Code of Practice
Control of Worker Exposure to Mercury in the Chlor-alkali Industry

Health 2

7th edition

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EURO CHLOR PUBLICATION

This document can be obtained from:
EURO CHLOR - Avenue E. Van Nieuwenhuyse 4, Box 2 - B-1160 BRUSSELS
Telephone: 32-(0)2-676 72 65 – Telefax: 32-(0)2-676 72 41

Euro Chlor

Euro Chlor is the European federation which represents the producers of chlorine and its primary derivatives.

Euro Chlor is working to:

- improve awareness and understanding of the contribution that chlorine chemistry has made to the thousands of products which have improved our health, nutrition, standard of living and quality of life;
- maintain open and timely dialogue with regulators, politicians, scientists, the media and other interested stakeholders in the debate on chlorine;
- ensure our industry contributes actively to any public, regulatory or scientific debate and provides balanced and objective science-based information to help answer questions about chlorine and its derivatives;
- promote the best safety, health and environmental practices in the manufacture, handling and use of chlor-alkali products in order to assist our members in achieving continuous improvements (*Responsible Care)*.

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Prior to 1990, Euro Chlor's technical activities took place under the name BITC (Bureau International Technique du Chlore). References to BITC documents may be assumed to be to Euro Chlor documents.

RESPONSIBLE CARE IN ACTION

Chlorine is essential in the chemical industry and consequently there is a need for chlorine to be produced, stored, transported and used. The chlorine industry has co-operated over many years to ensure the well-being of its employees, local communities and the wider environment. This document is one in a series which the European producers, acting through Euro Chlor, have drawn up to promote continuous improvement in the general standards of health, safety and the environment associated with chlorine manufacture in the spirit of *Responsible Care*.

The voluntary recommendations, techniques and standards presented in these documents are based on the experiences and best practices adopted by member companies of Euro Chlor at their date of issue. They can be taken into account in full or partly, whenever companies decide it individually, in the operation of existing processes and in the design of new installations. They are in no way intended as a substitute for the relevant national or international regulations which should be fully complied with.

It has been assumed in the preparation of these publications that the users will ensure that the contents are relevant to the application selected and are correctly applied by appropriately qualified and experienced people for whose guidance they have been prepared. The contents are based on the most authoritative information available at the time of writing and on good engineering, medical or technical practice but it is essential to take account of appropriate subsequent developments or legislation. As a result, the text may be modified in the future to incorporate evolution of these and other factors.

This edition of the document has been drawn up by the Health Working Group to whom all suggestions concerning possible revision should be addressed through the offices of Euro Chlor.

MAIN MODIFICATIONS IN THIS VERSION

Section	Nature
5.2.1	Update of OELs values

TABLE OF CONTENTS

ANNEXES

EXECUTIVE SUMMARY

This Code of Practice for Control of Worker Exposure to Mercury in the Chlor-Alkali Industry has been written for managers, plant engineers and local occupational health professionals to enable them to protect the health of workers against harmful effects of exposure to mercury.

Approximately 90% of all metallic mercury in inhaled air is absorbed in the body. In the case of chronic overexposure, mercury will accumulate in several organs because of its long half life in the body which varies between 50 and 90 days in most organs, and may possibly be for a period of years in the brain. Mercury is mainly excreted via urine and faeces.

The nervous system appears to be the most sensitive target for mercury toxicity. Although there is a lack of consistency across a large number of the published studies, several subclinical neurological effects have been reported. Mercury is also toxic to the kidney, and it is well recognised that high levels of exposure can lead to "nephrotic syndrome". Proteinuria and enzymuria, not associated with clinical disease or loss of function, have been reported at mercury in urine (HgU) levels of more than 30 µg/g creatinine[1]. Mortality studies have not shown an excess of death due to chronic renal disease.

It is known that mercury can easily cross the placenta and the foetal blood-brain barrier. Mercury could therefore conceivably affect the development of the unborn child and as a consequence will be classified in the EU for its developmental toxic effects (category 2 R61 - May cause harm to unborn child). Women of reproductive age, working with mercury, should be made aware of this potential hazard and if willing to become pregnant, should be advised to consult an occupational physician to discuss potential measures to be taken in her work situation to exclude possible damage to the unborn child. For women who are pregnant, have recently given birth, or are breastfeeding, EC Directive 92/85 is applicable. Mercury is not classified as a carcinogen.

It is of great importance to understand that a combination of plant design, good housekeeping and personal hygiene is essential to prevent the uptake of mercury into the human body. Mercury contaminated clothes in particular can be a significant source of exposure. This document contains advice on how to deal with these problems. Where significant exposure is anticipated, effective personal protective equipment should be used.

To minimize workers' exposure to mercury, Euro Chlor proposes a system for health management of mercury-related processes which is based on continuous

[1] The results of urinary mercury measurement are expressed in µg/g of creatinine in order to refer to a "constant" excretion parameter.

improvement. This approach is supported by a written health policy and management system which is communicated to all potentially exposed employees, in which the work processes related to the management of exposure to mercury are described, and responsibilities and tasks are delegated to responsible functions within the organisation. Items which should be covered in the health management system are at least the following:

- A short but adequate description of the health hazards of mercury.
- Personnel hygiene standards, such as personal protective equipment (PPE) use, clothing rules, smoking and eating in the workplace, etc.
- Processes for monitoring mercury in urine.
- Processes for monitoring mercury in air.
- Risk assessment and control processes.
- Health surveillance programmes.
- Actions to be taken if accidental exposure occurs.
- Information and training for employees.
- Record keeping.
- Internal audit processes.

All of these steps are described in this document.

The recommended monitoring programme is a key point which should comprise both personal air sampling and measurement of mercury in urine (biological monitoring - HgU). Biological measurement in urine compared to blood measurement is non invasive and reflects average exposure during the previous 3-4 months,

The aim of the urinary monitoring programme is to ensure that all individual HgU samples contain less than 50 µg Hg/g creatinine. As a consequence of this, the annual mean HgU of homogeneous groups is expected to be lower than 30 µg Hg/g creatinine, assuming a normal distribution in the data.

In general, testing should be more frequent in employees with higher potential exposure.

In order to be able to achieve the aim of individual HgU levels being less than 50 µg Hg/g creatinine, a testing frequency of at least 3-4 times a year is suggested for individuals with HgU levels above 20 µg Hg/g creatinine depending of the pattern of exposure. Stability of exposure can be assessed by frequent air analysis, preferably using personal monitoring techniques. When HgU levels are below 20 µg Hg/g creatinine, the frequency of urine sampling should be at least twice a year. This monitoring programme can be amended to meet national or local requirements.

The recommended work related action levels for individuals are:

Urinary mercury µg/g creatinine	**Frequency of Sampling per year**	**Management Action**
< 20	2	No action
20-30	≥ 4	No action
30-50	≥ 4	Review individual employee work practices
> 50	≥ 4	Remove from exposure to mercury, until below 30 µg/g creatinine

INTRODUCTION

This document provides guidelines to help manage and review work processes where the relation between mercury exposure and the health of workers is a relevant issue.

It is written so that each section in this document corresponds with chapters in Health 6, the self assessment audit document. This document is intended to act as the main reference document for Health 6.

Hence, this document reflects the best occupational health practices for work with mercury leading to a good health protection for workers in chlor-alkali plants using the mercury process.

To help the reader a list of abbreviations can be found in **appendix 6**.

1. ORGANISATION AND MANAGEMENT

Euro Chlor proposes a system for health management of mercury-related processes which is based on continuous improvement.

This system starts with a written health policy and management system, communicated to all employees, in which the work processes related to health management of mercury are described, and responsibilities and tasks are delegated to responsible functions within the organisation. Items which should be covered in the health management system are at least:

- A short but adequate description of the health hazards of mercury.
- Personnel hygiene standards, such as personal protective equipment (PPE) use, clothing rules, smoking and eating in the workplace, etc.
- Processes for monitoring mercury in urine.
- Processes for monitoring mercury in air.
- Risk assessment and control processes.
- Health surveillance programmes.
- Actions to be taken if accidental exposure occurs.
- Information and training for employees.
- Record keeping.
- Internal audit processes.

2. HEALTH HAZARDS OF MERCURY

2.1. TOXICOKINETICS OF MERCURY

Mercury is a heavy, silvery-white liquid with a high vapour pressure. Saturated air at 20°C and at 50°C contains 14 mg/m^3 and 125 mg/m^3 of mercury respectively. However, because of high ventilation rates in cell rooms it is unlikely that such levels will be achieved.

2.1.1. Absorption

Mercury can enter the body by three routes: inhalation, ingestion and through the skin. In the chlor-alkali industry the major route of exposure to mercury is by inhalation and this route will normally account for over 90% of all mercury absorbed into the body. Absorption through the skin occurs when the skin is exposed to mercury vapour and also when it is in direct contact with liquid metal (Hursch et al., 1989), although this route is likely to be insignificant in relation to inhalation for occupationally exposed individuals under normal hygiene conditions (see chapter 3).

2.1.2. Distribution

Following absorption, mercury is distributed widely in the body and is deposited in several organs, such as the brain, kidney and liver, where it will accumulate if there is repeated exposure. The kidney is the major organ of deposition, so mercury concentrations in the kidney are significantly higher than in other organs.

Following repeated exposure, mercury is retained in the body; the duration of retention varies from organ to organ, with the longest retention time occurring in the brain. Significant levels of mercury have been detected in the brain many years after exposure has ceased.

2.1.3. Excretion

Mercury is eliminated from the body via urine, faeces, sweat and expired air. Urine and faeces are the main routes of elimination. After short-term exposure, the faecal route predominates, and after long term exposure (approximately 40 days), urine is the major route (Clarkson et al., 1988).

After mercury is inhaled, the level of mercury in blood rises rapidly. With low-level, short-term exposure the maximum blood level is reached on the same day as exposure. It then decreases, with a first phase half-life of approximately 3 days, which accounts for about 90% of the absorbed mercury, and a second phase half-life of approximately 18 days (Hursch et al., 1976; Chevian et al., 1979).

There is no universally agreed biological half-life for mercury. Radioactive tracer studies in human volunteers have shown elimination of mercury from the body to follow a complicated pattern, with biological half-lives that differ for individual organs and also with time after the start of exposure. Studies on volunteers and workers have estimated the half-life for mercury, depending on the duration of the exposure, to be between 50 and 90 days (Barregard et al., 1992; Sallsten at al., 1993). In the kidneys the half-life of deposited mercury is 60-70 days, which means that total elimination time is approximately one year after exposure ceases.

2.2. TOXIC EFFECTS OF ELEMENTAL MERCURY

2.2.1. Acute Exposure

Acute intoxication will only result if there is accidental exposure to extremely high mercury concentrations (of the order of 1 to 3 mg/m^3). This situation is unlikely to happen if the Euro Chlor technical guidelines on plant management are followed.

Various organs and systems will be targeted, particularly the nervous and respiratory systems. Most symptoms develop immediately and usually completely resolve within a few days.

The clinical presentation of neurological and psychological symptoms and signs can be headache, fatigue, aching muscles, muscle twitching, abnormal nerve conduction, irritability, anxiety, mood swings, depression and aggressive behaviour.

Respiratory symptoms are prominent after short-term, high-level exposure. They include coughing, dyspnoea, tightness of the chest, shortness of breath, dryness of the throat and burning pain in the chest.

A slight, transient, proteinuria has also been reported after acute exposure to mercury (Bluhm et al., 1992; Adams et al., 1983).

Symptoms related to other organ systems can include rapidly resolving fever and weight loss. Short-term inhalation exposure to high concentrations of mercury resulted in cardiovascular effects (increased blood pressure and heart rate) and also gastrointestinal changes.

2.2.2. Chronic Exposure

2.2.2.1. The Nervous System

In cases of chronic mercury exposure the nervous system appears to be the most important target. Although there is a lack of consistency across a large number of studies, several subclinical neurological effects such as hand tremors, slowing of nerve conduction velocity, abnormal psychomotor-tests and adverse mood

changes have been reported. Consistent changes appear to occur in individuals with > 35 µg Hg/g creatinine in urine, although subtle behavioural effects may be detected between 20 to 30 µg Hg/g creatinine (SCOEL, 2007).

Where there has been exposure to high levels of mercury the clinical picture is characterised by loss of memory, insomnia, irritability, excessive shyness, hand tremor and emotional instability (erethism). This severe syndrome is not likely to occur with the current levels of exposure to mercury in the European chlor-alkali industry using a mercury process.

2.2.2.2. The Kidney

Mercury is toxic to the kidney. Reversible enzymuria, not associated with clinical disease or loss of function, has been reported at HgU levels in excess of 20 µg Hg/g creatinine (Ellingsen, 2000a; 2005). It is recognised that exposure to high levels (of the order of 0.5 mg/m^3) of mercury can lead to a nephrotic syndrome (WHO, 1991).

2.2.2.3. Other effects

Reproduction: It is known that mercury can easily cross the placenta and foetal blood-brain barrier. Menstrual disturbances, infertility and spontaneous abortion associated with mercury exposure have been reported, although the results of studies vary remarkably (Rowland et al., 1994). There is considerable evidence that mercury can also affect the development of the unborn child.

Cancer: There have been several mortality studies in chlor-alkali workers and, overall, they have not shown a statistically significant excess of cancer at any site in the body (Ellingsen et al., 1993; Boffetta et al., 1993). Mercury is not classified as a carcinogen (IARC 1993).

No excess mortality for other illnesses has been attributed to mercury (Ellingsen et al., 1993). When confounders for smoking are taken into account, no increased risk of mortality due to cardiovascular effect can be found in workers exposed to metallic mercury.

Allergic contact dermatitis has been reported as a rare phenomenon after mercury exposure (Kanerva et al., 1993).

For a more detailed description of health hazards of mercury, see Annex 1.

3. PERSONNEL HYGIENE STANDARDS

3.1. Introduction

It is of paramount importance to strive for continuous reduction in exposure to all chemicals in order to protect the health of workers in the chemical production and user industry. This particularly applies to mercury. Several hygiene principles are outlined below which can be used to significantly reduce exposure to mercury.

3.1.1. Principles of hygiene control

Regulations at both the EU and national level require consideration of a hierarchy of control measures to ensure that exposure to substances hazardous to health is adequately controlled. The levels of the hierarchy are listed in priority order and must be considered and applied in this order. Each level of the hierarchy should be applied sequentially in a way that is appropriate to the activity and consistent with the risk assessment. Most situations require several levels of the hierarchy to be used in order to adequately control the risk associated with exposure.

3.1.2. The hierarchy of hygiene control

- Eliminate the use of the hazardous substance wherever possible (e.g. by substitution with is a less dangerous substance).
- Take measures to enclose the process in order to reduce the potential for exposure to the hazardous substance wherever possible
- Control the exposure of the substance at the source using engineering control measures such as local exhaust ventilation (LEV);
- Provide adequate protective equipment (such as respiratory protective equipment – RPE).

This approach implies that personal protective equipment should be the last option to control exposure sufficiently below Occupational Exposure Levels (OELs).

3.2. Plant design/engineering controls/housekeeping

Details on this item are given in the ***Env Prot 11 - Code of Practice - Mercury Housekeeping***. This Code of Practice is based on the long term experience of the European chorine producers using mercury technology and gathers the best housekeeping (especially in the cell room) and maintenance practices (especially in the cell room) to minimise and avoid, if possible, mercury emissions in the environment (air, liquid effluents and soil). It proposes practical

short term and long term action programmes to guide companies in the organisation of their mercury “housekeeping”.

3.3. Clothing and Facilities

3.3.1. General

It must be emphasised that a very important pathway for exposure to mercury in the industry is via contaminated clothing, the presence of mercury in clothing appears to be very persistent. For this reason it is strongly advised that a complete set of work clothing (overall, sweater, shirt, trousers, underwear and socks) is provided for each operator.

The optimum frequency for changing overalls will depend on exposure levels:

- A minimum of twice a week for people working in areas where mercury can be present (e.g., the cell room, mercury warehouse, and mercury contaminated waste treatment unit).
- Every day, or more frequently, when work has been done with significantly elevated levels of mercury in the working environment air.

Disposable clothes can be used when working conditions are associated with a high mercury contamination risk, provided that the presence of other hazardous materials, eg caustic soda, has been taken into account. It is recommended that all protective clothing provided does not contain pockets, to prevent the unnecessary accumulation of mercury

To avoid spreading mercury from the industrial plant to domestic areas working clothes should never be taken home.

Clothing awaiting collection or being transferred to the laundry should be kept in a sealed bag or closed container clearly labelled as “mercury contaminated clothing”. Impervious outer clothing, usually made from PVC, may be required for some activities and must be maintained, worn and decontaminated correctly.

3.3.2. Changing and Eating Facilities

Important sources of incidental exposure to mercury are via contact with contaminating clothes, respirators and skin and through the smoking of mercury contaminated tobacco. As such, maximum effort should be put in minimizing these routes of exposure.

- Within the changing facility or locker room, there should be strict separation of clean and "dirty" areas so that outside clothes and footwear are kept in the clean area and work clothes and footwear in the "dirty" area. There should be no potential pathways for cross contamination.

- No eating, drinking or smoking should be allowed inside the workplace except within designated areas. No contaminated working clothes or plant footwear should be worn in the eating facilities.
- In particular, gloves should be stored or disposed of properly. Used gloves should not be kept in work clothes but should be stored properly.
- Changing and eating facilities should be kept clean and tidy.

3.3.3. Personal Hygiene

Employees should shower at the end of each work shift before leaving work, or more frequently if required. They must thoroughly wash their hands prior to eating, drinking or smoking.

3.3.4. Laundry

Clothing worn by personnel who are working in a Chlor-Alkali plant with the mercury process will be contaminated with mercury. Persons who handle such clothing have a potential for exposure.

- In order to avoid exposure of external personnel, it is highly recommended that laundering should be done at the work location, separated from laundering of other work clothes from the plant.
- If this is not feasible, the management of the external laundry should be informed about the health effects of mercury exposure, possible exposure routes, best practical working procedures and medical surveillance. A risk assessment for external laundering should be carried out.
- Mercury is very retentive in clothing. Oxidative bleach and acidic rinsing will assist in removal of mercury from clothing.
- Occasional measurements for mercury contamination of cleaned work clothes could be made to ensure that laundering has been effective.
- Waste mercury in washing water should be handled as contaminated mercury water and must be disposed of following national guidelines.

3.4. Personal protective equipment

3.4.1. Footwear

Suitable footwear must be provided. It should be made from material impervious to mercury and provide electrical insulation and resistance to corrosives. This footwear should be worn only in the plant and should not be taken home. Boot washes could be provided at specific locations to avoid contamination.

3.4.2. Gloves

Protective gloves should be provided for all operators. They should be made of material impervious to mercury and resistant to corrosives, e.g. PVC. They should be worn on all occasions when handling mercury, sampling or when there is a potential exposure to contaminated material. Gloves should be replaced on a regular basis, determined by the degree of contamination. This may need to be daily or even more frequently.

⇒ **Leather gloves should be prohibited from use in areas where mercury can be present (e.g., the cell room, mercury warehouse, and mercury contaminated waste treatment unit) because leather absorbs mercury. If leather gloves need to be used for specific tasks, they should be disposed of and destroyed after use, or at least every day.**

3.4.3. Respirators

Respirators should be worn when the Risk Assessment shows a risk of high exposure to atmospheric mercury.

Air monitoring should be performed using a mercury vapour indicator to permit a correct respirator selection. Be aware that air concentrations of mercury vapour will probably increase 10-20 times during clean-up due to the disturbance of liquid mercury, or due to contact with hot surfaces.

Storage of respirators should be done in a mercury-free environment. Respirators should be decontaminated properly after use. Different types of respiratory protection can be used depending on the airborne concentration of mercury vapour, as described in **Annex 2.** Additional information can be found in the ***GEST 92/171 – Personal protective equipment for use with chlorine***.

4. BIO-MONITORING OF MERCURY EXPOSURE

4.1. Recommendation and frequency

The measurement of mercury in urine is considered to be the best determinant of mercury body burden following long-term exposure. Urine sampling is a non invasive, easy and practical method to implement under occupational conditions. The measurement of mercury levels in blood can be useful in cases of short-term, higher-level exposures to mercury.

Mercury urinary figures reflect the exposure of the 3 or 4 previous months due to the relatively slow elimination of mercury from the human body. The aim of the recommended monitoring programme is for all individual HgU samples to be always below 50 µg/g creatinine. As a consequence, the annual mean of homogeneous groups should be lower than 30 µg Hg/g creatinine.

The frequency of testing will depend upon national recommendations and legislation, but testing should be more frequent in employees with higher exposure. A monitoring protocol should be established in consultation with the responsible occupational health professional. For individuals with HgU above 20 µg/g creatinine, testing frequency should be at least 4 times a year, depending on the pattern of exposure. The frequency of testing should be increased if the levels of mercury in urine increase. When levels are below 20 µg Hg/g creatinine, the testing frequency should mainly be determined by any changes in the working environment, with a minimum of 2 times a year (see also table section 7).

New employees who join a worker group where the mean HgU is greater than 30 µg/g creatinine should have their urine tested monthly for 6 months before the above regime is followed. Urinary mercury measurement should be performed prior to the start of work in which mercury exposure is likely.

N.B. Frequency of testing should be increased if it is known that the intensity of exposure will increase or where there has been unexpected exposure.

It is recommended that samples are taken at approximately the same time of the day. A sample taken before starting work or after showering at the end of a shift has the advantage that possible sample contamination is minimised.

It is strongly advised that HgU is measured in an exit sample when an exposed person is permanently leaving a job with an exposure risk.

4.2. Evaluation and reporting

Evaluation of urinary mercury results helps management to monitor and improve working conditions. It is important to identify the most highly exposed workers and the processes associated with highest exposure in order to improve the situation by applying preventive actions.

To improve the comparability of the results and achieve a better risk assessment, workers should be categorised into groups based on similar exposure (SEG). These groups are defined as representing workers performing the same tasks (e.g. electrical maintenance, production, etc.) and therefore exposed to similar levels of mercury. The tasks should be ranked depending on the magnitude of exposure. In cases of high individual results, the work practices of the individual worker should be reviewed carefully. New workers should be trained, but because of inexperience their exposure risk is usually higher than for other workers doing the same work and this needs to be considered by management when allocating tasks and comparing monitoring results. The same SEGs as defined for the monitoring of exposure in the environment should be used.

Mercury in urine levels should be checked for those individuals with infrequent exposure to mercury such as managers, secretaries and other workers who do

not often enter the cell room. These people are likely to be only lightly exposed and can be checked less frequently than others.

It is now well established that exposure is usually high and difficult to control when facilities which contained mercury are decommissioned. In general, unusual tasks may lead to higher exposure of workers. The Euro Chlor document ***Env. Prot. 3 – Decommissioning of Mercury Chlor-Alkali Plants*** is based on the experience of different chlorine producers in Europe and proposes a synthesis of the best applicable practices for health, safety and environmental protection during all stages from the shut down till the final disposal of materials for the decommissioning of a mercury technology based electrolysis unit.

To assess the hygiene management system and to facilitate continuous improvement of worker health protection the results for HgU and Hg concentrations in air should be summarised and discussed with plant management.

The mean levels of mercury in urine for each SEG should be reported each year. Groups with mean mercury in urine above 30 µg/g creatinine should be identified, and the atmospheric personal or static monitoring results and work practices for these groups should be reviewed. Sources of exposure for these groups should be identified - it may be necessary to obtain additional monitoring data to achieve this. Once identified, these sources of exposure should be eliminated or reduced.

It is useful to use the annual data to assess the general trend in performance on mercury hygiene issues at the plant level. One approach that can be used is to summarise the data collected for each year by identifying the number of workers whose highest individual HgU results fall into the following bands: ≥10, ≥ 20, ≥ 25, ≥ 30, ≥35, ≥ 50, ≥75, ≥100, ≥ 125 and above 150 µg/g creatinine. The data, when expressed in this way, can be used to establish the trend in performance when compared to summarised data from previous years.

It has also become practice for Euro Chlor to collect these data from member companies in order to establish trends in performance across its member companies. The data can also be used to identify areas in which Euro Chlor might be able to develop further guidance and advice to its members with the objective of further improving performance in mercury hygiene across the membership.

4.3. Quality Control

It is essential for the Hg and HgU testing laboratory to have high quality control procedures. The laboratory should participate in a national or international quality scheme in order to achieve and maintain reliability in the testing procedure.

4.4. Intra-Individual Variation

There is marked intra-individual variation in mercury in urine measurements and this is reflected in a reported coefficient of variation of 15-20% (Barregard et al., 1993; Cross et al., 1995). Thus, under stable exposure conditions the mercury in urine level fluctuates around an average value; values on two consecutive days often differ by 25% and can differ by up to 50%.

When considering the average mercury in urine level of a sufficient number of results for one employee, the variation of individual values tends to cancel each other out.

4.5. Diurnal Variation

There is a diurnal variation in the urinary excretion of mercury - excretion in the early hours of the morning exceeds that in mid afternoon. When measurements are made at the same time of the day the individual worker coefficient of variation in measurement can decrease by up to 14% (Piotrowski et al., 1975). Diurnal variation is less significant compared with other sources of variability (Calder et al., 1984), but it is nevertheless advised samples are taken at the same time of the day, whenever possible.

4.6. Correction for Dilution Effects

In the occupational setting it is not practicable to collect 24 hour or even 12 hour specimens of urine and measurements are made on spot samples. The level of mercury in urine is affected by dilution or concentration of the urine, as may occur with a high or low fluid intake respectively. To minimise this effect, mercury concentrations should be corrected for creatinine content of the urine and expressed as µg/g creatinine.

Nevertheless, experience has shown that this correction can only be considered as valid in a defined range of creatinine concentration in urine. The usually accepted range is 0.5 to 2.5 g creatinine per litre of urine; if the urine is too diluted or too concentrated, the mercury in urine measurement should be repeated the following working day; if this is not possible, the worker will temporarily be moved to a function without exposition to mercury.

4.7. Relationship between workplace and bio-monitoring levels

The relationship between measured air concentrations of mercury (Hg-air) and mercury content in urine and blood (HgU, HgB) is complex. There is a large reported variation in the ratios Hg-air/HgU and Hg-air/HgB. This is likely to be due to changes in the intensity of exposure over the days previous to

monitoring, but also by analytical factors the toxicokinetics of the chemical and other influencing factors like, health status, body mass, smoking habits, alcohol intake. In addition, comparable with other compounds, the uptake of mercury from the ambient air into the body depends on the workload, reflected in the respiratory rate. The use of a respirator will distort this relationship because air sampling is not usually performed inside the respirator when it is used.

The ratios between personal atmospheric monitoring and mercury in urine and blood (µg/m³:µg/g creatinine, or µg/m³ mg/l blood) used by SCOEL (2007) and as such used as a basis for the proposed IOEL are 1:1.4 (Urine) and 1:0.48 (Blood). However, in a small German (H.F. Bender et al. 2006) and a Norwegian (Nordhagen et al., 1994) studies within the chlor-alkali industry confirmed the significant variation between Hg-air and HgU.

4.8. Non-Occupational Levels of Mercury in Blood and Urine

In Europe, for individuals with no occupational exposure the level of mercury in urine is usually less than 5 µg/g creatinine. A significant amount of mercury can be ingested with food, especially fish, but this is mainly methyl mercury which is not excreted by the kidney. Thus, whilst the dietary intake of fish will have little effect on urinary levels of mercury, it will influence blood levels. In Europe it is accepted that mean blood mercury levels are usually less than 6 µg/l, although in studies where fish is eaten 4 times/week, levels of up to 44.4 µg/l have been reported (Cross et al., 1995). It is now possible to measure inorganic and organic mercury in blood separately.

5. MONITORING OF MERCURY IN THE WORKING ENVIRONMENT

5.1. Introduction

Exposure assessment is the process of estimating or measuring the intensity, frequency, and duration of exposure to an agent. Ideally, it describes the sources, pathways, routes, magnitude, duration and patterns of exposure, the characteristics of the population exposed, and the uncertainties in the assessment. Exposure assessment in the workplace is performed for mercury by determining the concentration of mercury in the urine of relevant workers.

5.2. Exposure Assessment - Workplace

There should be an agreed schedule of personal and area atmospheric mercury monitoring. The frequency and number of tests undertaken is determined by an exposure assessment based on a comprehensive survey for both routine plant

operation and maintenance activities. Additional testing will be required in the event of unplanned incidents.

Commonly accepted industrial hygiene policy recommends that:

- Exposure to mercury should be kept as low as reasonably practicable.
- In any case an exposure should not exceed the national legal occupational exposure limit.
- Where no regulation exists or when the regulations are less stringent than the company believes prudent, the company should take into account current EU recommended value by the Scientific Committee on Occupational Exposure Limit (SCOEL) as published recently in 2007. The SCOEL proposal is currently subject of a discussion for EU approval as indicative exposure limit value (IOELV).

5.2.1. Occupational Exposure Limits

8 hour TWA values for mercury and its inorganic divalent compounds (as Hg)

Source	8-hour TWA (µg/m^3)	Year	Hg electrolysis
EU-OEL (SCOEL)	20	2009	
Austria	50	2007	No
Belgium	20	2011	
Bulgaria	50	2004	No
Czech Republic	50	2007	
Denmark	25	2007	No
Finland	50	2007	
France	50	2008	
Germany	20	2011	
Greece	100	1999	
Hungary	80	?	
Italy	20	2009	
Netherlands	50	2007	No
Norway	20	2009	No
Poland	20	2009	
Portugal	25	?	No
Romania	50	2006	
Slovakia	100	?	
Slovenia	100	2001	No
Spain	25	2010	
Sweden	30	2005	
Switzerland	50	2009	
United Kingdom	25 - withdraw in 2005	2002	
Russian Federation	5	2009	
US / ACGIH2	25	1994	

Biological Limit Values/Biological Exposure Indices (BLVs/BEIs/BATs) for mercury

BLVs/BEIs/BATs based on levels of Hg in urine

Source	BLV/BEI	Comments	Year
EU-BLV (SCOEL) Proposal	BLV: 30 µg Hg/g creatinine	Total inorganic mercury in urine	2002
Germany	BAT: 25 µg Hg/g creatinine		
Hungary	BMGV: 50 µg Hg/g creatinine		2000
Italy	BMGV: 35 µg Hg/g creatinine		
Slovakia	BMH: 25 µg Hg/g creatinine		
Slovenia	BMV: 30 µg Hg/g creatinine	No Hg electrolysis units	
Spain	BMGV: 35 µg Hg/g creatinine	Value at end of work week	2001
Romania	BMGV: 35 µg Hg/g creatinine		
United Kingdom	BMGV: 35 µg Hg/g creatinine	Random sampling	2005
US / ACGIH2	BEI: 35 µg Hg/g creatinine	Total inorganic mercury in urine / sampled pre-shift	1993

BLVs/BEIs based on levels of Hg in blood.

Source	BLV/BEI	Comments	Year
EU-BLV (SCOEL) Proposal	BLV: 10 µgHg/l	Total inorganic mercury	2002
Germany	BAT: 25 µg Hg/l		
Italy	BMGV: 15 µg Hg/l		
Romania	BMGV: 10 µg Hg/l		
Sloakia	BMGV: 25 µg Hg/l		
Spain	BMGV: 15 µg Hg/l	Beginning of work day	2001
US / ACGIH2	BEI 15 µg/l	Total inorganic mercury in blood / sampled at end of shift at end of workweek	1993

Short Term Exposure Limit

There is no specific Short Term Exposure Limit (STEL) determined for mercury, except in a few countries. Nevertheless, excursions above the TLV, even where the TLV-TWA (Time Weighted Average) is 25 µg/m^3, should be controlled. Excursions in worker exposure levels may exceed 3 times the value of the 8h-TLV/TWA for no more than a total of 30 minutes during a workday, and under no

circumstances should they exceed 5 times this value, provided the 8h-TLV/TWA is not exceeded.

15-minute STEL values for mercury and its inorganic divalent compounds (as Hg)

Source	**15 minutes STEL (µg/m^3)**	Year
Austria	500	2003
Czech Republic	150	2007
Germany	800	2007
Hungary	320	2007
Italy	25	2009
Netherlands	500	2007
Romania	150	2006
Slovakia	800	?
Switzerland	400**	2007
Russian Federation	10	2009

** inhalable aerosol

5.2.2. Similar Exposure Groups

The preferred approach is to subdivide the exposed population into similar exposure groups (see also chapter 4.2 for SEG) with the respect to exposure. Where a group of workers is performing identical or similar tasks at the same place and has a similar exposure profile, monitoring the mercury exposure of any worker in the Group provides data useful for predicting the exposures of the remaining workers.

When it is difficult to establish a SEG, for example because “short and high” exposures occur without any regular pattern, it is better to establish two or more specific SEGs. For example, one SEG could cover the more routine activities of the worker shifts without the “short and high” irregular exposure pattern, and another SEG could cover the exposure from the specific task alone.

5.2.3. Qualitative assessment

On the basis of the information collected during an initial characterisation, workers believed to have a similar exposure profile are grouped in similar exposure groups. By characterisation of SEGs it is possible to establish a SEG priority ranking. A certified predictive tool, e.g. Estimation and Assessment of Substance Exposure (EASE developed by the British HSE) or others, or area measurements from the past can be used for this characterisation.

5.2.4. Quantitative assessment

5.2.4.1. Introduction

A competent person in industrial hygiene should define the environmental air monitoring programme. The goal of this programme is to establish the sampling plan (e.g. sampling frequency, location, and sampling duration), to select the

air monitoring equipment and methods, to evaluate data and analysis of the samples, and to verify whether:

- the 8-hour time-weighted average concentrations of mercury in the breathing zone exceed the specified limit;
- established control measures are functioning properly or if additional control measures are needed.

To compare an exposure level of mercury with the exposure limit it is necessary to know the concentration of mercury in the breathing zone extrapolated to the same reference period as that used for the limit value (i.e. 8 hours for mercury).

5.2.4.2. Judging the monitoring data

It is obvious that when the arithmetic mean of the measured concentrations is below the OEL, one cannot be sure that the OEL will always be complied with. On the other hand, a zero probability of exceeding the OEL is not realistically achievable because an exposure limit might be exceeded for a brief period. In the relevant European standard (i.e. EN 689: guidance for the assessment of exposure by inhalation to chemical agents) and in the strategy for assessing occupational exposures of the AIHA, several decision making and statistical tools are presented. They all stress that personal exposure to a substance needs to be controlled sufficiently below the corresponding OEL. In other words, the concentrations of mercury found in a series of measurements should be substantially below the OEL.

For most substances a threshold value of 5% probability of exceeding the OEL is commonly accepted in industrial hygiene practice, and is also the value described in Annex D of the European Standard EN 689.

But since mercury is a high hazard, chronically-acting substance the long-term mean over weeks or months is a better parameter than evaluation of the 5% probability of 8-hour work shifts. For chronically acting substances an occasional high exposure is not critical because it is the arithmetic mean that best summarises the total mass absorbed by a person.

The Land's "exact" procedure is a good statistical method to evaluate the mean mercury concentration. The Land's procedure calculates exact confidence limits for the true arithmetic mean of a log-normal distribution. With this method the one-sided 95% Upper Confidence Limit (UCL1, 95%) of the arithmetic mean is calculated. If the calculated UCL1, 95% is below the OEL, there is at least 95% confidence that the arithmetic mean exposure is less than the OEL (See American Industrial Hygiene Association (AIHA) - Assessing and Managing Occupational Exposure).

Statistical tools such as Altrex (INRS, France), Hygenist (http://www.tsac.nl/) can be applied to determine the statistical parameters.

Details of monitoring objectives, sampling and analytical methods and equipment, as well as the decision tree? to define the frequency and number of periodic measurements are described in the guideline ***Analytical 6 – Determination of Mercury in Gases*** and in **Annex 3** "Monitoring of mercury in workplace– details" and in "ISO 17733 (Workplace air - Determination of mercury and inorganic mercury compounds - Method by cold-vapour atomic absorption spectrometry or atomic fluorescence spectrometry)"

6. RISK ASSESSMENT

A risk assessment is a formal process of quantifying the probability of a harmful effect to workers from, in this case, metallic mercury. .

An effective health risk assessment of work processes related to mercury requires the involvement of trained employees and line-management, supported by HSE professionals or Occupational Health Advisers. A complete risk assessment process should include the following steps:

- Identification (section 4) of the health hazards of mercury
- Assessment of the health risks of mercury (chapters 4, 6 and 7). This means monitoring in order to quantify exposure to mercury and comparison of these monitoring data with the applicable occupational exposure limits.
- Women of Reproductive Capacity: In a risk assessment special attention should be given to susceptible groups, such as women of reproductive age who work with mercury. Mercury can easily pass the placental membrane and the blood-brain barrier. Women of reproductive age, working with mercury, should be made aware of this potential hazard and if willing to become pregnant should be advised to consult an occupational physician to discuss potential measures to be taken in her work situation, to exclude possible damage to the unborn child. For women who are pregnant, have recently given birth or are breastfeeding, EC Directive 92/85 requires a risk assessment of working conditions. As a consequence, the employer must reduce the exposure of pregnant and breastfeeding women to mercury to prevent harm to their unborn or breastfeeding child.
- Health surveillance (section 8)
- Record keeping (section 11): Exposure and health surveillance records provide feedback on health trends, and help to identify problem areas for action. They also help to fulfil legal requirements, and provide documentation in case of any compensation. As a rule, exposure and health surveillance records should be kept for several years, because of the long delay between some types of exposure and effects.
- Review of the findings should be performed regularly and recorded properly in a written report.

7. RISK MANAGEMENT

After assessment of the risks (section 5 and 8) a written action plan should be made to define clearly which health risks should be eliminated. Priorities and a time schedule should be provided for the actions.

Additionally, an explanation should be given for situations in which it is not possible, for technical or economic reasons, to comply with internal or external exposure limits (BEI or OEL).

The programme to be implemented should be documented, archived, and communicated to all whom it concerns. It should be clearly listed whether, where and why exposure to mercury cannot be limited without use of personal protective equipment.

Euro Chlor recommends the following action levels for individuals:

Urinary mercury µg/g creatinine	**Frequency of Sampling per year**	**Management Action**
< 20	2	No action
20-30	≥ 4	No action
30-50	≥ 4	Review individual employee work practices
> 50	≥ 4	Remove from exposure to mercury, until below 30 µg/g creatinine

8. HEALTH SURVEILLANCE

The health surveillance programme is meant to detect any adverse effects of exposure as early as possible. It should be applicable for employees, including employees of contractor companies, who have the potential for exposure to mercury. The health surveillance programme should be under the overall direction of a (occupational) physician. ***Analytical 11 – Determination of Mercury and Creatinine in Urine*** is the Euro Chlor guideline for the analyses that need to be performed.

In this section the minimum requirements for the programme are listed. The detailed nature of the examinations is left to the professional judgment of the physician. To be able to design an appropriate programme, the physician should take into account the job requirements as well as the health effects of mercury. The programme should at least consist of a pre-placement examination, a

periodical examination, an exit examination and a description of what has to be done in case of accidental exposure.

The minimum requirements of physical examinations related to potential mercury exposure are the following:

Pre-placement examination:

- Work and personal medical history, especially for disorders that potentially could be related to mercury exposure such as renal, neurological or psychiatric diseases.
- Urine analysis (blood, protein, mercury baseline)

Periodic and exit examination

- update of medical history as described before
- relevant examination of functions based upon history, e.g. neurological examination
- urine analysis (blood, protein, mercury)

Determination of the frequency of periodical biological monitoring has to be based on the risk assessment of similar exposed groups (see section 4).

The results of the mercury health surveillance programme (made anonymous) could be used for information and training to management and workers.

An example checklist that can be used for health surveillance is presented in **Annex 4**.

9. HEALTH-RELATED ACTIONS IN CASE OF OVER-EXPOSURE

9.1. Introduction

In this chapter over-exposure means extraordinary, mostly accidental, short and high exposure. Mostly, in such cases, no direct measurements of mercury in air have been performed. Nevertheless, based on the circumstances under which such incidents have been taken place, it is evident that there is at least a very strong suspicion that there has been a very high exposure.

9.2. Actions to be taken:

First Aid:

Immediate decontamination should be taken place.

- First-aiders should avoid direct contact and chemical protective clothing and breathing protection should be worn, if necessary.

- Contaminated clothing, shoes and leather goods (e.g. watchbands, belts) should be removed
- Quickly and gently excess chemical should be blotted or brushed away.
- After that the skin should be washed gently and thoroughly with water and non-abrasive soap for 5 minutes or until the chemical is removed.
- Medical attention should be called immediately.
- Contaminated clothing, shoes and leather goods should be discarded.

9.3. Medical surveillance after the incident.

- Complete medical assessment including clinical examination to seek clinical signs of mercury intoxication (tremor, neuro-psychochological disorders, stomatitis, gastrointestinal symptoms, cutaneous lesions, blood pressure).
- Complete assessment of renal function, including the measurement of biological parameters of renal function (urea, creatinine, N-acetyl-ß-glucosaminidase (NAG), ß2-microglobulin, albumin).
- The assessment of lung function.
- The determination of blood mercury levels.
- An electrocardiogram and if necessary, electromyography, visual test and electroencephalogram.
- In case of severe symptoms of mercury intoxication, treatment with chelating agents DMPS (2,3-dimercapto-1-propanesulfonate) or DMSA (meso-2,3-dimercaptosuccinic acid) effective in reducing kidney mercury concentrations, by accelerating the elimination of mercury, could be considered.
- Follow-up of the victim until the symptoms are disappeared and/or the mercury in urine levels have returned to acceptable levels, should take place on a regular basis.

10. INFORMATION AND TRAINING FOR EMPLOYEES

European directive 89/391 CEE requires employers to provide appropriate information and training to all employees, including workers from outside undertakings, potentially exposed to a risk to their health and safety.

A well-defined training programme should be established by employers and provided, before initial assignment and at least annually, to workers who potentially may be exposed to mercury in order to protect them and their co-workers as well as to enable them to perform their work in a competent, safe, and environmentally sound manner.

This programme should consider the following topics:

- Understanding of mercury toxicity.
- Adequate working practice including protective clothing, personal hygiene and emergency procedures.
- Housekeeping practices and decontamination procedures including safe use of mercury and storage practice.
- Exposure limit values, exposure control management and employee exposure measurements (ambient and personal monitoring) together with availability of written procedures and hazard information.
- Medical surveillance program and biological monitoring.
- Control of knowledge and regular refresher training; feedback from audit.

All training should be documented and the contents regularly updated.

OSHA directive CPL 02-02.006: "Inorganic Mercury and its Compounds" provides training recommendations.

Euro Chlor prepared and distributed largely a poster on the basic "do's and don'ts" to reduce exposure to mercury in a cell room (***Health 8 – Mercury Do's and Don'ts Poster***); this document is available in several languages.

11. RECORDS

Records should be kept in order for the following reasons:

- They are necessary for proper medical surveillance
- They could be used for future medical research
- They could be useful in case of any claim for compensation

Moreover, systematic use of the recorded data is fundamental for the process of continuous improvement in the control of exposure to mercury.

Directors, engineers and physicians are responsible for organising their records and for their preservation. Records should be kept in compliance with national and local regulations and must be carefully protected. They could be archived on paper or electronically.

Records should be kept for several decades. Generally speaking, it is necessary to organize such records in a way that an outside body could understand how the processes were managed. This is of fundamental importance for mercury to ensure that knowledge of hazards, hygiene, bio-monitoring, risk assessment is not slowly forgotten when all European mercury electrolyses have been decommissioned.

A detailed list of records is presented in **Annex 5**.

11.1. Medical records

Occupational health records should be comprehensive and available for occupational health personnel. During and after employment they should be stored according to general practice and national law. These records are of crucial importance in cases of claims for compensation. Hence they should at least be present and readily available during site employment and post employment (in accordance with national legislation).

11.2. Records of exposure

- Results from mercury in air monitoring should be attached to relevant SEGs and stored in the administrative HSE records, preferably for "eternity" or according to company policy and/or national law. Through the SEGs the individual members should have these results stored in their individual occupational health records.
- Results from the exposure and biological sampling monitoring programme should be stored in the individual health records of the persons monitored. Statistics from these programmes should also be stored in the administrative HSE records connected to the relevant SEGs under the conditions of storage mentioned above.
- Records of accidental exposures should be kept according to 11.1 and 11.2.

11.3. Training Records

Participation of workers in "formal" external or internal training programmes related to both HSE and for their specific jobs should be recorded in their personnel file, available for inspection by occupational health personnel.

12. INTERNAL AUDIT

Management of the health of employees with regard to mercury exposure, as described in this document, should be monitored by at least an internal audit system. A questionnaire which can be used for this is contained in the Euro Chlor document ***Health 6***.

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Appendix 1 - Health Hazards: detailed evaluation

1. TOXICOKINETICS

1.1. Absorption

Inhalation is the primary route of entry into the body for elemental mercury. Dermal penetration is usually not a significant route of exposure to inorganic mercury.

Approximately 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, possibly because of its enterogastric conversion to divalent mercury and subsequent binding to sulfhydryl groups. Dermal absorption of elemental mercury is limited. It is estimated that dermal absorption contributes approximately 2.6% of the absorbed mercury following exposure to elemental mercury vapour in the air; the other 97.4% of absorbed mercury occurs through inhalation.

The absorption, blood levels, and excretion of mercury were evaluated in nine healthy volunteers (two males, seven females) exposed to mercury vapour in air at a concentration of 400 $\mu g/m^3$ for 15 min. This exposure corresponded to a dose of 5.5 nmol mercury/kg body weight. Samples of exhaled air, blood, and urine were collected for 30 days after exposure. The median retention of elemental mercury after 30 days was 69% of the inhaled dose. This corresponds to the estimated half-life of approximately 60 days for elemental mercury.

1.2. Distribution

The lipophilic nature of elemental mercury results in its distribution throughout the body. Elemental mercury dissolves in the blood upon inhalation, and some remains unchanged. Elemental mercury in the blood is oxidized to its divalent form in the red blood cells. The divalent cation exists as a diffusible or non-diffusible form. The non-diffusible form exists as mercuric ions that bind to protein and are held in high-molecular-weight complexes, existing in equilibrium with the diffusible form. In the plasma, the mercuric ion is predominantly non-diffusible and binds to albumin and globulins.

The high lipophilicity of elemental mercury in solution in the body allows it readily to cross the blood–brain and placental barriers. In mice, the uptake of mercury across the placenta appears to increase as gestation progresses. Levels of mercury in the foetus of the mouse are approximately 4 times higher after exposure to elemental mercury vapour than after mercuric chloride administration, and are 10–40 times higher for rats. The transport of mercuric ion is limited at the placental barrier by the presence of high-affinity binding sites.

Mercury distributes to all tissues and reaches peak levels within 24 h, except in the brain, where peak levels are achieved within 23 days. The longest retention of mercury after inhalation of mercury vapour occurs in the brain. Japanese workers who died 10 years after their last exposure to elemental mercury vapours still had high residual levels of mercury in their brains.

While the primary organs of mercury deposition following inhalation exposure to elemental mercury vapours are the brain and kidney, the extent of deposition is dependent upon the duration of exposure and, to a greater extent, the concentration to which the organism is exposed.

1.3. Metabolism

The available evidence indicates that the metabolism of all forms of inorganic mercury is similar for humans and laboratory mammals. Once absorbed, elemental and inorganic mercury enter an oxidation–reduction cycle. Elemental mercury is oxidized to the divalent inorganic cation in the red blood cells and lungs. Evidence from animal studies suggests the liver as an additional site of oxidation. Absorbed divalent cation from exposure to mercuric mercury compounds can, in turn, be reduced to the metallic or monovalent form and released as exhaled elemental mercury vapour.

Once inhaled into the lungs, elemental mercury vapours rapidly enter the bloodstream. The dissolved vapour can undergo rapid oxidation, primarily in the red blood cells, to its inorganic divalent form by the hydrogen peroxide–catalase pathway. It is believed that the rate of oxidation is dependent on (1) concentration of catalase in the tissue; (2) endogenous production of hydrogen peroxide; and (3) availability of mercury vapour at the oxidation site.

The oxidation of elemental mercury may also occur in the brain, liver (adult and foetal), lungs, and probably all other tissues to some degree. In the brain, unoxidized elemental mercury can be oxidized and become trapped in the brain, because it is more difficult for the divalent form to exit the brain via the blood–brain barrier. Autoradiographic studies suggest that mercury oxidation also occurs in the placenta and foetus, although the extent of oxidation is not known.

1.4. Elimination and excretion

Elimination of mercury occurs primarily through the urine and faeces, with the expired air, sweat, and saliva contributing to a much lesser extent.

The urine and faeces are the main excretory pathways of elemental and inorganic mercury compounds in humans, with an absorbed dose half-life of approximately 1–2 months. After a short-term high-level mercury exposure in humans, urinary excretion accounts for 13% of the total body burden. After long-term exposure, urinary excretion increases to 58%. Exhalation through the lungs and secretion in saliva, bile, and sweat may also contribute a small portion to the excretion process. Humans inhaling mercury vapour for less than an hour expired approximately 7% of the retained dose of mercury. Inorganic mercury is also excreted in breast milk. The overall rate of elimination of inorganic mercury from the body is the same as the rate of elimination from the kidney, where most of the body burden is localized.

Elimination from the blood and the brain is thought to be a biphasic process, with an initial rapid phase in which the decline in the body burden is associated with high levels of mercury being cleared from tissues, followed by a slower phase with mercury clearance from the same tissues. An even longer terminal elimination phase is also possible because of accumulation or persistence of mercury, primarily in the brain.

In a study of former chlor-alkali workers exposed to elemental mercury vapour for 2–18 years (median 5 years), Sallsten et al. (1993) found that the elimination of mercury in

urine was well characterized by a one-compartment model, with an estimated half-life of 55 days.

Age is a factor in the elimination of mercury in rats following inorganic mercury exposure, with younger rats demonstrating significantly higher retention than older rats. This age-dependent difference in the rate of mercury excretion may reflect differences in the sites of mercury deposition (i.e., hair, red blood cells, skin).

2. BIOMARKERS OF EXPOSURE

Urine samples are considered to be the best determinant of mercury body burden from long-term exposure to elemental and inorganic mercury. Blood samples are useful primarily in cases of short-term, higher-level exposures to these forms, but are not as reliable an indicator of total body burden in longer-term exposures. Most analytical methods do not differentiate between inorganic and organic mercury, so total mercury concentrations in blood reflect the body burden of total mercury. Inorganic forms of mercury are not excreted to any significant extent in scalp hair, making hair an inappropriate biomarker of inorganic mercury exposure.

Occupational studies show that recent mercury exposure is reflected in blood and urine. However, at low exposure levels (<0.05 mg mercury/m^3), correlation with blood or urine mercury levels is low. Blood levels of mercury peak sharply during and soon after short-term exposures, indicating that measurements of blood mercury levels should be made soon after exposure. The half-life of mercury in the blood is only 3 days, attesting to the importance of taking blood samples as soon after exposure as possible. In the case of low-level long-term exposure, urine samples provide the best indicator of body burden.

Urinary mercury measurement is reliable and simple and provides rapid identification of individuals with elevated mercury levels. It is a more appropriate marker of exposure to inorganic mercury, since organic mercury represents only a small fraction of urinary mercury. Urinary mercury levels correlate better than blood inorganic mercury concentrations with exposure following long-term, low-level occupational exposure to elemental mercury vapour. There may be marked diurnal variation in the urinary concentration of mercury.

Based on a systematic review of high-quality studies, the International Commission on Occupational Health and the International Union of Pure and Applied Chemistry Commission on Toxicology estimated that a mean value of 2 µg/l was the background blood level of mercury in people who do not eat fish. These levels are "background" in the sense that they represent the average levels in blood in the general population and are not associated with a particular source of mercury exposure. However, the intra- and inter-individual differences in these biomarkers are substantial, possibly due to dental amalgam (urine) and ingestion of contaminated fish (blood).

Several studies have reported a correlation between airborne mercury and mercury in blood and urine; however, results vary, and it is not known whether the ratio between concentrations in urine (HgU) and blood is constant at different exposure levels. Limiting the analysis to studies in which the exposure had been assessed using personal breathing zone mercury measurements, it was estimated that in continuous 8 h/day occupational exposure, an airborne mercury concentration of 1 mg/m^3 leads to an average urinary mercury concentration of 1.4 mg (7 µmol)/litre (variation between

individual studies: 0.7–2.3 mg [3.5–11.5 µmol]/litre; seven studies) and to an average blood mercury concentration of 0.48 mg (2.4 µmol)/litre (0.17–0.81 mg [0.85–4.0 µmol]/litre; six studies) (Cross et al., 1995). However, in a short study run by the German chlorine industry in 2004, using data from 6 sites, the ratio between personal air measurements and urine values varied from 0.6 to 4.2, with variable correlations within the same plant. Considerable variability was observed in Hg air/HgU ratios within workers in the same plant. If the average individual results are used as a single measurement, no correlation between the HgU and Hg Air results can be demonstrated in data from this study (Euro Chlor 2007).

A specific study on chlor-alkali workers in Norway also showed wide variations in the correlation of mercury values in air and urine depending on the job type: maintenance workers had the lowest correlation (r=0.24) whereas the correlation for cell room operators was more predictable (r=0.70) (Norhagen et al., 1994). This demonstrates the difficulty in establishing straightforward correlations between urine and air levels of mercury.

Relationships between urinary and blood mercury concentrations with signs and symptoms of exposure are less clear. Exposure to elemental or other inorganic forms of mercury can be verified by examining the urinary mercury concentration. Urinary mercury concentrations normally expected in an asymptomatic population would be <10 µg/l. Background levels of urinary mercury, adjusted for creatinine, in an unexposed population are generally expected to be 5 µg mercury/g creatinine.

3. EFFECTS ON HUMANS

3.1. Symptoms and signs in acute intoxications

Mercury will cause severe disruption of any tissue with which it comes into contact at a sufficient concentration, but the two main effects of mercury poisoning are neurological and renal disturbances. In general, the ingestion of acute toxic doses of any form of mercury will result in the same terminal signs and symptoms, namely shock, cardiovascular collapse, acute renal failure and severe gastrointestinal damage. Acute oral poisoning results primarily in haemorrhagic gastritis and colitis; the ultimate damage is to the kidney. Clinical symptoms of acute intoxication include pharyngitis, dysphagia, abdominal pain, nausea and vomiting, bloody diarrhoea and shock. Later, swelling of the salivary glands, stomatitis, loosening of the teeth, nephritis, anuria and hepatitis occur. Exposure for a few hours to 1-3 mg/m^3 may give rise to pulmonary irritation and destruction of lung tissue and occasionally to central nervous system disorders.

3.2. Neurotoxicity

The central nervous system is probably the most sensitive target for elemental mercury vapour exposure. Similar effects are seen after all durations of exposure; however, the symptoms may intensify and become irreversible as exposure duration or concentration increase. A wide variety of cognitive, personality, sensory, and motor disturbances have been reported. 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,

electromyographic abnormalities), headaches, polyneuropathy (paraesthesia, stocking-glove sensory loss, hyperactive tendon reflexes, slowed sensory and motor nerve conduction velocities), and performance deficits in tests of cognitive function. Some long-term exposures to elemental mercury vapour have resulted in unsteady walking, poor concentration, tremulous speech, blurred vision, performance decrements in psychomotor skills (e.g., finger tapping, reduced hand–eye coordination), decreased nerve conduction, and other signs of neurotoxicity. Recent studies using sensitive tests for psychomotor skills, tremor, and peripheral nerve function suggest that adverse effects may be associated with very low exposures. A recent study of 75 formerly exposed workers examined using an extensive neuropsychological test battery found that deficits in motor function, attention, and possibly the visual system may persist for years after termination of occupational exposure, but previous exposure did not appear to affect the workers' general intellectual level or ability to reason logically.

3.2.1. Occupational exposure

Several studies have reported significant effects on tremor, cognitive skills or other central nervous system effects among groups exposed occupationally to similar or slightly higher levels. Tremor, abnormal Romberg test, dysdiadochokinesis, and difficulty with heel-to-toe gait were observed in thermometer plant workers subjected to mean personal breathing zone air concentrations of 0.076 mg/m^3 (range of 0.026–0.27 mg/m^3) (Ehrenberg et al., 1991).

In a cross-sectional study of 36 workers with no less than 10 years of exposure (average 16.9 years) to mercury vapour in a chlor-alkali plant, disturbances in tests on verbal intelligence and memory were more frequent among the exposed group members having blood mercury levels above 75 nmol/l and urinary mercury above 280 nmol/litre (the median values for the exposed group) (Piikivi et al., 1984). Using the relationship developed above, these would correspond to an air mercury concentration of 31–40 µg/m^3.

In another study of 41 male chlor-alkali workers (Piikivi & Tolonen, 1989), electroencephalograms (EEGs) were compared with those of 41 age-matched referents from mechanical wood processing plants. The exposure was assessed as the time weighted average blood mercury concentrations (59 [standard deviation (SD) 12.6] nmol/l), based on an average of 22 (SD 5.7) measurements during an average 15.6 (SD 8.9) years of exposure. Using the relationship mentioned in section 4.7 of the main document, this would correspond to an air mercury concentration of 25 µg/m^3. The exposed workers had significantly slower and more attenuated EEGs than the referents; this difference was most prominent in the occipital region.

In a study (Piikivi & Hänninen, 1989) in which the population studied largely overlapped with that in the study of Piikivi & Tolonen (1989), subjective symptoms and psychological performance of 60 male workers in a chlor-alkali facility were compared with those among 60 age-matched referents from the mechanical wood industry. The average length of exposure was 14 years, and all test subjects had been exposed for at least 5 years. The time weighted average blood mercury concentration among the mercury-exposed group averaged 51.3 (SD 15.6) nmol/l, with a range of 24.7–90 nmol/l. While no exposure-related perceptual motor, memory, or learning ability disturbances were observed, the exposed workers reported an increase in memory disturbances, sleep disorders, anger, fatigue, and confusion, compared with the controls. The authors considered that the three-shift work of the mercury-exposed workers was a possible

cofactor behind the increased symptoms, with the exception of memory disturbances. Using the relationship mentioned in section 4.7. of the main document, the average blood mercury concentration in the mercury-exposed group would correspond to an air mercury concentration of 25 μg/m^3.

Arm-hand steadiness showed a decline of borderline statistical significance among 43 workers exposed (exposure duration 5.3 [SD 3.9] years) to mercury vapour, compared with non-exposed referents (Roels et al., 1982), in the lowest exposure group, whose blood mercury concentration at the time of the study was 10–20 μg/litre; using the relationship mentioned in section 4.7. of the main document, this blood mercury concentration would correspond to an air mercury concentration of 21–42 μg/m^3.

Fawer et al. (1983) measured hand tremors in 26 male workers exposed to elemental mercury in 3 different industries. The mean duration of exposure was 15.3 years. At the time of the study, the average urinary mercury concentration was 11.3 μmol Hg/mol creatinine (20 μg/g creatinine). The mean mercury level (time weighted average) measured using personal air monitors was 0.026 mg/m3 (three subjects were exposed to >0.05 mg/m3). As referents 25 control males working in the same facilities, but not exposed to mercury, were chosen. Hand tremors were measured in the subjects using an accelerometer both at rest and while holding 1250 g. The highest peak frequency of the acceleration (i.e., the frequency corresponding to the highest acceleration) was greater in exposed men than in controls ($P < 0.001$) and was significantly related to duration of exposure and age but not to the urine and blood Hg. Smoking was not accounted for and might have confounded the study.

In a further study in Belgium, Roels et al. (1985) compared subjective symptoms and psychometric test results of 131 male workers exposed to mercury for an average of 4.8 years and 54 females in different industries with those from sex-, age-, weight-, and height-matched referents. Subjective symptoms were more prevalent among the mercury-exposed workers, but were not related to level or duration of exposure, and were considered to be exposure-related by the author. Of the large number of psychometric test results, only hand tremor was related to mercury exposure, and in males only. The average blood mercury concentration at the time of the study was 14 μg/litre (95th percentile, 37 μg/litre) in males and 9 μg/litre (95th percentile, 14 μg/litre) in females. Using the relationship developed in section 4.7. of the main document, the average blood mercury concentration in males and females would correspond to air mercury concentrations of 29 and 19 μg/m^3, respectively.

Abnormal nerve conduction velocities have also been observed in workers from a chloralkali plant with a mean urinary mercury concentration of 450 μg/litre (Levine et al., 1982). These workers also experienced weakness, paraesthesia, and muscle cramps. Prolongation of brainstem auditory-evoked potentials was observed in workers with urinary mercury levels of 325 μg/g creatinine (Discalzi et al., 1993), and prolonged somatosensory-evoked potentials were found in 28 subjects exposed to 20–96 μg mercury/m^3 (Langauer-Lewowicka & Kazibutowska, 1989).

Another investigation studied possible neuropsychological effects among former chloralkali workers with past exposure to mercury vapour. Seventy-five formerly exposed workers who had been examined with an extensive neuropsychological test battery were compared with 52 referents frequency-matched for age. The tests measured general cognitive function, motor and psychomotor function, attention, memory, and learning. The groups were similar in educational level, age, and verbal comprehension. The mean exposure time to mercury vapour in the index group was 7.9

(range 1.1-36.2) years with an annual mean urinary mercury concentration of 539 (range 41-2921) nmol/l. The mean time since the cessation of exposure was 12.7 (range 1.0-35.0) years. Performance on the grooved pegboard and the Benton visual retention test was poorer among the formerly exposed workers when compared with the referents. In addition the subjects who had experienced the highest intensity of exposure (cumulative urinary mercury index higher or equal to 550 nmol/l/year) had a poorer performance on the trailmaking test, part A and B, on the digit symbol test, and on the word pairs test (retention errors). The results suggest a slight persistent effect of mercury vapour exposure on the central nervous system, mainly involving motor functions and attention, but also possibly related to the visual system. Previous exposure does not seem to have affected the workers' general intellectual level or their ability to reason logically (Mathiesen et al., 1999).

Neuropsychological effects were examined in 47 mercury vapour exposed male chlor-alkali workers with current low concentrations of urinary mercury (mean HgU 5.9 nmol/mmol creatinine (Cr). Their average duration of exposure was 13.3 years, and the calculated mean concentration of U-Hg was 9.0 nmol Hg/mmol Cr per year (exposure intensity) during their time of exposure. They were compared with 47 age-matched male referents in a cross-sectional study. The two groups were not statistically significantly different with respect to neuropsychological test performance or number of self-reported subjective symptoms. The test results of the Static Steadiness Test, which assesses tremor, were not associated with exposure to mercury vapour. However current smokers had more hand tremor than non-smokers. Statistically significant associations were found between indices of current exposure (the concentration of inorganic mercury in whole blood) and the results of the WAIS Digit Symbol Test and the Benton Visual Retention Test (number of correct responses). This could indicate a small effect of current exposure on visuomotor/psychomotor speed and attention, and immediate visual memory. Whether the association found between the historical exposure intensity and the Digit Symbol Test results may represent long-term consequences of exposure cannot be determined in this study (Ellingsen et al., 2001).

Ngim et al. (1992) reported that dentists with an average of 5.5 years of exposure to low levels of elemental mercury demonstrated impaired performance on several neurobehavioural tests. Exposure levels measured at the time of the study ranged from 0.0007 to 0.042 mg/m^3, with an average of 0.014 mg/m^3. Mean blood mercury levels among the dentists ranged from 0.6 to 57 µg/litre, with a geometric mean of 9.8 µg/litre. The performance of the dentists on finger tapping (motor speed measure), trail making (visual scanning measure), digit symbol (measure of visuomotor coordination and concentration), digit span, logical memory delayed recall (measure of visual memory), and Bender-Gestalt time (measure of visuomotor coordination) was significantly poorer than that of controls. The exposed dentists also showed higher aggression than did controls. Furthermore, within the group of exposed dentists, significant differences were reported to have been observed between a subgroup with high mercury exposure compared with a subgroup with lower exposure. These exposure severity subgroups were not compared with controls, and average exposure levels for the subgroups were not reported. Using the relationship developed in section 4.7. of the main document, the geometric mean blood mercury concentration of 9.8 µg/litre would correspond to an air mercury concentration of 20 µg/m^3.

Echeverria et al. (1995) evaluated the behavioural effects of low-level exposure to mercury among dentists. The exposed group was defined as those dentists with urinary mercury levels greater than 19 µg/litre; those with lower urinary mercury

concentrations comprised the unexposed group. Exposure thresholds for health effects associated with elemental mercury exposure were examined by comparing behavioural test scores of 19 exposed dentists (17 males, 2 females) with those of 20 unexposed dentists (14 males, 6 females). The mean urinary mercury concentration was 36.4 µg/litre for exposed dentists and below the level of detection for unexposed dentists in this study. Significant urinary mercury dose–effects were found for poor mental concentration, emotional lability, somatosensory irritation, and mood scores (tension, fatigue, confusion). Using the relationship developed in section 4.7. of the main document, the mean urinary mercury concentration of 36.4 µg/l would correspond to an air mercury concentration of 26 µg/m^3.

More recently, Meyer-Baron et al. (2002) performed a meta-analysis of published studies on neurobehavioural functions in occupationally exposed individuals. Out of a total of 44 studies, 12 studies were included in the analysis. The results obtained were based on a total of 686 exposed and 579 control subjects. There was evidence of a dose-response relationship of effect sizes. Nine significant performance effects were noted at mean urinary concentrations below 35 µg/g creatinine. Most of these appeared at urinary excretions between 22 and 29 µg/g creatinine, two effects appeared at 18 and 19 µg/g creatinine and two appeared at 34 µg/g creatinine. The authors concluded that their meta-analysis provided notable evidence for neurobehavioural impairments due to occupational mercury exposure at low concentrations.

3.2.2. Exposure from dental amalgam

Although several studies have demonstrated that some mercury from amalgam fillings is absorbed, no relationship has been observed between mercury release from amalgam fillings and mercury concentration in the brain.

In a cross-sectional study, Saxe et al. (1995) reported that cognitive function among 129 Catholic nuns, 75–102 years of age, was not related to the number or surface area of occlusal dental amalgams.

Bagedahl-Strindlund et al. (1997) evaluated Swedish patients with illnesses thought to be causally related to mercury release during dental restorations and mapped the psychological/psychiatric, odontological, and medical aspects of 67 such patients and 64 controls through questionnaires and a limited psychiatric interview. The most striking result was the high prevalence of psychiatric disorders (predominantly somatoform disorders) in the patients (89%) compared with the controls (6%). The personality traits differentiating the patients were somatic anxiety, muscular tension, psychasthenia, and low socialization. More patients than controls showed alexithymic traits. The prevalence of diagnosed somatic diseases was higher, but not sufficiently so to explain the large difference in perceived health. The multiple symptoms and signs of distress displayed by the patients could not be explained either by the odontological data or by the medical examination. The number of amalgam-filled surfaces did not differ significantly between patients and controls; 19% of the patients lacked amalgam fillings.

Malt et al. (1997) evaluated the physical and mental symptomatology of 99 self-referred adult patients complaining of multiple somatic and mental symptoms that they attributed to their dental amalgam fillings. No correlation was found between number of dental fillings and symptomatology. In addition, the dental amalgam group reported higher mean neuroticism than two comparison groups. The authors concluded that self-referred patients with health complaints attributed to dental amalgam are a heterogeneous group of patients who suffer multiple symptoms and frequently have

mental disorders. Similarly, Berglund & Molin (1996) measured urinary and blood mercury concentrations and estimated the amount of mercury release from dental amalgam among patients who had symptoms that they themselves thought were caused by amalgam. When compared with subjectively healthy referents, no difference was observed between the mercury status of the patients and referents.

Grandjean et al. (1997) evaluated the effects of chelation therapy versus a placebo on improvement for patients who attribute their illness to mercury from amalgam fillings. Of the symptom dimensions studied among the 50 patients examined, overall distress, somatization, obsessive–compulsive, depression, anxiety, and emotional lability were found to be increased. Following administration of succimer (meso-2,3-dimercaptosuccinic acid) at 30 mg/kg body weight for 5 days in a double-blind, randomized, placebo-controlled trial, urinary excretion of mercury and lead was considerably increased in the patients who received the chelator. Immediately after the treatment and 5–6 weeks later, most distress dimensions had improved considerably, but there was no difference between the succimer and placebo groups. The findings did not support the idea that mercury had caused the subjective symptoms of the patients.

In a case–referent study of 68 patients with Alzheimer disease and 34 referents, Saxe et al. (1999) observed no relationship between the disease and mercury exposure from amalgam fillings or concentration of mercury in the brain.

Altmann et al. (1998) compared visual functions in 6-year-old children exposed to mercury in a cohort of 384 children (mean age 6.2 years) living in three different areas of East and West Germany. After adjusting for confounding effects, some of the contrast sensitivity values were significantly reduced with increasing mercury concentrations. The authors concluded that even at very low urinary mercury levels, subtle changes in visual system functions can be measured[2]...

Siblerud & Kienholz (1997) investigated whether mercury from silver dental fillings (amalgam) may be an etiological factor in multiple sclerosis (MS). Blood findings were compared between MS subjects who had their amalgams removed (n = 50) and MS subjects with amalgams (n = 47). MS subjects with amalgams were found to have significantly lower levels of red blood cells, haemoglobin, and haematocrit compared with MS subjects with amalgam removal. Thyroxine (T4) levels were also significantly lower in the MS amalgam group, which had significantly lower levels of total T-lymphocytes and T-8 (CD8) suppressor cells. The MS amalgam group had significantly higher blood urea nitrogen (BUN) and BUN/creatinine ratio and lower serum immunoglobulin G. Hair mercury was significantly higher in the MS subjects compared with the non-MS control group (2.08 versus 1.32 mg/kg), suggesting an exposure to organic forms of mercury.

3.3. Renal effects

Incidents involving short-term exposure to high concentrations of mercury vapour have resulted in a range of effects, from mild transient proteinuria or slight changes in urinary acid excretion to frank proteinuria, haematuria, and/or oliguria to acute renal failure, with degeneration or necrosis of the proximal convoluted tubules.

[2] Note from Euro Chlor: due to the non-standard way of expressing the measured mercury in urine levels (μg mercury/24 hr) and the difficulty to collect 24 h sampling in a correct way, it is considered that the evidence for an association is, at most, weak.

Several studies have indicated that occupational exposure to elemental mercury causes increased urinary excretion of several proteins, such as ß-galactosidase, N-acetyl-ß-glucosaminidase (NAG), transferrin, ß2-microglobulin, or even albumin. Buchet et al. (1980) reported such effects in chlor-alkali workers with urinary mercury levels in excess of 50 µg/g creatinine …, and Roels et al. (1982) among two groups of workers exposed to elemental mercury with median urinary mercury levels above 71 µg/g creatinine. No sign of renal dysfunction was observed among 62 workers of a chlor-alkali or zinc-mercury amalgam factory, where the mean urinary mercury concentration was 56 µg/g creatinine (Lauwerys et al, 1983). Slight changes, mostly linked to tubular dysfunction, were observed in the study of Roels et al. (1985) (described in section 3.2.1 of this appendix) at a mean urinary mercury concentration of 30 µg/g creatinine. In a study in which several markers of renal changes were measured in a cohort of 50 workers exposed to elemental mercury and in 50 control workers, the exposed workers excreted an average of 22 µg mercury/g creatinine (31.9 µg/litre); their mean duration of exposure was 11 years. The main renal changes associated with exposure to mercury were indicative of tubular cytotoxicity (increased leakage of tubular antigens and enzymes into urine) and biochemical alterations (decreased urinary excretion of some eicosanoids and glycosaminoglycans, and lowering of urinary pH). The concentrations of anti-DNA antibodies and total immunoglobulin E in serum were also positively associated with the concentration of mercury in urine and blood, respectively. The renal effects were mainly found in workers excreting more than 50 µg mercury/g creatinine (Cardenas et al., 1993).

Another study investigated renal function and immunologic markers among chlor-alkali workers with long-term low exposure to mercury vapour. Forty-seven currently exposed workers were compared with reference workers matched for age in a cross-sectional design. The mean urinary mercury concentration was 5.9 nmol/mmol creatinine (Cr) for the exposed workers and 1.3 nmol/mmol Cr for the referents. The chlor-alkali workers had been exposed for an average of 13.3 (range 2.8-34.5) years. The activity of N-acetyl-beta-D-glucosaminidase in urine (U-NAG) was higher in the exposed workers. Associations were observed between current urinary mercury, cumulative urinary mercury, and cumulative urinary mercury per year (intensity) and U-NAG, autoantibodies to myeloperoxidase (anti-MPO) and proteinase 3 in serum,. The activity of U-NAG and anti-MPO was increased in the workers with the highest exposure, as assessed by their mean intensity of exposure. The highest activity of U-NAG was observed in the exposed workers with the lower concentrations of selenium in whole blood. The study indicates an effect of exposure on the kidney proximale tubule cells, possibly modified by individual selenium status, and an effect mediated by neutrophil granulocytes (Ellingsen, 2000a).

A recent publication (Efskind et al., 2006) describes a study which contained a sub-cohort of 41 formerly exposed and 40 referents from the study of Ellingsen (2000a). This follow-up showed full normalization when compared with referents. The levels of U-NAG were statistically significantly higher in the exposed group while they were exposed, but had declined to control levels when measured more than 4 years after cessation of Hg exposure. Also, both groups in the sub-cohort (exposed and referents) showed a statistically significant increase in U-NAG with age.

Eti et al. (1995) examined urinary mercury concentration and NAG excretion in 100 volunteers (18–44 years old) divided into two groups, with (66) or without (34) amalgam fillings. The authors concluded that although there was a very small difference in urinary NAG which probably indicates an apparent renal effect from metal absorbed

from amalgam fillings, this is insufficient to be a public health hazard for renal injury. A similar study by Herrström et al. (1995) used several proteins, including NAG, as markers of renal damage in 48 Swedish volunteers. These authors also failed to detect any significant indication of renal dysfunction or damage from amalgam.

3.4. Respiratory effects

Respiratory symptoms are a prominent effect of short-term, high-level exposure to elemental mercury vapours. The most commonly reported symptoms include cough, dyspnoea, and chest tightness or burning pains in the chest. Chronic cough has been reported in subjects exposed to elemental mercury vapour for several weeks. Workers accidentally exposed to mercury vapours at an estimated concentration of up to 44.3 mg/m^3 for 4–8 h exhibited chest pains, dyspnoea, cough, haemoptysis, impairment of pulmonary function (i.e., reduced vital capacity), diffuse pulmonary infiltrates, and evidence of interstitial pneumonitis. X-ray analyses of the lungs have primarily shown diffuse infiltrates or pneumonitis. Pulmonary function may also be impaired. Airway obstruction, restriction, and hyperinflation, as well as decreased vital capacity, have been reported. Decreased vital capacity has been reported to persist for 11 months after termination of exposure. In the more severe cases, respiratory distress, pulmonary oedema (alveolar and interstitial), lobar pneumonia, fibrosis, and desquamation of the bronchiolar epithelium have been observed.

3.5. Cardiovascular effects

Short-term inhalation exposure to high concentrations of elemental mercury vapour from the heating of elemental/inorganic mercury resulted in increased blood pressure and palpitations. Exposures of longer durations due to spills or occupational exposures have also been reported to result in increased blood pressure and increased heart rate.

In a study of causes of mortality in 1190 chlor-alkali workers in Sweden, a borderline significant excess was found with a mortality rate (standardised mortality rate: SMR) of 1.3 both for ischemic heart disease and cerebrovascular disease when allowing for a latency time of 10 years. The average U-Hg levels, according to the authors, would probably be around 100 µg/l (≈75 µg/g), i.e. around 200 µg/l in the 1950s, 150 µg/l in the 1960s and less than 50 µg/l in the 1980s (Barregård et al., 1990). Smoking habits were not known in this cohort. It was assumed that they were similar to the general Swedish population at the time, and a sample of 81 men, alive in 1984 and born between 1900 and 1939, showed an equal prevalence of current smoking (30%) as the then general population. Estimates by the authors showed that a 5% excess in the prevalence of smokers would not distort the statistical significance.

A parallel study of 674 chlor-alkali workers, employed before 1980, was performed in Norway (Ellingsen et al., 1993) with an average exposure level of U-Hg of 100 µg/l (≈75 µg/g). This study showed no excess of mortality from either vascular, cardiac, or CNS (central nervous system) causes, compared with the cohort from the general population. The SMRs were around unity (generally slightly below 1.0), when time under exposure or different latency times were accounted for. Hence, these results do not indicate an increased cardio-toxic or vascular risk. The prevalence of current smoking in this cohort was 56%, which was 15-20% higher than in the general population and was accounted for in the analysis.

In a study of causes of mortality in an American cohort (2133 subjects) exposed to metallic mercury no effect was found on cerebro-vascular deaths, even in the highest exposed sub-cohort of 858 workers with an exposure as U-Hg exceeding 300 µg/l (≈230 µg/g) (Cragle et al., 1984). There was also no excess of cardiovascular diseases in this sub-cohort (personal information from D. Cragle to Barregård 1990).

3.6. Gastrointestinal effects

A variety of gastrointestinal effects have been reported in humans following short-term inhalation exposure to high concentrations of elemental mercury vapour. A number of case studies have reported stomatitis (inflammation of the oral mucosa) following short-term exposure to high concentrations of elemental mercury vapours, occasionally accompanied by excessive salivation or difficulty swallowing. Stomatitis has also been observed in occupational settings in which workers were exposed to elemental mercury vapours for prolonged periods. Short-term exposure to high levels of mercury can also produce abdominal pain, nausea, and diarrhoea. Drooling, sore gums, ulcerations of the oral mucosa, or diarrhoea were observed in five of nine workers in a thermometer manufacturing plant.

Patients who were hypersensitive to mercury (indicated by positive patch tests) developed stomatitis at the sites of contact with amalgam fillings (Veien, 1990). The contact stomatitis faded when amalgam fillings were removed, but persisted in one patient who chose to leave them in place.

Bratel et al. (1996) investigated (1) healing of oral lichenoid reactions (OLR) following the selective replacement of restorations of dental amalgam, (2) whether there were differences in healing between contact lesions (CL) and oral lichen planus (OLP), and (3) whether there was a difference in healing potential when different materials were selected as a substitute for dental amalgam. Patients included in the study presented with OLR confined to areas of the oral mucosa in close contact with amalgam restorations (CL; n = 142) or with OLR that also involved other parts of the oral mucosa (OLP; n = 19). After examination, restorations of dental amalgam that were in contact with OLR in both patient groups were replaced. The effect of replacement was evaluated at a follow-up after 6–12 months. In the CL group, the lesions showed a considerable improvement or had totally disappeared in 95% of the patients after replacement of the restorations of dental amalgam (n = 474). This effect was paralleled by a disappearance of symptoms, in contrast to patients with persisting CL (5%), who did not report any significant improvement. The healing response was not found to correlate with age, gender, smoking habits, subjective dryness of the mouth, or current medication. However, the healing effect in patients who received gold crowns was superior to that in patients treated with metal-ceramic crowns ($P < 0.05$). In the OLP group (n = 19), 63% of the patients with amalgam-associated erosive and atrophic lesions showed an improvement following selective replacement. OLP lesions in sites not in contact with amalgams were not affected. Most of the patients (53%) with OLP reported symptoms also after replacement. From these data, the authors concluded that in the vast majority of cases, CL resolves following selective replacement of restorations of dental amalgam, provided that a correct clinical diagnosis is established. The authors note that metal-ceramic crowns did not facilitate healing of CL to the same extent as gold crowns.

3.7. Hepatic effects

Inhalation of mercury vapours produced by the heating of an unknown quantity of liquid mercury resulted in acute poisoning in a young child, which included hepatocellular effects. In another case, a man who died following short-term, high-level exposure to elemental mercury vapours was found to have hepatomegaly and central lobular vacuolation at autopsy.

3.8. Irritation and sensitization

Inhalation, oral, or dermal exposure to elemental mercury vapours has resulted in erythematous and pruritic skin rashes. Other dermal reactions to mercury exposure include heavy perspiration and reddened or peeling skin on the palms of the hands and soles of the feet, typically associated with acrodynia.

Red and burning eyes and conjunctivitis have been observed in people exposed to high concentrations of elemental mercury vapours.

3.9. Reproductive effects

Several studies found no effect on fertility following long-term inhalation exposure to elemental mercury in humans. Alcser et al. (1989) reported no effect on fertility in a retrospective cohort study of male workers exposed for at least 4 months in a US Department of Energy plant. Urinary mercury concentrations among the workers ranged from 2144 to 8572 µg/litre. Lauwerys et al. (1985) used questionnaires to assess the fertility of male workers exposed to mercury vapour from various industries (i.e., zinc-mercury amalgam, chlor-alkali, or electrical equipment product plants) and found no statistically significant difference in the number of children born to the exposed group compared with a matched control group. The concentration of mercury in the urine of these exposed workers ranged from 5.1 to 272.1 µg/g creatinine. Erfurth et al. (1990) and McGregor & Mason (1991) found no correlation between mercury exposure and prolactin, testosterone, luteinizing hormone, and follicle stimulating hormone levels and blood or urinary mercury levels in male workers occupationally exposed to mercury vapours.

An older study of 349 women exposed to elemental mercury vapour in the workplace reported that complications of parturition (toxicosis, abortions, prolonged parturition, haemorrhagic parturition) were increased compared with 215 unexposed controls (Mishonova et al., 1980). This study, however, had limited reporting and detail concerning the methods used. In contrast, no increase in spontaneous abortions was observed among dental assistants (potentially exposed to mercury vapour) in a historical prospective study of pregnancy outcomes among women in 12 occupations (Heidam, 1984). Similarly, no relationship between the amalgam fillings prepared per week and rate of spontaneous abortions or congenital abnormalities was observed in a postal survey in California, USA (Brodsky et al., 1985). No excess in the rate of stillbirths or congenital malformations was observed among 8157 infants born to dentists, dental assistants, or technicians, nor were the rates of spontaneous abortions different from the expected values (Ericsson & Källén, 1989). However, Rowland et al. (1994) reported that the fecundity of female dental assistants who prepared more than 30 amalgam fillings per week was only 63% (95% confidence interval 42–96%) of that of unexposed controls, although dental assistants with lower mercury exposure were more fertile than the referents (Rowland et al., 1994).

Menstrual cycle disorders were more frequent among women working in a mercury vapour lamp factory (exposures to mercury had been >50 µg/m3, but had decreased to <10 µg/m3 at the time of the study) than among referents (De Rosis et al., 1985). Among the married females in the factory, there was a higher prevalence of primary subfecundity and of dislocations of the hip in the newborns. The authors noted that the frequency of this anomaly varies between different regions in Italy. No excess was observed in the rate of spontaneous abortions.

3.10. Genotoxic effects

There is little information concerning the potential genotoxicity of elemental mercury. The overall findings from cytogenetic monitoring studies of workers occupationally exposed to mercury compounds by inhalation or accidentally exposed through ingestion provided no convincing evidence that mercury adversely affects the number or structure of chromosomes in human somatic cells.

3.11. Cancer

There is no sound evidence from epidemiological studies indicating that inhalation of elemental mercury produces cancer in humans. Although Cragle et al. (1984) found an increased incidence of lung, brain, and kidney cancers within an exposed cohort when compared with the general population, these incidences were not elevated in comparison with the referent cohort. Further, Kazantzis (1981) examined the incidence of cancers among workers exposed to a variety of metals, including mercury, and found no increases. No excess of cancer of the kidney or nervous system was found among a cohort of 674 Norwegian men exposed to mercury vapour for more than 1 year in two chlor-alkali plants (Ellingsen et al., 1993). An excess of lung cancer (type not specified) was found in Swedish chlor-alkali workers, but these workers had also been exposed to asbestos (Barregard et al., 1990). An excess of brain cancer was observed among Swedish dentists and dental nurses (Ahlbom et al., 1986; McLaughlin et al., 1987), while no excess risk of overall cancer mortality or of brain cancer was observed among dentists who were US Armed Forces veterans (Hrubec et al., 1992).

3.12. Other effects

Elevated white blood cell count was observed in a 12-year-old girl with a 6-month exposure to mercury vapour resulting from a spill of elemental mercury in her home (Fagala & Wigg, 1992). In another case-study report, thrombocytopenia and frequent nosebleeds were reported in two of four family members exposed to mercury vapour in their home as a result of an elemental mercury spill (Schwartz et al., 1992).

Increases in tremors, muscle fasciculations, myoclonus, or muscle pains have been reported following short-term, medium-term, or long-term exposure to elemental mercury vapour.

Some, but not all, studies have reported changes in autoimmune response. Some studies have also suggested that mercury can lead to increased susceptibility to infections, autoimmune diseases, and allergic manifestations. Allergic contact dermatitis has been reported but is a rare phenomenon (Kanerva et al., 1993).

Forty-seven chlor-alkali workers exposed to mercury vapour for an average of 13.3 years were compared with 47 referents matched for age in a cross-sectional study of thyroid

function. The mean urinary mercury concentration in the exposed workers was low compared with other studies of chlor-alkali workers: 5.9 nmol mmol-1 creatinine (range 1.1-16.8) versus 1.3 nmol/mmol creatinine (range 0.2-5.0 nmol mmol-1 creatinine) in the reference group. The median serum concentration of reverse triiodothyronine (rT3) was statistically significantly higher in the exposed subjects compared with the referents (268 pmol/l and range 161-422 pmol l-1, versus 240 pmol l-1 and range 129-352 pmol l-1; P = 0.009). The difference between the exposed subjects and the referents was most pronounced in the highest exposed sub-groups. The free thyroxine (T4)/free T3 ratio was also higher in the highest exposed subgroups compared with the referents. The median serum concentration of tumour necrosis factor alpha (TNF-alpha) was lower in the exposed subjects (7.3 pg ml-1 and range 4.4-69.7 pg ml-1 versus 8.0 pg ml-1 and range 6.0-34.6 pg ml-1; P = 0.004). Exposed subjects with the lowest urinary iodine (<67.8 nmol mmol-1 Cr) had higher serum concentrations of reverse T 3 and a higher free T4/free T3 ratio than the other subjects, suggesting that a low concentration of iodine in urine may be a risk factor for increased serum concentrations of reverse T3 and the free T4/free T3 ratio in subjects exposed occupationally to mercury vapour. The study could indicate a slight effect of low mercury vapour exposure on the function of the enzyme type I iodothyronine deiodinase, possibly modified by comparatively low urinary iodine concentrations (Ellingsen et al., 2000b).

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Appendix 2 - Respirators and protection factors

4. Air purifying respirator (chemical cartridge)

The decision on whether to use a half-face or full-face mask depends on the suspected concentration of mercury, the protection factor that is required, and potential existence of other risks. An Hg-P3 filter is the minimum that must be used and, depending on other risks, a combination of filters may be required.

For example, for combined exposure to chlorine and mercury a B-Hg-P3-filter should be used.

The service life of the respirator depends on the concentration of the mercury, but can also be reduced by the different factors (worker exertion level, cartridge variability, temperature and humidity of the air, other contaminants).

Normally, and especially for substances such as mercury which lack adequate warning properties like odour, taste, or irritation, an air purifying respirator should not be used unless the respirator has an adequate end of service life indicator (e.g., for Hg this could be a change of colour to indicate saturation).

Air purifying respirators should not be used in atmospheres:

- containing less than 19 % oxygen
- immediately dangerous to life or health (e.g. in case of high accidental exposure to chlorine)
- where carbon monoxide concentration exceeds 25 ppm

The respirators used for protection against mercury are generally small, easily maintained and do not greatly restrict the wearer's movements. However, they have a higher breathing resistance and limited useful service life time, and should be replaced very often. It is very important to discard the cartridge at least 30 days after opening the packaging, but for practical reasons it is highly recommended to replace the cartridge every week (or earlier depending on the concentration of mercury at the workplace). An important disadvantage to the use of such respirators is the negative air pressure created inside the respiratory inlet covering during inhalation, which can cause air contaminants to penetrate the covering if it fits poorly. Care should be taken to provide each wearer with a respirator that fits properly.

See Table 1 below for a selection guide, protection factors and maximum allowable concentrations.

Powered air-purifying respirators use a blower to pass contaminated air through a filter medium. The filter medium may be located in a belt-mounted holder or in the headpiece (helmet).

5. Air supplied respirators

Supplied air-line respirators use a central source of compressed breathing air delivered to the wearer through an air supply line. Air-line respirators are available as on-demand, pressure-demand and continuous-flow configurations. The central source of compressed air could be from a compressed air cylinder, a compressor, or purified compressed plant air.

The breathing air must be free of unwanted contaminants and must comply with the requirements of breathing air. The location of the compressor intake is critical to the purity of air supplied to the respirator user.

Couplings for breathing air must be incompatible with couplings and outlets for non-respirable compressed air and other gases.

The great advantage of air-line respirators is that they may be used for longer continuous periods. The breathing resistance and discomfort are minimal, On the other hand, the trailing air supply severely restricts the wearer's mobility and may fail due to hose damage.

Self-contained breathing apparatus (SCBA) is not proposed here as this equipment is mostly used for emergency situations and not for routine work, as it is heavy to work with over longer periods.

6. Assigned protection factors (APF)

The assigned protection factor (APF) of a respirator reflects the level of protection that a properly functioning respirator would be expected to provide to a population of properly fitted and trained users. For example, an APF of 10 for a respirator means that a user

could expect to inhale no more than one tenth of the airborne contaminant present.

Various groups have proposed factors for the different types of respirators available. The table 1 below gives an example with the British Standards Institution (BSI), and the Dutch Occupational Hygiene Society (DOHS) factors.

If these APFs are taken into account in combination with the fact that exposure needs to be controlled sufficiently below the exposure limit (0.025 mg Hg/m3), the following general rules are recommended:

- If exposure is > 0.025 mg Hg/m^3 and < 0.050 mg Hg/m^3 an air purifying half-mask (type Hg-P3 filter) can be used.
- If exposure is > 0.050 mg Hg/m^3 and < 0.250 mg Hg/m^3 an air purifying full-face mask (type Hg-P3 filter) can be used.
- If exposure is > 0.250 mg Hg/m^3 full-face air supplied respirators (pressure demand or continuous flow) are recommended.

Table 1. APFs for various types of respirators and a selection guide for Hg respirators (based on recommendations from BIS and DOHS).

Respirator Class and Type	**BSI/DOHS *APF***	***Max. allowable conc. (mg/m^3 Hg)***	
Air Purifying with HG-filter			
Half-Mask	10	10 x 0.025 =	**0.250**
Full-Face	20	20 x 0.025 =	**0.500**
Powered Air Purifying HG-filter			
Quart/Half-Mask TM1	10	10 x 0.025 =	**0.25**
Quart/Half-Mask TM2	20	20 x 0.025 =	**0.5**
Quart/Half-Mask TM3	20	20 x 0.025 =	**0.5**
Full-Face TM3	40	40 x 0.025 =	**1**
Hood or Helmet TH1	10	10 x 0.025 =	**0.25**
Hood or Helmet TH2	20	20 x 0.025 =	**0.5**
Hood or Helmet TH3	40	40 x 0.025 =	**1**
Supplied Air (by hose)			
Half-Mask	10	10 x 0.025 =	**0.250**
Full-Face	40	40 x 0.025 =	**1**
Mouthpiece	100	100 x 0.025 =	**2.5**
Hood	40	40 x 0.025 =	**1**

Supplied Air

Helm Light Duty LDH1	10	10 x 0.025 =	**0.25**
Half-Mask-Continuous	20	20 x 0.025 =	**0.5**
Helm Light Duty LDH2	20	20 x 0.025 =	**0.5**
Full-Face Light Duty LDM1	20	20 x 0.025 =	**0.5**
Full-Face Light Duty LDM2	20	20 x 0.025 =	**0.5**
Helm Light Duty LDH3	40	40 x 0.025 =	**1**
Full-Face Demand/ under pressure	40	40 x 0.025 =	**1**
Helm or Hood (constant air)	40	40 x 0.025 =	**1**
Full-Face Light Duty LDM3	40	40 x 0.025 =	**1**
Full-Face (constant air)	100	100 x 0.025 =	**2.5**
Half blouse (constant air)	100	100 x 0.025 =	**2.5**
Suite (constant air)	200	200 x 0.025 =	**5**
Mouth Piece (constant air)	1000	1000 x 0.025 =	**25**
Mouth Piece (demand – overpressure)	2000	2000 x 0.025 =	**50**
Full Face (demand – overpressure)	2000	2000 x 0.025 =	**50**

Appendix 3 - Monitoring of mercury in the workplace: details

In this annex, details are provided on the objectives of mercury monitoring campaigns, sampling and analytical methods and the required equipment. A decision framework also is presented that allows definition of the frequency and number of measurements

1. Monitoring objectives

There are different monitoring objectives or strategies that can be grouped into three categories: initial, periodic and diagnostic monitoring.

1.1. Initial Monitoring

Initial monitoring is used to assess compliance with an occupational exposure limit (OEL). In this first step fewer measurements (e.g., 3 samples) can be sufficient. Depending of the results of these, it is possible either to stop, or perform more measurements.

1.2. Periodic Monitoring

Periodic monitoring is used to evaluate exposure profiles that are judged to be acceptable. The frequency of measurement depends on the Exposure Rating, which is the level of exposure in relation to the exposure limit.

If documented properly, periodic monitoring data can also be used for future epidemiological studies.

In the case of periodic compliance monitoring, only personal monitoring techniques are appropriate

1.3. Diagnostic Monitoring

Diagnostic monitoring should be conducted when identifying the exposure source in order to understand how sources, tasks, and other variables (e.g., production rates) contribute to worker exposure. The results from this can help an industrial hygienist devise the most appropriate and efficient control strategies for unacceptable exposures.

If the source is from a fixed piece of equipment, area-monitoring techniques are appropriate. If the source moves with the worker or depends on individual work practices, personal monitoring techniques are appropriate. In most cases, measurement averaging times should reflect process cycle times rather than the exposure limit averaging times. Using a longer averaging time will simply average out the actual data required for problem identification. For example, when one intends to monitor exposure during a task of 10 minutes, but sampling time began 10 minutes before this and continues 10 minutes after the task, the measurement is of a concentration equal to 10'/30' of the real concentration during the task.

2. Monitoring methods

Monitoring and analysis methods for determination of mercury in air are given in the Euro Chlor guideline ***Analytical 6 – Determination of Mercury in Gasses***.

2.1. Personal air monitoring

The best estimate of an individual's exposure is obtained by taking breathing zone samples (personal sampling) for the entire working period. This optimum is not always practical and the actual sampling time should be arranged so that it mostly covers the potentially highest exposures.

2.2. Active sampling

The active sampling method is the most common method to assess the personal exposure to mercury of workers.

The principle is that a measured volume of air is drawn by a pump through an adsorption (Hopcalite)-tube. After the exposure the amount of mercury is determined with the analytical method ISO 17733 (Workplace air - Determination of mercury and inorganic mercury compounds - Method by cold-vapour atomic absorption spectrometry or atomic fluorescence spectrometry).

2.3. Passive or diffuse sampling

The theory of this sampling approach is that a sampler collects molecules transported to the adsorbent by molecular diffusion. The effective sampling rate of such a device can be predicted from Fick's law, provided that the diffusion constant of the compound is known.

Both passive and pumped sampling have been validated and fulfil the requirements as defined in the European standards EN 482 (*General requirements for the performance of procedures for the measurement of chemical agents*) and 838 (*Diffuse samplers for the determination of gases and vapours*) regarding workplace atmospheres:

2.4. Attaching the sampling equipment to the worker

Attach the tube or monitor to the operator, preferably on the lapel, and not more than 30 cm away from the nose-mouth region. The tube should be connected to the pump that is attached to a belt around the waist of the worker.

2.5. Area monitoring

Area (or fixed-point) measurements can be done with the same devices that are used for personal air monitoring, but with a direct Reading Monitor. Area-monitoring can be used to identify background concentrations at the workplaces and are useful to measure the effectiveness of control measures (e.g., local exhaust ventilation) over a period of time.

Area-point measurements should not be used to verify compliance with OELs.

2.6. Direct Reading Monitors

A general feature of direct-reading instruments is the short averaging time, which makes these monitors suitable for objectives such as detection of peak exposures, source detection, detection of contamination of clothes, shoes and cupboards, and trend analyses of back ground concentrations.

These monitors are **not suitable** for assessing the personal exposure of workers in comparison with OELs. However, they are very useful for measuring background levels so that trends of concentrations in the workplace can followed, with the aim of preventing higher exposures of workers.

There are many mercury monitors on the market, including Shaw-city, Baccharah and EPM.

When a monitor is used to make decisions on acceptable/unacceptable concentrations, the monitor **must be calibrated** directly before and after the measurements. Therefore each site needs its own calibration set.

If calibration is not carried out at each site immediately before measurement the experience at some sites is that errors of more than 100 % are possible.

3. Analytical method

The analytical method most sites use is based on the ISO 17733 method (Workplace air - Determination of mercury and inorganic mercury compounds - Method by cold-vapour atomic absorption spectrometry or atomic fluorescence spectrometry).

This method also describes sampling procedures.

3.1. Factors that could affect results

From the company SKC (supplier of sampling equipment) the following information is available about factors that could affect results in a major way:

- the order in which reagents are added;
- whether the exact amount is added; and
- how long the mixture is agitated for;

3.2. Breakthrough during sampling

A standard Hopcalite or Hydrar tube consists of 200 mg of a mixture of copper and manganese oxide in one section. To detect "breakthrough" two tubes connected to each other should be used. When a significant amount is found in the second tube, breakthrough from the first tube has occurred. In line with international recommendations (e.g., NIOSH - National Institute Occupational Safety and Health - US), if the second section holds less than 10 % of the mass of the first tube (W2W1 <10%), one may conclude that no perceptible breakthrough occurred. Under this condition, the sample is valid and the sum of W1 and W2 is used to calculate the concentration.

This means that the laboratory must analyse the two tubes separately and report the W1 and W2 to the industrial hygienist in charge of the measurement campaign.

3.3. Maximum flow rate and sample volume

The most important question a hygienist must ask when using a sorbent tube and to avoid unacceptable breakthrough is, what total volume of air should be sampled?

NIOSH defines a breakthrough volume (V_b) in its analytical methods for relatively worst-case conditions. Breakthrough volume is the maximum volume that can be sampled, when relative humidity (RH) = <80 % and the airborne concentration = 2x the occupational exposure limit, so that

W_B/W_F <10%.

The NIOSH 6009 method for mercury recommends a flow rate between 150 ml and 250 ml per minute and a maximum sample volume of 100 litres. Under these conditions one can sample between 400 and 660 minutes before reaching the breakthrough volume. The maximum airborne concentration is not known.

4. Air sampling plan

4.1. General

To compare an exposure level of mercury with the exposure limit it is necessary to know the concentration of mercury in the breathing zone extrapolated to the same reference period as that used for the limit value (i.e., 8 hours for mercury).

At least 75% of the reference period should be sampled. However, if during more than 25% of the reference period no exposure occurs, a sampling time less than 75% is permissible. For work shifts greater than 8 hours, the exposure limit should be extrapolated and the sampling time should be 75% of the longer work shift.

4.2. Pump out or on when respirator is used?

To assess compliance of an SEG (Similar Exposure Group) with the exposure limit, the whole exposure period should be sampled without taking use of a respirator into account, as stated in European guidelines and OSHA standards (29 CFR).

> **"Employee exposure" and other similar language referring to the air agent level to which an employee is exposed means the exposure to the airborne agent that would occur if the employee were not using respiratory protective equipment".**

If an exposure of a SEG is not in compliance with the exposure limit, workers must immediately wear respirators and technical measures must be considered to control exposure. If the technical measures cannot reduce exposure to an acceptable level, workers should continue to wear respirators.

In such a case one can measure the "**daily intake**" of workers as follows. Sample the most exposed task(s) for which the worker must wear adequate respirators and calculate the daily intake by subtracting this concentration from the result of the compliance measurement result for the same job. Take into account the protection factor of the respirator (e.g., a full face mask with an A2 cartridge has a protection

factor of 200, which means that the concentration measured for the specific task must be divided by 200).

4.3. Initial monitoring

If the initial qualitative assessment of an SEG cannot be classified as acceptable or unacceptable, initial monitoring is used to assess adherence to an exposure limit (OEL). The number of measurements varies from 3 to 6 samples.

Number of measurements

- In the first step of the first exposure assessment three measurements are enough.
- If one or more measurements exceed the exposure limit, the exposure profile is judged as unacceptable. Take control-measures or reassess the exposure profile. It can be that the measured shift was not a normal (normative) day. If it is plausible that the high concentration is not representative of normal operations, it is possible to discard the result and take another measurement.
- If it appears that the mean concentration of the three measurements is above 10% of the OEL, three or more further measurements should be performed.
  The distribution should be verified as log-normal and the Upper Confidence Limit ($UCL_{95\%,\ 95\%}$) should be calculated by Land's "Exact" method. Judge if the exposure is acceptable or unacceptable (see 5.2.4 in the main document) and decide if periodic measurements are necessary.

4.4. Periodic measurements

If an SEG is judged as acceptable, periodic measurements must be organized with the aim to verify on a regular basis that exposure conditions have not changed and that technical exposure control measures are working properly.

In many countries the seasons and time of day will have some influence on the level of exposure due to climatic changes or work practice differences.

In the case of mercury the measurement strategy for some exposure SEGs's should be based on measurements in two seasons and across different shifts.

Exposure Mean Concentration: ≥10% and <100% OEL

The following measurement strategy should be performed:

1. Establish in each season one day of monitoring, e.g. monitor on February 5th, May 5th, August 5th and November 5th.
2. On the day before monitoring randomly select in each SEG a worker for monitoring in each of three consecutive shifts (e.g. the Day-, Evening- and Night-shifts.)
3. Carry out the measurements (including at times when there is less working activity) on that specific day for the selected worker. Keep in mind that measurements for the specific worker are not of primary importance; it is the function or activity that the worker is carrying out that is the focus.
4. For every SEG use the new measurements and the previous nine measurements taken the year(s) before to calculate the arithmetic mean concentration. Verify if the distribution is log-normal and calculate the $UCL_{95\%,\ 95\%}$ by Land's "Exact" Method.

Judge if the exposure is acceptable (see 5.2.2.3).

Exposure Concentration: ≥1% and <10% OEL

The following measurement strategy should be performed:

1. Establish one day of monitoring in the worst-case season, e.g. monitor on August 5th.
2. On the day before monitoring randomly select in each SEG a worker for monitoring in each of three consecutive shifts (e.g. the Day-, Evening- and Night-shifts.)
3. Carry out the measurements (including at times when there is less working activity) on that specific day for the selected worker. Keep in mind that measurements for the specific worker are not of primary importance; it is the function or activity that the worker is carrying out that is the focus.
4. For every SEG use the new measurements and the previous nine measurements taken the year(s) before to calculate the arithmetic mean concentration. Verify if the distribution is log-normal and calculate the UCL95%, 95% by Land's "Exact" Method.

Exposure Concentration: < 1% OEL

Additional sampling is not necessary for an SEG with an exposure concentration less than 1% of the OEL.

4.5. Quality control

It is essential that the laboratory performing analysis of the air samples has a sound quality program and participates in inter-laboratory tests.

Appendix 4 - Health surveillance of mercury exposure

1. General medical history (anamnesis)

1.1. At employment specific emphasis should be put on the following conditions which might preclude employment with occupational exposure to Hg at a chlor-alkali plant:

1.1.1. History of chronic kidney disease

1.1.2. History or symptoms of (chronic) Central Nervous System (CNS) disease

1.1.3. History or symptoms of peripheral neuropathy

1.1.4. Woman planning for pregnancy (advice)

1.1.5. Other medical conditions

1.2. Follow up on the same items as 1.1

2. Objective signs

2.1. Urine analysis

2.1.1. Qualitative (stix for protein, blood)

2.1.2. Quantitative for protein/albumine if chronic kidney disease or diabetes

2.1.3. Microscopy if newly detected proteinuria (looking for cylinders)

2.2. Blood analysis

2.2.1. Serum creatinine

2.2.2. Total protein/albumin if clinical proteinuria

2.3. Orientational neurology

When?

- At least at pre-employment examination
- If symptoms from CNS or PNS

What?

- Covering the items at the end of this annexe

3. Risk communication

3.1. Check the person's knowledge of the potential health hazards of Hg exposure

3.2. Check the knowledge of his/her own exposure (as monitored by e.g. air Hg conc. or urine Hg) and compare with the registered factual data (personal or SEG) and correct incorrect opinions

3.3. Check personal habits in hygiene and use of PPEs and inform of improved practice if necessary.

3.4. Check knowledge on rules and regulations in performing his occupational duties. Tutoring, if important gaps exist, might be a means of improvement in "high" exposure

3.5. Advise change in occupational environment if continuous high exposure in spite of correctional measures/training

3.6. Consider advising change in employment status if increased susceptibility by other predisposing health conditions (especially at pre-employment medical examination, e.g., high exposure to organic solvents for more than 5 years, diabetes, TIA, stroke, alcohol abuse)

3.7. Temporary change in exposed occupations/tasks during pregnancy and breast-feeding

Check-list for simplified neurological examination

Opticus reflexes
Pupils' light/closeness reflex

Motor nerve responses
Oculomotion
N. Facialis function
N. Glossopharyngeus
N. Hypoglossus

Sensory (touch and pain needle-point)
The 3 branches of trigeminus
Upper limbs
Truncus front
Lower limbs
Joint position 1 toe

Tendon reflexes
Jaw
Triceps
Biceps
(Ante-)Brachial
Patellar
Achilles

Cutaneous reflexes
Abdominal
(Cremaster)
Plantar

Co-ordination tests
Finger-finger
Finger-nose
Knee-heel
Balance (Romberg's tests)

Tonus
Alternating supination/pronation
Spasticity
Rigidity
Tremor at rest
Tremor: intentional

Reference on specific questionnaires for neurological examination: Hogstedt C, Andersson K, Hane M. "A questionnaire approach to the monitoring of early disturbances in central nervous functions" in: Aitio A, Riihimaki V, Vainio H, eds. Biological Monitoring and Surveillance of Workers Exposed to Chemicals. Washington DC, Hemisphere, 1984:275–87

Appendix 5 - Records and management of all documentation

Records should be kept in order for various reasons:

- They are necessary for a proper medical surveillance
- They are the basis of the process of continuous improvement
- They could be used for future medical research
- They could be useful in case of a lawsuit.

Directors, engineers and physicians are responsible for organizing and preserving their records.

The records have to be in compliance with national and local regulations.

The records have to be kept for several decades. Generally speaking, it is necessary to organize such records in a way that somebody who never saw the particular facility could understand how everything was managed. This is especially important in the case of mercury as all European mercury electrolyses are supposed to be shut down before 2020: after this, knowledge about mercury hazards, hygiene, bio-monitoring, and risk assessment will slowly be forgotten.

As in the case of all archives, industrial and medical records have to be protected against fire and other disasters. Records could be archived on paper or electronically. Nevertheless, because it could be difficult in the course of a lawsuit to prove that records have not been modified, it is recommended that at least the most important documents are printed and archived.

If there are successive editions of particular records, each of them has to be numbered, dated and kept.

The following list of records is not necessarily exhaustive and is set out according to the chapters in the appropriate audit (Health 6).

Organisation and management

- Occupational Health policy
- Occupational Health and Industrial Hygiene missions and responsibilities

Health hazards of mercury

- Documents used in training of workers about mercury hazards
- Documents used to information visitors about mercury hazards

Personal hygiene standards

- Documents used for training in housekeeping
- Documents on the supplying and washing of working clothes
- Documents about Personal Protective Equipment (PPE)
- Results of audits of the hygiene management system

Biomonitoring of mercury exposure

- Method of analysis and external quality controls of the laboratory

- Method of surveillance, including frequency of measurements and controls after high results
- Information on the worker regarding his personal results

Monitoring of mercury in the working environment

- Method of analysis and external quality controls of the laboratory
- Method of surveillance, including frequency of measurements and controls after high results
- Information on the worker regarding his personal results and the results from his Similar Exposure Group

Risk assessment

- Document assessing the risk: hazard x level of exposure
- Risk assessment: duration, frequency, risk

Risk management

- Technical and organisational tasks
- Assessment of dependence on PPE
- Assessment of compliance with Biological Exposure Index (B.E.I.)

Health surveillance

- Individual medical files
- Document showing the urinary mercury results of Similar Exposure Groups (anonymised)

Health-related actions in case of over-exposure

- Check list in case of over-exposure

Information and training for employees

- Document showing the involvement of the occupational physician in the provision of information and training of employees about mercury hazards
- Document about information/training on mercury risks in all jobs/tasks and how this has been critically evaluated
- Standard operating procedures, including instructions about mercury risks
- Scheduled training programme
- Lists of participants at information meetings

Internal audit

- Records of all audits (questionnaires and answers)

Appendix 6 - List of abbreviations

ACGIH: American Conference of Governmental Industrial Hygienists

AIHA: American Industry Hygiene Association

BEI: Biological Exposure Indice

EASE: Estimation and Assessment of Substance Exposure

HSE: Health Safety and Environment

LEV: Local Exhaust Ventilation

OEL: Occupational Exposure Limit

OSHA: Occupational Safety and Health Administration

RPE: Respiratory Protective Equipment

SCOEL: Scientific Committee on Occupational Exposure Limits

SEG: Similar Exposure Group

STEL: Short Term Exposure Limit

TLV: Threshold Limit Value

TWA: Time Weighted Average

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Avenue E Van Nieuwenhuyse 4

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B-1160 Brussels

Belgium

Tel: +32 2 676 7211

Fax: +32 2 676 7241

e-mail: eurochlor@cefic.be

Internet: http://www.eurochlor.org