level exposure to elemental mercury vapors. The most commonly reported symptoms include cough, dyspnea, and chest tightness or burning pains in the chest¹⁵. In the more severe cases, respiratory distress, pulmonary edema, lobar pneumonia, fibrosis, and desquamation of the bronchiolar

epithelium have been observed¹⁵.

Cardiovascular effects after short-term inhalation of elemental mercury vapor include increased blood pressure and heart rate¹⁵. Risks of coronary heart diseases increase with mercury toxicity¹⁶.

Gastrointestinal effects are stomatitis, abdominal pain, nausea, and/or diarrhea that may occur following short-term exposure to elemental mercury vapors, occasionally accompanied by excessive salivation or difficulty swallowing¹⁵.

Toxic effects of mercury on reproductive system are variable. Menstrual cycle disorders are more frequent among women working in a mercury vapor¹⁷ as well as in animal studies¹⁸.

The technicians estimate the amount of crude oil in rock samples by soaking it in elemental mercury. Mercury evaporates in room temperature and the technicians are exposed to mercury vapor.

No such studies on mercury poisoning among oil workers were done in Libya before.

Aim of the Study :

To determine blood mercury levels (BMLs) among a group of technicians exposed to mercury during their work.

Methods and Material :

The study group consists of 76 technicians (72 males and 4 females). The range of workers' age is from 24 years to 52 years with mean age (38

± 12.4 Yrs). The duration of exposure is from 4 Years to 18 Years, with an average (9.4 ± 4.7 Yrs). The study was conducted in 2010 in Tripoli, Libya.

The data collected includes complete medical history, duration of exposure, diet, smoking, alcohol consumption, working time,, vacations, safety precautions, physical symptoms(table 1). and blood analysis

Detailed physical and neurological examinations were performed on each technician by specialized doctors. Special attention was focused on possibility of presence of gingivitis, dysarthria, tremor, finger to nose test, heel to knee and shin test, gait disturbance, muscle strength, and tendon reflexes.

Blood samples were collected for the measurement of mercury levels, sugar, and renal and liver function tests. The blood samples were analysed for Mercury levels in Germany by atomic absorption spectrometer in Biosencia laboratory.

Table 1: Potential Mercury-related Symptoms

Body weight loss	Numbness
Gum pain	Memory impairment
Hypersalivation	Bad temper
Hyperhidrosis	Back pain
Nightmare/insomnia	Blurred vision
Speech problems	Tremor
Dizziness	Writing difficulty
Fatigue	Slow mental response
Inattention	Unsteady gait

The control group consists of 25 volunteers (15 males and 10 females) with ages 23 years to 48 years (average 32 ± 9.76 Years). The group had been collected randomly with the consent of the involved individuals. The individuals in the control group are nonsmokers and with normal Libyan dietary habits. They are eating fish as canned tuna and sometimes as fresh-cooked fish (1-2 times per month). The selection is done in this way to minimize the possible effects of diet and smoking as sources that may increase BMLs.

Results :

In the study group, the BMLs ranged from 0.8-19 $\mu\text{g/L}$ and the mean blood mercury level is 5.86 $\mu\text{g/L}$. There are no significant findings in the clinical examinations.

In the control group, the BMLs ranged from 0.1 – 3.5 $\mu\text{g/L}$, with mean blood mercury level 1.7 $\mu\text{g/L}$.

Analysis of all other data revealed no significant findings.

Discussion :

The mean blood mercury level in the control group is nearly similar to those in other countries. In the literature review there are different values for BMLs. Table (2) shows some different BMLs in different countries.¹⁹⁻²⁸

Table (2) Blood Mercury blood levels in non-exposed populations

Country	No. of Subjects	Mean level $\mu\text{g/L}$	Level range $\mu\text{g/L}$	References ¹⁹⁻²⁸
Belgium	497	13	0.1-47	Lauwerys et al. (1978)
Italy	110	6.36		Alimonti A. et al (2005)
UK	88	8.8	1.1-42	Sherlock et al. (1982)
Poland	270	11.3	2.5-24	Szucki & Kurys (1982)
Germany		2		Ewers U et al (1999)
USA	1709	1.02 0.82	0.85-1.2	Schober et al. (2003) Jones et al. (2010)
South Korea	293 581	8.63 3.92	1.48 – 45.7	Eun-mi Jo (2010) Kim NY et al. (2012)
Canada	492	1.6	0.8 – 11.2	<i>Dewailly E et al. (2001)</i>
Sweden	106	0.34	0.04 – 1.6	<i>Johansson N et al. (2002)</i>

In Libya, there are no studies about the reference level of blood mercury in the Libyan population. This control group is too small to be considered as a reference population. The reference population should be sufficiently large to cover a representative part of the general population and to enable an evaluation of the effect of relevant confounders on the

level of a toxin in human biological materials (e.g., age, sex, tobacco smoking, amalgam fillings, special nutritional habits)²³.

The range of BMLs among the study group is 0.6–16 µg/L and the mean level is 5.86 µg/L. According to the blood mercury levels, the study group is divided into 4 subgroups (Table 3).

Table (3) Subgroups according to MBLs and Clinical Manifestations

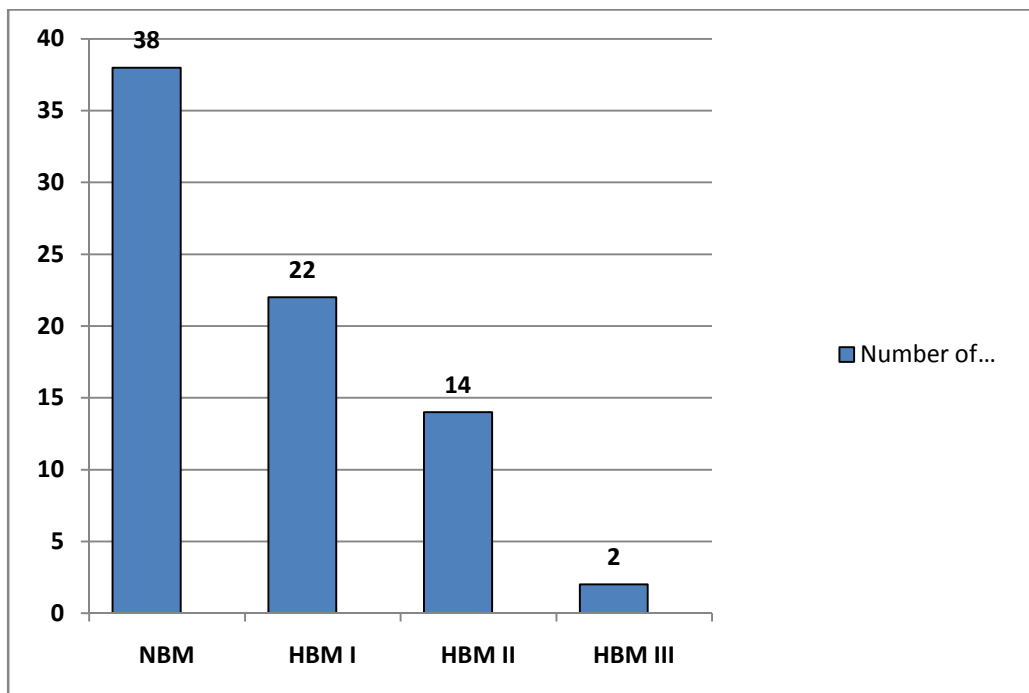
Subgroup	Blood Mercury Level µg/L	Number
NBM	1.7 or less	38
HBM I	> 1.7 – 5	22
HBM II	> 5 - < 15 without clinical manifestations	14
HBM III	> 5 with clinical manifestations/ or more	2

- 1) Normal Blood Mercury (NBM): a subgroup with “*normal value*”, in which the blood mercury levels are considered normal i.e. 1.7 µg/L or less. In this subgroup there are 38 individuals i.e. 50% of the study group.
- 2) High Blood Mercury I (HBM I): this subgroup is considered as a “*check value*”, which means an elevated mercury level in blood above that in the control group and up to 5µg/L, in which other different possible sources of mercury should be eliminated. At this level there is no consideration of adverse health effects²³. This subgroup includes 22 individuals (28.59%).
- 3) High Blood Mercury II (HBM II): where blood mercury levels are

above 5µg/L and below 15 µg/L but without clinical manifestations of mercury intoxication. This value is considered as an “*observation value*” where the exposure to mercury should be stopped and the individuals are put under close observation. The level 15 µg/L was considered by the Commission on Human Biological Monitoring of the German Federal Environmental Agency²³. This subgroup includes 14 individuals (18.4%).

- 4) High blood Mercury Level III (HBM III): the blood level in this subgroup is considered as the “*intervention value*”. It includes those with BMLs above 5µg/L but with the presence of clinical manifestation of mercury toxicity, or those with BMLs 15 µg/L²³ or more. The individuals in this subgroup will undergo specific management one of which is chelation therapy in addition to repeated investigations of mercury levels in the biological fluids. This subgroup includes 2 technicians (0.26%); one with BML 15 µg/L and the other with BML 16 µg/L. All technicians examined in the study group are without clinical manifestations of mercury intoxication.

Fig.1 Distribution of BML among study group



People using mercury in their work are having the risk of exposure to mercury from other sources similar to the population they are living with. These sources should be considered when we are planning for their safety precautions or when we are managing them to decrease their mercury blood levels. The workers, as a part of the general population, will be exposed to mercury through the diet (especially fish), air, water, tobacco smoking and dental amalgams²³. Also, personal use of skin-lightening creams and soaps, mercury use for cultural purposes can result in substantial elevations of human mercury exposure.

In order to decrease the exposure to different types of mercury, several countries

and international organizations have established levels of daily or weekly mercury intakes estimated to be safe (or without appreciable risk to health), based on available information. The Joint FAO/WHO Expert Committee on Food Additives (JECFA), which also evaluates chemical contaminants in the food supply, has established provisional tolerable weekly intakes (PTWIs) for total mercury at 5 µg/kg body weight and for methylmercury at 1.6 µg/kg body weight.²⁸

Further rules had been established to protect those working with mercury from its hazardous effects. The most important are the legal exposure limits for mercury during work. In United States of America, the Occupational Safety and Health Administration (OSHA-USA) gives permissible exposure limit for mercury vapor (0.1 mg/m³ of air as a ceiling limit. The National Institute for Occupational Safety and Health (NIOSH-USA) has established a recommended exposure limit for mercury vapor of 0.05 mg/m³ as a Time Weighted Average (TWA) for up to a 10-hour workday and a 40-hour workweek. The American Conference of Governmental Industrial Hygienists (ACGIH-USA) has assigned mercury vapor a threshold limit value (TLV) of 0.025 mg/m³ as a TWA for a normal 8-hour workday and a 40-hour workweek.²⁹

All different sources of mercury should be put in consideration when planning for the safety of workers with mercury including diet where amount and type of fish must be selected to be lesser than 5 µg/kg body weight and for methylmercury at 1.6 µg/kg body weight. Another

important issue is that concerning the prohibition of use of dental amalgams among people who are with higher risk of mercury to toxicity. Because of risks of dental amalgams Denmark, Sweden and Norway have banned the use of dental amalgams and other countries are going to follow them.³⁰

Conclusion :

- 1- Mercury blood levels in 38/76 (50%) of Libyan technicians were high.
- 2- All individuals in the study group are free of clinical manifestations.
- 3- Control group is too small to be considered as a reference level and larger groups must be studied to estimate reference level of blood mercury among Libyan population.

Recommendations :

- 1) Further studies should be carried in the Libyan population to estimate reference level of blood mercury.
- 2) Mercury exposure must be decreased by following standard safety precautions and recommendations.
- 3) People who are working in occupations with higher risk of mercury poisoning must be educated about their diet, habits. and they should not use dental amalgams containing mercury.

References:

- 1) *Goldwater LJ. (1936): From Hippocrates to Ramazzini: early history of industrial medicine. Ann Med Hist8:27.*
- 2) *Risher JF (2003): Elemental Mercury & Inorganic Mercury*

- Compounds: Human Health Aspects. WHO (medline).*
- 3) Jao-Tan C, Pope E. (2006): *Cutaneous poisoning syndromes in children: a review. Curr Opin Pediatr*18(4):410-6
 - 4) Friberg L, Mottet NK. (1989): *Accumulation of methylmercury and inorganic mercury in the brain. Biol Trace Elem Res ;*21:201-206
 - 5) Jung-Duck Park, Wei Zheng (2012): *Human Exposure and Health Effects of Inorganic and Elemental Mercury. J Prev Med Public Health;* 45:344-352
 - 6) Clarkson TW. (1997). *The toxicology of mercury. Crit Rev Clin Lab Sci.;*34: 369–403
 - 7) Weil M, Bressler J, Parsons P, Bolla K, (2005): *Blood mercury levels and neurobehavioral function. JAMA.* 293(15):1875-82.
 - 8) Takeuchi T, Eto K, Kinjo Y, Tokunaga H. (1996): *Human brain disturbance by methylmercury poisoning, focusing on the long-term effect on brain weight. Neurotoxicology.* 17(1):187-90.
 - 9) Kang-Yum E, Oransky SH (1992) *Chinese patent medicine as a potential source of mercury poisoning. Veterinary and Human Toxicology,* 34(3):235–238.
 - 10) Carvalho CM, Chew EH, Hashemy SI, Lu J, Holmgren A. (2008): *Inhibition of the human thioredoxin system. A molecular mechanism of mercury toxicity. J Biol Chem.* 283(18):11913-23
 - 11) Basu N, Scheuhammer A, Rouvinen-Watt K, Evans R, Grochowina N, Chan L. *Effects of mercury on muscarinic cholinergic receptor*

- subtypes (M1 and M2) in captive ink. Neurotoxicology. Mar 2008;29(2):328-34.*
- 12) Huang CF, Hsu CJ, Liu SH, Lin-Shiau S. (2008): *Neurotoxicological mechanism of methylmercury induced by low-dose and long-term exposure in mice: oxidative stress and down-regulated Na⁺/K⁺-ATPase involved. Toxicol Lett. 176(3):188-97.*
- 13) Bellinger D, Trachtenberg F, Barregard L, et al. (2006): *Neuropsychological and renal effects of dental amalgam in children: a randomized clinical trial. JAMA. 295(15):1775-83.*
- 14) Boyd A, Seger D, Vannucci S, Langley M, Abraham J (2000): *Mercury exposure & cutaneous disease. J Am Acad Dermatol. 43:81-90.*
- 15) ATSDR (1999): *Toxicological profile for mercury (update). Atlanta, GA, US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, March.*
- 16) Virtanen j, Voutilainen S, Rissanen T, Mursu K, Tuomainen T, et al (2005): *Mercury, Fish Oils, and Risk of Acute Coronary Events and Cardiovascular Disease, Coronary Heart Disease, and All-Cause Mortality in Men in Eastern Finland. Atherosclerosis, Thrombosis, and Vascular Biology, 25, 228-233.*
- 17) De Rosis F, Anastasio SP, Selvaggi L, Beltrame A (1985): *Female reproductive health in two lamp factories: effects of exposure to inorganic mercury vapour and stress factors. British Journal of*

- Industrial Medicine*, 42:488–94.
- 18) Davis B, Price H, O’connor R, et al (2001): *Mercury Vapor and Female Reproductive Toxicity*. *Toxicological Sciences*,59, 291-296.
- 19) Lauwerys [R](#), [Buchet JP](#), [Roels H](#), [Hubermont G](#).(1978). *Placental transfer of lead, mercury, cadmium, and carbon monoxide in women. I. Comparison of the frequency distributions of the biological indices in maternal and umbilical cord blood*. [Environ Res](#). Apr;15(2):278-89
- 20) Alimonti A, Bocca B, Mannelia E, Petrucci F, Zennaro F, Cotchini R, D’ippolito C, Agresti A, Caimi S, Forte G (2005). *Assessment of reference values for selected elements in a healthy urban population*. *Ann Ist Super Sanità*;41(2):181-87
- 21) Sherlock, JC; Lindsay, DG; Hislop, J; et al. (1982) *Duplication diet study on mercury intake by fish consumers in the United Kingdom*. *Arch Environ Health* 37(5):271-78.
- 22) Szucki & Kurys (1982). *Mercury content in the blood and hair in the general population*. [Rocz Panstw Zakl Hig](#). 33(3):143-48 (translated from Polish).
- 23) Ewers U, Krause C, Schulz C, Wilhelm (1999): *Reference values and human biological monitoring values for environmental toxins. Report on the work and recommendations of the Commission on*

- Human Biological Monitoring of the German Federal Environmental Agency. Int Arch Occup Envir Health;72: 255-60*
- 24) Schober E, Sinks, Jones, Michael Bolger, McDowell M., Osterloh, Spencer Garrett, Canady, Dillon, Yu Sun, Joseph, Mahaffey. (2003). *Blood Mercury Levels in US Children and Women of Childbearing Age, 1999-2000. JAMA. Vol 289, 13, 1667-74.*
- 25) Jones L., Parker J, Pauline Mendola. (2010). *Blood Lead and Mercury Levels in Pregnant Women in the United States, 2003–2008. NCHS Data Brief No. 52 (data from the 1999-2008 continuous National Health and Nutrition Examination Survey (NHANES))*
- 26) Eun-Mi Jo, Byoung-Gwon Kim, Yu-Mi Kim, Seung-Do Yu, Chang-Hun You, Joon-Youn Kim, and Young-Seoub Hong (2010). *Blood Mercury Concentration and Related Factors in an Urban Coastal Area in Korea. J Prev Med Public Health. Sep;43(5):377-86.*
- 27) Kim N, Ahn S, Ryu D, Chi B, Kim H. et al. (2012): *Effects of lifestyles on blood mercury in Korean adults. Human & Experimental Toxicology –15;1-9*
- 28) Dewailly E, Ayotte P, Bruneau S, Germain L, Levallois P, Weber JP. (2001). *Exposure of the Inuit population of Nunavik (Arctic Quebec) to lead and mercury. Arch Environ Health; 56:350–57*
- 29) Johansson N, Basun H, Winblad B, Nordberg M. (2002).

Relationship between mercury concentration in blood, cognitive performance, and blood pressure, in an elderly urban population. Biometals; 15:189–95.

- 30) WHO (United Nations Environmental Programme) 2008: *Guidance for Identifying Populations at Risk from Mercury Exposure.*
www.who.int