

Animal welfare, etológia és tartástechnológia



Animal welfare, ethology and housing systems

Volume 9

Issue 3

Különszám/Special Issue

Gödöllő

2013

THE CONTENT OF MERCURY IN BODY OF MEN TREATED IN CRACOW ONCOLOGY AND SPECIALIST HOSPITALS

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ABSTRACT

The aim of the study was to determine the content of mercury both in sick (cancerous) and healthy men in relation age and state of health. The samples coming from men lived in Cracow and area around of the city. Healthy tissues were collected during autopsy, cancerous tissues during surgery.

The content of mercury in the samples was measured using cold vapor atomic absorption spectrometry method (CVAAS). The fragments of esophagus, stomach, small intestine, large intestine, liver, pancreas, kidney, bladder and skin tissues (healthy tissues) and stomach, large intestine, kidney and bladder (cancerous tissues), were placed in a special crucible directly after defrosting and weighing.

The average mercury content in healthy men was significantly higher in the age group 51-90 years old (0,027 ppm), compared to young men (0,008 ppm). Similar differences were obtained for sick men - older men have a slightly higher content of mercury in the organs (0,106 ppm) than men younger (0,088 ppm).

Key words: mercury, neoplasm, CVAAS, cancer

Introduction

Mercury is a heavy metal occurring 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_2^{2+} 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 (**Clifton, 2007**). Mercury poisoning can result in several diseases, including acrodynia (pink disease) (**Bjorklund, 1995**), Hunter-Russell syndrome, and Minamata disease (**Davidson et al., 2004**). 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. 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). Mercury irreversibly inhibits selenium-dependent enzymes (see below) and may also inactivate S-adenosyl-methionine, which is necessary for catecholamine catabolism by catechol-o-methyl transferase. Due to the body's inability to degrade catecholamines (e.g. epinephrine), a person suffering from mercury poisoning may experience profuse sweating, tachycardia (persistently faster-than-normal heart beat), increased salivation, and hypertension (high blood pressure).

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 dysfunction (e.g. Fanconi syndrome) or neuropsychiatric symptoms such as emotional lability, memory impairment, and/or insomnia. Thus, the clinical presentation may resemble pheochromocytoma or Kawasaki disease. An example of desquamation (skin peeling) of the hand of a child with severe mercury poisoning acquired by handling elemental mercury is this photograph in **Horowitz, et al. (2002)**.

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 (**USEPA, 1997**). Exposure to mercury can occur from breathing contaminated air, (**ATSDR, 1999**) from eating foods that have acquired mercury residues during processing, (**Dufault et al., 2009**) from exposure to mercury vapor in mercury amalgam dental restorations, (**Levy, 1995**) 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 (**Goldman and Shannon, 2001**). Human-generated sources, such as coal plants, emit about 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, nonferrous metal production, cement production, waste disposal, human crematoria, caustic soda production, pig iron and steel production, mercury production (mostly for batteries), and biomass burning (**Pacyna et al., 2006**).

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 (**USEPA, 1997**).

Material and methods

Research was conducted on samples taken from different segments of human digestive tract and human urinary tract. Healthy tissues were taken during autopsy, cancerous tissues during surgery from Military Hospital, PROSMED Health Center in Cracow and Cancer Pathology the Oncology Centre of Maria Skłodowska-Curie in Cracow. Permission for research was given by Local Bioethical Commission. Samples were taken from 24 healthy men (20-90 years old). Fragments of normal tissues were taken from esophagus (n=24 samples), stomach (n=24 samples), small intestine (n=24 samples), large intestine (n=24 samples), liver (n=22 samples), pancreas (n=22 samples), kidney (n=22 samples), bladder (n=21 samples) and skin (n=8 samples). Fragments of cancerous tissues were taken from 265 sick men (20-90 years old) from stomach tumor (n=18 samples), large intestine tumor (n=138 samples), kidney tumor (n=98 samples), bladder tumor (n=11 samples). Average mass of each sample hesitated from 0,5-1g.

Mercury contents were detected using CVAAS methods. This method does not require mineralization and allows the measurement directly in tissue. The small fragments of tissues were placed into the special crucible directly after defrosting and precise weighing. Results of weighting were recorded in the computer using the program, which operate spectrophotometer MA2. The measuring was repeated three times for each sample. All results were expressed in ppm.

Results

Results were analyzed statistically by using Statistica 10 program. In statistical description the following statistics were used: the arithmetic mean and standard deviation. Normality of the distribution was examined by Shapiro-Wilk test. Because none of the normal distributions were noticed, Kruskal-Wallis test for independent trials was used. Statistical analysis was to use the U Mann-Whitney test, to check the differences in the average content of mercury between the two age groups separately for men, healthy and sick. Results are shown in Figure 1 and Figure 2.

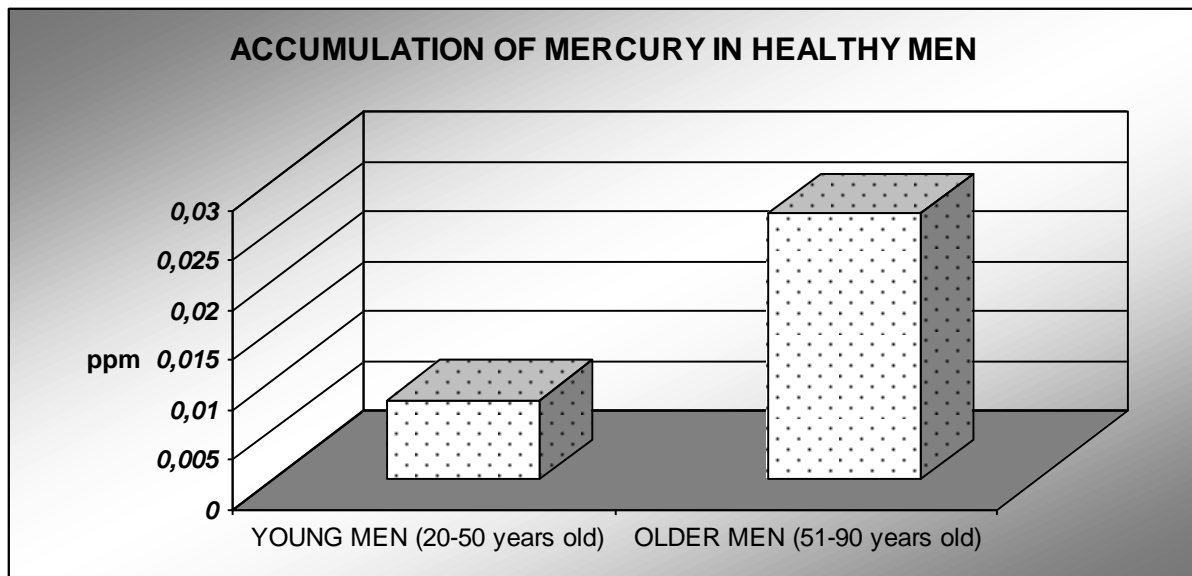


Fig. 1

Accumulation of mercury in healthy men organs

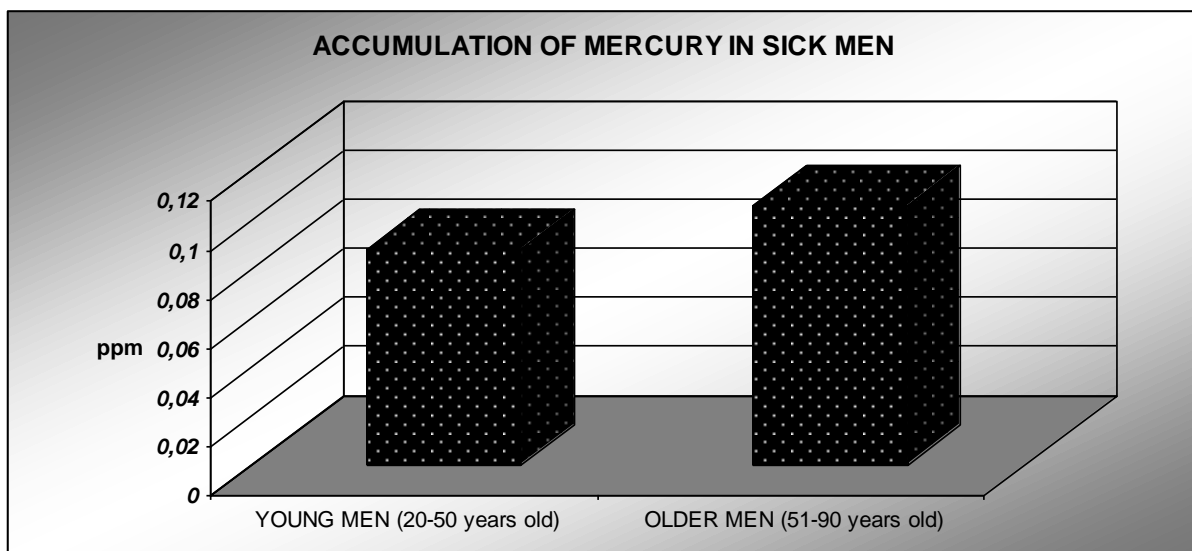


Fig. 2

Accumulation of mercury in sick men organs

Mean content of mercury for tissues young (<50) healthy men is 0,008 ppm. The average content of mercury for tissues older (>50) healthy men is 0,027 ppm. The average content of mercury for tissues young (<50) sick men is 0,088 ppm. The average content of mercury for tissues older (>50) sick men is 0,106 ppm. Statistically significant differences were found between healthy and sick men tissues. In the case of healthy tissues, mercury concentrations was higher in older men.

Discussion and conclusions

In our study we determined presence of Hg in cancerous and normal (healthy) tissues of similar men's organs (bladder, kidney, esophagus, stomach, small intestine, large intestine, liver, pancreas and skin). Compounds of mercury tend to be much more toxic than the elemental form, 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. 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.

Metals – such as mercury - are involved in different physical and chemical processes, which are common in cancer development. There are different effects caused by metals in the process of carcinogenesis, such as: damage in DNA, formation of DNA cross-links, the interference of metals with DNA-repair enzymes. Some metals affect the transduction signal by initiation of gene expression disturbances and intracellular communication. Moreover metals may cause of negative effects of the immune system function and disturbances of cellular homeostasis. It is crucial to realize that mercury is especially dangerous because it can be found in things and places which are surrounding us for example tuna has a capacity to accumulate high quantities of mercury in muscles.

It can be stated unequivocally much higher mercury content in cancerous tissues of sick men (0,106 ppm and 0,088 ppm), compared to healthy tissues (control group) (0.027 ppm and 0,008 ppm). Older people have a greater tendency to accumulate heavy metals in their tissues, perhaps because the metals are not the ability to direct carcinogenesis, may interact with other carcinogenic factors, such as metals can inhibit the DNA repair process, increasing the likelihood of mutation and, as a thus - cancer (**Hartwig, 1998, Hu et al., 2004a, 2004b**). The nucleic acids are capable of intermolecular interaction with different chemical compounds and metal ions.

References

- ATSDR Mercury ToxFAQ. 1999. ToxFAQs: Mercury. Agency for Toxic Substances and Disease Registry. Retrieved 2007-07-25.
- Bjørklund, G. 1995. Mercury and Acrodynia. *Journal of Orthomolecular Medicine* 10, 145–146.
- Clifton, J.C. 2007. Mercury exposure and public health. *Pediatr Clin North Am* 54 (2): 237–69.
- Davidson, P.W., Myers, G.J., Weiss, B. 2004. Mercury exposure and child development outcomes. *Pediatrics* 113 (4 Suppl): 1023–9.
- Dufault, R., LeBlanc, B., Schnoll, R. et al. 2009. Mercury from chlor-alkali plants: measured concentrations in food product sugar. *Environ Health* 8 (1): 2.
- Goldman, L.R., Shannon, M.W. Technical report: mercury in the environment: implications for pediatricians. *Pediatrics* 108 (1): 197–205.
- Hartwig, A. 1998. Carcinogenicity of metal compounds: possible role of DNA repair inhibition. *Toxicology Letters* 102-103, 235-239.
- Horowitz, Y., Greenberg, D., Ling, G., Lifshitz, M. 2002. Acrodynia: a case report of two siblings. *Arch Dis Child* 86 (6): 453.
- Hu, W., Feng, Z., Tang, M.S. 2004. Chromium(VI) enhances (+/-)-anti-7beta,8alpha-dihydroxy-9alpha,10alpha-epoxy-7,8,9,10 tetrahydrobenzo[a]pyrene-induced cytotoxicity and mutagenicity in mammalian cells through its inhibitory effect on nucleotide excision repair. *Biochemistry* 43, 14282-14289.
- Hu, W., Feng, Z., Tang, M.S. 2004. Nickel (II) enhances benzo[a]pyrene diol epoxide-induced mutagenesis through inhibition of nucleotide excision repair in human cells: a possible mechanism for nickel (II)-induced carcinogenesis. *Carcinogenesis* 25, 455-462.
- Levy, M. 1995. Dental Amalgam: toxicological evaluation and health risk assessment. *J Cdn Dent Assoc* 61: 667–8, 671–4.
- Pacyna, E.G., Pacyna, J.M., Steenhuisen, F., Wilson, S. 2006. Global anthropogenic mercury emission inventory for 2000. *Atmos Environ* 40 (22): 4048–63.
- United States Environmental Protection Agency. 1997. *Mercury Study Report to Congress 3*. Washington, D.C.: United States Environmental Protection Agency.
- United States Environmental Protection Agency. 1997. *Mercury Study Report to Congress 4*. Washington, D.C.: United States Environmental Protection Agency.