

# Impact of occupational elemental Mercury exposure on the health of miners in the Idrija Mercury Mine

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**Abstract:** We are presenting the incidence of elemental mercury vapours ( $\text{Hg}^0$ ) intoxications, some results of post-mortem studies, mortality causes among miners and its possible relation with  $\text{Hg}^0$  exposure. The miners of the Idrija Mercury Mine were exposed to fibrogenic dusts, ionizing radiation and  $\text{Hg}^0$ . In the initial years following the Second World War, the incidence of mercury poisoning in miners reached up to 145 cases per year. Postmortem studies have shown that mercury accumulations in ex-miners are predominant in the endocrine glands, kidneys and brain tissue. An increased mortality due to lung cancer, pneumoconiosis, nephritis/nephrosis, ischaemic heart diseases (IHD) and suicide was detected in workers of the Idrija Mercury Mine.

**Key words:** elemental mercury, occupational exposure, miner's health

## INTRODUCTION

Mercury was discovered accidentally in Idrija around the year 1490. Over a period of 500 years, the Idrija Mine has produced a total of 147,000 tons of mercury. After the Second World War, the mine operated with full capacity until 1977. After this period, mercury production began to decline and excavation works were stopped in 1994, when the mine's gradual shutdown was begun.

Teophrastus Paracelsus was the first to write about »mercurialism« among Idrija's workers in his book »Von der Bergsucht und anderen Krankheiten« (1527). Johannes Antonius Scopoli was the first physician appointed in Idrija in 1754. He described in great detail the symptoms of "mercurialism" among pit workers and the preventive measures re-

quired during exposure to elevated concentrations of  $\text{Hg}^0$  vapour (SCOPOLI, 1771).

Occupational exposure in mercury mining and the classical symptoms and signs of mercurialism have been well described by many authors and reviewed in WHO monographies (1967, 1991, 2003). The association between  $\text{Hg}^0$  exposure and long-term mercury toxicity effect is complicated because the onset of symptoms and signs may be delayed for up to several years (ELLINGSEN ET AL., 1993; LETZ ET AL., 2000). The aim of our study is to briefly present the characteristics of work in the Idrija Mercury Mine, the incidence of  $\text{Hg}^0$  intoxications, the results of biological monitoring studies and post-mortem studies, and the causes of mortality among miners in the past 50 years and its possible relation to  $\text{Hg}^0$  exposure.

## WORKING CONDITIONS AND INCIDENCE OF MERCURY INTOXICATION

Intermittent exposure to  $Hg^0$  was characteristic for miners of the Idrija Mercury Mine. During the course of their work, miners were exposed to silica dusts, ionising radiation and elemental mercury. In areas where native mercury (naturally occurring  $Hg^0$ ) is found, the mercury concentrations are considerably increased (over  $2.5 \text{ mg/m}^3$ ). Radon concentrations in upper parts of the mine were found to be  $700\text{-}21000 \text{ Bq/m}^3$ .

Miners and other workers at the smelting plant in Idrija suffered mostly from subacute and chronic mercury poisoning. Their clinical picture is dominated by tremor, oropharyngeal syndrome, and cretismus mercurialis. Their urine mercury concentrations varied from 300 to  $1000 \text{ }\mu\text{g}$  in 24-hour urine samples (HRIBERNIK, 1950; HUNTER, 1957; KOBAL, 1975). In the years following the Second World War, the incidence of mercury poisoning was very high: 145 workers were intoxicated in 1953 (HRIBERNIK, 1950). In the years from 1964-1967, 25 % of the workers in the mine were engaged in the extraction of native mercury in areas with very high  $Hg^0$  concentrations (up to  $3 \text{ mg/m}^3$  in unventilated areas). In this period, the number of occupational intoxications within the mercury mine exceeded 10 % of the number of employed workers (e.g., in 1946 there were as many as 104 intoxications). As regards the number of working days lost, occupational intoxication was the third major cause after respiratory diseases and injuries (KOBAL, 1964).

## RESEARCH ACTIVITIES RELATED TO BIOLOGICAL MONITORING

In the period after 1964 the Health Service focused its activities more intensively on the preventive health care of workers and became increasingly involved in research activities. In the above-mentioned period, miners' exposure was assessed by measuring mercury in various biological media and focused catalase activity in erythrocytes as a potential biological effect of  $Hg^0$  exposure.

In order to estimate the relationship between  $Hg$  concentrations in inhaled air and internal doses of mercury in the blood, individual dosimetry was performed on groups of pit workers exposed to low  $Hg$  concentrations in air. The correlation and regression between mercury levels in the blood and inhaled breath ( $r=0.96$ ;  $P=0.000$ ;  $Y=350x \pm 9.7$ ), between urine and blood ( $r=0.68$ ,  $P<0.001$ ,  $Y=1.35x \pm 14.3$ ) and between  $Hg^0$  in exhaled breath and blood – erythrocytes ( $r=0.93$ ,  $P=0.000$ ,  $Y=3.98x \pm 15.9$ ) were evaluated.

The results obtained enable definition of the ratio between the air blood mercury concentration ( $1 \text{ g/m}^3$ :  $0.54 \text{ }\mu\text{g/L}$ ), the blood-urine mercury concentration ( $1 \text{ }\mu\text{g/L}$ :  $2.7 \text{ }\mu\text{g/L}$ ), and the air-urine mercury concentration ( $1 \text{ }\mu\text{g/L}$ :  $1.0 \text{ }\mu\text{g/L}$ ), which served as a basis for the application of biological action levels in miners during intermittent exposure. Based on a blood level of  $35 \text{ }\mu\text{g/L}$  recommended by WHO (1976) as a potential biological action level and our quoted results, a urine mercury concentration of  $60 \text{ }\mu\text{g/L}$  was used as the biological action level.

The blood excretion kinetics displayed two phases: a fast initial phase, and a slow excretion phase ( $C_{(T)} = 43.6 e^{-0.117T} + 8.2 e^{-0.0035T}$ ). The concentration of mercury in blood increases immediately after exposure. The excretion kinetics of mercury in urine varied significantly from person to person. Increased mercury concentrations in urine were usually observed a few days after exposure. The excretion half-time of mercury derived from concentrations of exhaled breath was very short immediately after exposure, ranging from 26 to 120 minutes in the first two hours after exposure, and from 11 to 28 hours in the second to twentieth hour after exposure (KOBAL, 1991).

As a marker of biological effect monitoring the catalase activity in erythrocytes was determined before and during exposure. Our results suggest that catalase activity in erythrocytes also depends on current concentrations of »elemental« mercury in the blood. At an average mercury concentration in blood of 50 µg/L, a significant decrease of catalase activity in erythrocytes was observed (KOBAL, 1991; KOBAL ET AL., 1999).

Due to ethical and methodological obstructions, the determination of miners' U-Hg has remained a basis for the biological monitoring of exposure. Following the intensification of target medical examinations the applications of "allowable time" (duration and frequency) of exposure to native Hg based on Haber's Law (KOBAL, 1975) and other health safety measures (KOBAL, DIZDAREVIČ, 1997), a continuous decrease in the incidence of mercury intoxications was achieved. The number of intoxicated workers fell from 104 in 1964 to 2 in 1975.

## LONG –TERM HEALTH EFFECTS

### Postmortem studies of miners

Postmortem studies (KOSTA ET AL., 1975; FALNOGA ET AL., 2002) have shown that the accumulation of mercury in ex-miners who had been occupationally exposed to mercury was higher than among the inhabitants of Idrija. Most of the mercury was retained in the pituitary gland (19000 – 64000 ng/g of fresh tissue), thyroid gland (18000 – 41000 ng/g of fresh tissue), kidneys (9430 – 18530 ng/g of fresh tissue), liver (108 – 791 ng/g of fresh tissue) and brain tissue (dentate nucleus). Several years after the cessation of exposure, significant amounts of Hg and selenium were found in various tissues of retired mercury miners. Selenium co-accumulation was notable only in samples with mercury concentrations over 2 µg/g. In these cases, the Hg/Se molar ratio equated (1:1) or exceeded unity.

### Mortality causes among miners

We participated in an IARC multicentric study of cancer risk (BOFFETA ET AL., 1998; BOFFETA ET AL., 2001). The project involved the study of mortality among the workers of four mercury mines in Spain, Slovenia, Italy and the Ukraine. A slight increase in overall mortality was observed among workers of the Idrija Mercury Mine (SMR 1.18, CI 1.09 - 1.28) and the Almaden Mine in Spain (SMR 1.14, CI 1.09 - 1.20). In miners of the Idrija Mercury Mine, an increase in mortality from lung cancer (SMR 1.89, 95 % CI 1.45 – 2.41), pneumoconiosis (SMR 13.6, 95 % CI 2.81 – 39.9), nephritis – nephrosis (SMR 1.60, 95 % CI 0.44 – 4.10), IHD (SMR 1.60, 95 % CI 1.35 – 2.02) and suicide (SMR 1.23, 95 % CI 0.88 – 1.68) was found.

The increased lung cancer risk was limited to miners of the Idrija Mercury Mine and miners of the Nikitovka Mercury Mine in the Ukraine. Co-exposure to silica dust and underground exposure to radon could be additional contributing factors increasing the lung cancer risk among the miners of these two mines (IARC, 1997; BOFFETTA ET AL., 1998). Mortality due to nephritis and nephrosis was increased in miners from Spain and Slovenia. The increased risk was associated with the increasing duration of employment. The co-exposure to silica dust and Hg<sup>0</sup> could be associated with increased mortality from kidney disease owing to the known nephrotoxic effect of both agents (WHO, 1967; IARC, 1997; KOBAL ET AL., 2004). Increased mortality due to IHD was found only among miners of the Idrija Mercury Mine, while increased mortality due to hypertension was observed in miners of Almaden and Nikitovka in the Ukraine. Limited information is available on the effects of chronic mercury exposure and the risk of cardiovascular disease (SALONEN ET AL., 1995, BOFFETTA ET AL., 2001). The results of our recent study (KOBAL ET AL., 2002) support the assumption that long-term occupational exposure to Hg<sup>0</sup> enhances the formation of free radicals even several years after termination of occupational exposure, and could therefore be one of the risk factors for increased lipid peroxidation and increased mortality due to ischaemic heart disease found

among mercury miners of the Idrija Mine. Long-term intermittent occupational exposure to Hg<sup>0</sup> in interaction with alcohol remains a plausible explanation for depression and negative self-concept in ex-mercury miners established by our ongoing study in the period after exposure (KOBAL ET AL., 2004). This can significantly decrease resistance towards psychosocial stress factors, and increase the risk of suicide.

## CONCLUSIONS

Biological monitoring ensures that the exposure of miners to native mercury does not entail an unacceptable health risk – mercury intoxication, but was unable to prevent the increased absorption of Hg<sup>0</sup> in about 40 % of exposed miners. Long-term increased Hg<sup>0</sup> absorption, co-exposure to silica dust and radon, and miners' lifestyle-related factors could be responsible for the long-term health effects observed in miners of the Idrija Mercury Mine: increased risk of mortality from lung cancer, nephritis-nephrosis, ischaemic heart diseases, and suicide. The presented observations further support efforts to reduce the occupational level of Hg<sup>0</sup> to the lowest observed adverse effect level, which can prevent not only the known toxic effect but also the potential long-term effects of Hg<sup>0</sup> exposure especially in interaction with other risk factors.

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