MEDICAL SURVEILLANCE
GUIDELINES 2023

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TABLE OF CONTENTS

INTRODUCTION

Occupational health practice should meet the aims of occupational health which have been defined by the ILO and WHO in 1950 and updated as follows by the ILO/WHO Joint Committee on Occupational Health in 1995:

Occupational health should aim at:

- The promotion and maintenance of the highest degree of physical, mental and social well-being of workers in all occupations;
- The prevention amongst workers of departures from health caused by their working conditions;
- The protection of workers in their employment from risks resulting from factors adverse to health;
- The placing and maintenance of the workers in an occupational environment adapted to his physiological and psychological capabilities;
- To summarise, the adaptation of work to man and of each man to his job.

The main focus in occupational health is on three different objectives:

- The maintenance and promotion of workers' health and working capacity;
- The improvement of working environment and work to become conducive to safety and health;
- Development of work organisations and working cultures in a direction which supports health and safety at work and in doing so, promoting a positive social climate and smooth operation that may enhance productivity of the undertakings. The concept of working culture is intended in this context to mean a reflection of the essential value systems adopted by the undertaking concerned. Such a culture is reflected in practice in the managerial systems, personnel policy principles for participation, training policies and quality management of the undertaking.

Occupational health is a multifaceted activity concerned with the prevention of ill health in employed populations. This involves the consideration of the two-way relationship between work and health. Its aim is to prevent illness, rather than cure ill health from wherever it arises at the workplace.

Occupational medicine is preventative medicine practised in the workplace by safeguarding and promoting health and wellbeing in the workplace.

The problems facing the occupational health team is attempting to establish a link between work and health, as each employee brings a legacy of genetic, social, dietary and environmental factors affecting health to the workplace.

This manual, updated November 2022, serves to strengthen medical surveillance through clinical management, relieving uncertainty about the implications of illness for working life, and enabling sound advice on the steps to be taken for the best outcome.

DEFINITIONS and ABBREVIATIONS

ACGIH	**A**merican **C**onference of **G**overnmental **I**ndustrial **H**ygienists
ATSDR	**A**gency for **T**oxic **S**ubstances and **D**isease **R**egistry
Ambient	The measurement of assessment of agents at the workplace. It evaluates ambient exposure and health risk compared to an appropriate reference. In industry, ambient monitoring usually means monitoring the airborne concentration of a chemical.
BAT	Biological tolerance level derived from German "**B**iologische **A**rbeitsstoff-**T**oleranzwerte". The BAT value describes the occupational medical and toxicological derived concentration for a substance, its metabolites or an effect parameter in the corresponding biological material at which the health of an employee generally is not adversely affected, even when the person is repeatedly exposed during long periods. BAT values are based on a relationship between external and systemic exposure or between the systemic exposure and the resulting effect of the substance. The derivation of the BAT value is based on the average of systemic exposures.
BEI	**B**iological **E**xposure **I**ndex is a value for assessing biological monitoring results and is intended as a reference guideline
Biological effect monitoring	The measurement of biological effects resulting from absorption of chemicals. (For clarification, the measurement of changes to any functions, for example liver functions, kidney function, full blood count etc. Also includes non-pathology tests, lung function, X-rays, etc.)
Biological monitoring	A planned program of periodic collection and analysis of body fluid, tissues, excreta or exhaled air in order to detect and quantify the exposure to, or absorption of, any substance or organism by human beings. (For clarification, the measurement of specific substances, for example lead, mercury and chrome.)
Body burden	This refers to the total amount of a specific chemical/agent in the body of an individual at the time of sampling. It is an indication of personal exposure over time to a specific chemical which has the tendency to accumulate within the different tissue reservoirs.
CDC	**C**entre for **D**isease **C**ontrol
CL	The **C**eiling **L**imit is the maximum airborne concentration of an HCA determined over the shortest analytically practicable period of time-which does not exceed 15 minutes.
D	**D**iscretionary

Dose	The amount of a chemical/agent absorbed or retained in an organism during a specific time interval.
Epidemiology	The study of the distribution and determinants of health-related states and events in employee populations, and the application of the study to control health problems.
ES	**E**nd of **S**hift
EWW	**E**nd of **W**ork **W**eek
Health Monitoring	Systematic continuous or repetitive health-related activity designed to lead, and if necessary, to corrective actions.
Health surveillance	The periodic medico-physiological examination of exposed workers (prior, during and on leaving the workplace) with the objective of early diagnosis of occupational disease, the protection of health and the prevention of occupational-related disease.
HCA	**H**azardous **C**hemical **A**gents are any chemical substance (solid, liquid, gas, vapour, dust, powder, etc.) that have the potential to cause harm. This is usually a chemical listed in the tables published in the Regulations for Hazardous Chemical Agents of the Occupational Health and Safety Act.
HEG	**H**omogeneous **E**xposure **G**roups
I	**I**nhalable fraction
ICOH	**I**nternational **C**ommission of **O**ccupational **H**ealth
IDLH	**I**mmediately **D**angerous to **L**ife or **H**ealth
IFV	**I**nhalable **F**raction and **V**apour
Internal dose	Biological monitoring attempts to estimate the internal dose on the basis of our knowledge on the fate of the chemical in the body. Depending on the chemical and the analysed biological parameter, the term internal dose may cover different concepts. It may also refer to the **amount of chemical recently absorbed.** Internal dose may also mean the **amount of chemical stored in one or in several body compartments or in the whole body** (integrated exposure or specific organ dose). This usually applies to cumulative toxic chemicals.
ILO	**I**nternational **L**abour **O**rganisation

Medical Surveillance (OHSA)	A planned program of periodic examination of employees (which may include clinical examinations, biological monitoring or medical tests) by an occupational health practitioner or, in prescribed cases, by an occupational medicine practitioner.
MSDS	**M**aterial **S**afety **D**ata **S**heet
NC	**N**ot **C**ritical
NIOSH	**N**ational **I**nstitute of **O**ccupational **S**afety and **H**ealth
BEI Notations	**As noted in the ACGIH BEI documentation and the Hazardous Chemical Agent regulations of March 2021**
B	Background - the determinant may be present in specimens from subjects not occupationally exposed. Such background levels are incorporated into the BEI
Nq	Non-quantitative - BM should be considered but no BEI could be determined due to insufficient data
Ns	Non-specific - determinant is non-specific and is also observed after exposure to other chemicals
Sq	Semi-quantitative determinant is an indicator of exposure to the chemical, but the quantitative interpretation of the measurement is ambiguous. These determinants should be used as a screening test if a quantitative test is not practical or as a confirmatory test if the quantitative test is not specific and the origin of the determinant is in question.
SKIN	Danger of cutaneous absorption
CARC	Carcinogenicity - based on the GHS categorisation, including category 1A and 1B
RSEN	Respiratory sensitisation
DSEN	Dermal sensitisation
OEL (OHSA)	**O**ccupational **E**xposure **L**imit values set by the Minister, which represents the airborne concentration of an HCA and where the exposure standard may be: An 8-hour time weighted average (TWA) A ceiling limit (CL) A short-term exposure limit (STEL)
OSHA	**O**ccupational **S**afety and **H**ealth **A**dministration
OHSAct	**O**ccupational **H**ealth and **S**afety **Act** (85 of 1993)
PLSWW	**P**rior to **L**ast **S**hift of **W**ork **W**eek
PPE	**P**ersonal **P**rotective **E**quipment
PS	**P**rior to **S**hift

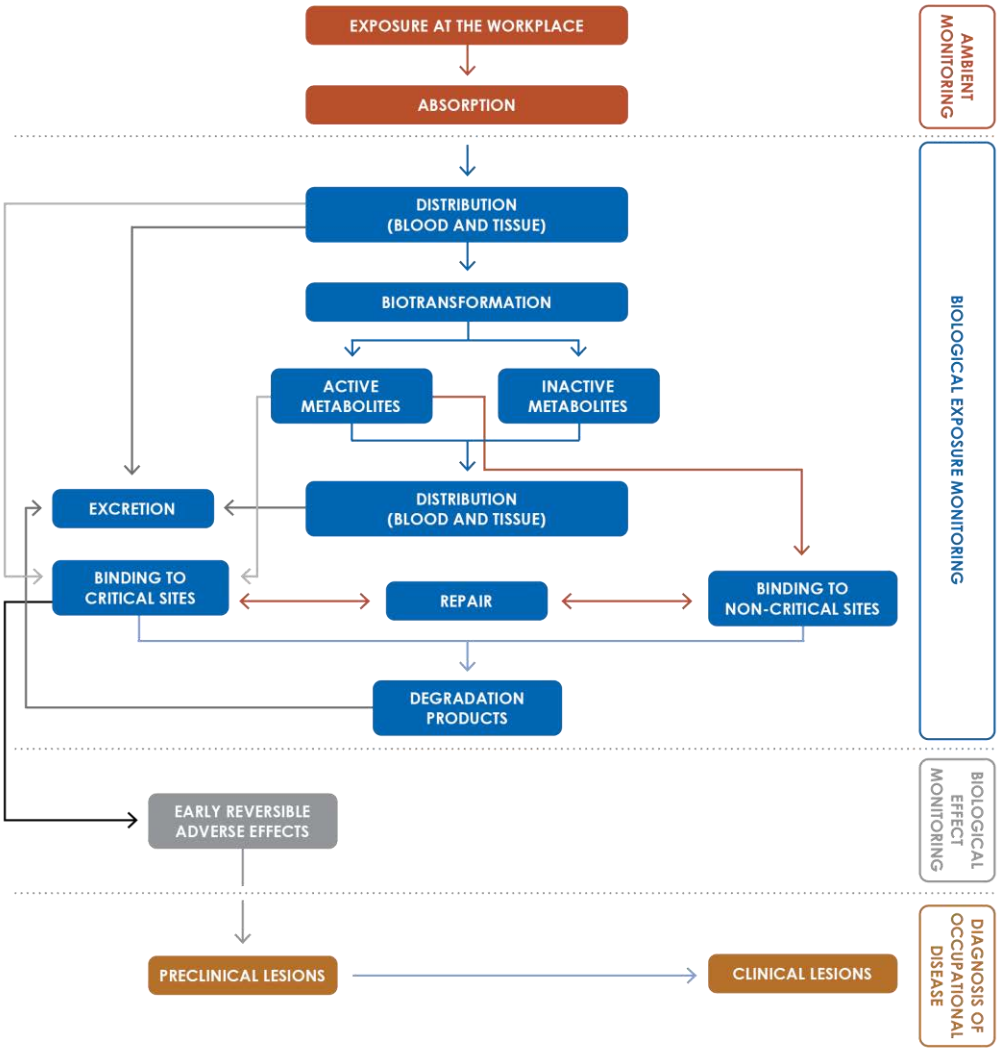
R	**R**espirable fraction
RL	The **R**estricted **L**imit
STEL	The **S**hort-Term **E**xposure **L**imit is the time-weighted average maximum airborne concentration of an HCA over a 15 minute period.
TWA	**T**ime **W**eighted **A**verage is the maximum average airborne concentration of a HCA, calculated over an 8 hour working day period for a 5-day working week.

1. MEDICAL SURVEILLANCE

The objective of medical surveillance is to cover and record the spectrum of potential effects of a hazardous substance on an employee from absorption of the substance through to clinical disease.

The basis of medical surveillance is the following up on the fate of a chemical from the environment that is exerting biological effects from the environment to target molecules in the worker.

The figure below shows the fate of the chemical absorbed from the environment and the different monitoring programs that the occupational health team can follow to manage exposure within a complete medical surveillance program.



1.1 AMBIENT MONITORING

By measuring the external exposure to a chemical the health risk can be assessed. In the industry ambient monitoring usually means monitoring the airborne concentration of a chemical. Threshold limit values (TLVs) and Occupational Exposure Levels (OELs) are then used to establish the presence or absence of a health hazard. Depending on the type of air sampling system selected – stationary (area) or personal (worker breathing zone) – the estimate of the risk may be carried out on a group or an individual basis. Ambient monitoring is more suitable than biological monitoring for the detection of acute exposure to chemicals. It can often be quickly applied to potentially hazardous conditions. If hazardous conditions are found, preventive measures can be instituted before severe adverse health effects occur. Ambient monitoring is usually more practical than a biological method to identify emission sources and evaluate the efficiency of engineering control measures. A single ambient monitoring operation may prevent overexposure of many individuals and more regular, longer sample methods may be required to better understand exposures.

1.2 BIOLOGICAL MONITORING

Biological monitoring measures the biochemical concentrations of HCA and/or their metabolites in biological samples for exposed individuals, e.g., blood lead for inorganic lead exposure, or urinary arsenic for inorganic arsenic exposure. The aim is to measure the degree of absorption into the body by measuring indicators in representative biological samples, typically urine or blood (usually not related to the target organ).

Biological monitoring approaches
The pathology tests currently used for monitoring of exposure to chemicals can be classified into three categories:

1. Determination of the chemical or its metabolites in biological media
The majority of tests currently available for biological monitoring of exposure to chemicals rely on the determination of the chemical or its metabolites in biological media. The biological media most commonly used are urine, blood and is less frequently exhaled air. It is also possible to analyse biological materials, such as faeces, adipose tissue, hair, nail or saliva.

According to their specificity, these tests can be classified into two subgroups:

- **Selective tests** are based on the direct measurement of the unchanged chemicals or their metabolites in biological media. The unchanged substance is measured when:
    - It is not or is poorly bio transformed;
    - When there is no knowledge about the metabolites (no toxicokinetics data);
    - When the level of exposure is too low for a significant amount of metabolite to be produced;
    - When a high degree of specificity is required (a metabolite may be common to several exposures);

    - When sensitive methods for detecting the metabolites are not available.

- **Non-selective tests** are used as non-specific indicators of exposure to a group of chemicals. As an example of nonselective exposure tests, one can cite the determination of nonselective urinary mutagenicity and thio-ether assays for coal-tar product exposure. The selective method for this exposure will be 1-Hydroxypyrene. Because of their lack of specificity (for instance, thio-ether excretion may be increased by non-mutagenic or carcinogenic exogenous or endogenous substances and is influenced by smoking) and the existence of a large individual variability, these tests usually cannot be used to monitor exposure on an individual basis. It is, however, possible that, when an adequate control group is used as reference, they may be useful as qualitative tests to identify exposed groups.

2. Quantification of (reversible, non-adverse) biological effects related to the internal dose

This second category of tests includes those based on the quantification of non-adverse effects which are related to the internal dose. Most of these tests are non-specific. The development of these tests usually requires some knowledge of the mechanism of action of the chemical. An example of these tests is the use of the inhibition of pseudocholinesterase activity in serum to assess exposure to organophosphorus compounds.

3. Measurement of the amount of active chemical interacting with the target and non-target (surrogate) molecules

Contrary to the preceding exposure tests, those belonging to this third category directly or indirectly estimate the amount of chemical interacting with the sites of action. When they are feasible, i.e., when the target site is easily accessible, these tests have the potential to assess the health risk more accurately than any other monitoring procedure. The determination of carboxy haemoglobin is an example that has been used in occupational medicine for a long time.

Comparison of Ambient and Biological Monitoring of Exposure

Biological monitoring of exposure offers several advantages over environmental monitoring to evaluate the internal dose and hence to estimate the health risk. Biological monitoring takes into consideration inhalation, ingestion and dermal absorption, where ambient monitoring only takes inhalation into consideration. Personal hygiene habits vary from one person to another. The lack of care in personal hygiene can lead to significant ingestion of the substance (skin contamination, smoking, eating or drinking in the work area). The incorrect use of protective clothing (e.g. gloves) can result in increased skin contamination and absorption. Because of its capability to evaluate the overall exposure (whatever the route of entry), biological monitoring has the advantage that it can be used to test the effectiveness of various protective measures, such as gloves, masks, and barrier creams.

Biological monitoring also takes into account non-occupational background exposure (leisure activity, residency, dietary habits, smoking, etc.) and extra occupational exposures which may also be expressed at the biological level. The internal load therefore comprises the total external (environmental and occupational) exposure.

Great inter-individual variation also exists in the absorption rate of a chemical through the lungs, skin or gastrointestinal tract as well as in the capacity for metabolising and excreting the substance. In some cases, even if strict personal hygiene measures are implemented so that the pollutant can enter the organism only by inhalation (in addition to the amount transported by mucociliary clearance from the lungs to the gastrointestinal tract), there is no reason to always postulate the existence of a relationship between the airborne concentration and the amount absorbed.

Many physico-chemical and biological factors preclude the existence of such a correlation (e.g., type of compound [for example, exposure to a same ambient level of soluble or insoluble metal compound does not result in the same biological level], particle size distribution [inhalable or respirable fraction], variation in workload influencing ventilatory parameters and cardiac output and hence the alveolar air or blood concentrations of volatile organic solvents, etc.). A biological parameter may take all these various toxicokinetic factors into consideration.

For all of the above reasons, it is clear that for many industrial pollutants, the measurement of the concentration in air (in particular for low level exposures) may not necessarily prevent an excessive intake by exposed workers. Moreover, in the case of exposure to chemical substances that exhibit their toxic action at the site of contact (e.g., eye mucosa or lung irritants, respiratory tract carcinogens) and are poorly absorbed, a biological parameter reflecting the internal dose is not necessarily related to the health risk. Only a few biological tests have been proposed for identification or monitoring of chemicals present at the interface between the environment and the body (skin, gastrointestinal mucosa, respiratory tract mucosa).

Finally, for many chemicals, the toxicokinetics (metabolism of the substance) and toxicodynamics (quantitative relationships among external exposure, internal exposure and adverse effects) data are still too limited to propose a valid and practical biological method for assessing the risk of overexposure.

From the above considerations, it is clear that both ambient monitoring and biological monitoring of exposure represent two complementary approaches for health risk assessment in industry.

Sampling for biological monitoring
Biological media
The majority of available biological tests rely on the analysis of breath, blood or urine. The choice of the medium depends on several factors, such as the kinetics (appearance and half-time of the biological parameter), the convenience of sample collection, or the possibility of sample contamination.

It is important to clearly identify the worker and obtain consent before collecting any samples for biological monitoring.

1. Blood
Blood constitutes the main vehicle for the transport and distribution of chemicals in the body. Therefore, most systemically active substances or their metabolites can be found in blood. It can be used for measuring most inorganic chemicals and for

organic substances which are poorly bio-transformed and have a sufficient half-time. Moreover, the determination of an unchanged substance in blood may have a greater specificity than the determination of its metabolites in urine. Blood is also useful for the measurement of substances that bind to macromolecules, for example, surrogate molecules such as haemoglobin.

Some practical considerations have to be taken into account as depending on the substance; the analysis should be performed on whole blood, plasma, serum or erythrocytes.

The biological parameter to be assessed can either be equally distributed between the different blood constituents or accumulate in a particular blood compartment (e.g., red cells). Hemolysis of red blood cells, a frequent phenomenon occurring during blood sampling, transport, storage or mishandling may lead to erroneous results of analyses performed on plasma or serum. Also, some chemicals or their metabolites can be transported in blood free or bound to proteins.

The blood concentration of many volatile solvents frequently has the same significance as that in alveolar air. It reflects either the most recent exposure when blood is collected during exposure or the exposure during the preceding day if blood is collected 16 hours after the end of exposure. The blood concentration of some cumulative organic chemicals (e.g., polychlorinated biphenyls) mainly reflects the body burden, the blood level of these chemicals being related to their concentration in the main storage compartment. Blood collection may be considered as too invasive by workers.

Guidelines on blood collection

Responsibilities of the company
- Provide a clean environment
- Clean working surfaces
- Chair(s) for employee(s)
- Clean water to wash arms with Savlon or soap or, if at all possible, a shower and a clean uniform
- Preferably paper towels to dry arms

The practitioner taking the blood sample must ensure the worker has:
- Given informed consent for biological monitoring
- Showered and have put on a clean uniform, or
- Come directly from home and therefore was not exposed to the work environment,
- Remove the contaminated uniform as far as possible to expose the arm from the shoulder to the hand. The arm should be washed with clean water and soap (or Savlon), rinsed off with clean water and dried well, preferably with a paper towel or similar material.

Procedure for taking a blood sample
- The type of blood container required should be ascertained from the laboratory analysing the sample (EDTA/SST/Fluoride)
- Apply tourniquet
- Clean the venipuncture area with alcohol (use water if blood alcohol is tested)
- Insert needle, place the tube into the needle holder and release the tourniquet
- Complete blood-taking procedure

- Remove filled blood-tube and tilt gently several times
- Remove the needle from the arm and cover venipuncture site with clean swab, pressing firmly for about two minutes or until bleeding has stopped
- Do not bend the arm
- Apply plaster
- Verify patient details and label tube accordingly

2. Urine

Urine is easy to collect, the procedure is non-invasive and large volumes can be collected. It is usually suitable for monitoring water soluble metabolites of organic chemicals and several inorganic chemicals (metals). These tests are more readily accepted by the workers as they are less invasive than blood collection. In the case of exposure to substances with short biological half-times or with fluctuating airborne concentrations, the level of a metabolite in urine collected at the end of the shift is usually a better indicator of the average exposure during the shift than the concentration of the substance itself in exhaled air or blood samples. The latter (concentration of the substance in exhaled air or blood) is more effectively influenced by the very recent exposure.

The concentration of a substance in urine generally reflects its mean plasma level during the period of urine accumulation in the bladder, but for some substances the amount stored in the kidneys may also influence the urinary level.

Except in the case of exposure to substances with long half-times, measurements performed on 24-hour specimens might be more representative than those performed on spot samples. However, 24-hour samples are not frequently carried out in routine biological monitoring programs. In the case of exposure to rapidly excreted substances, such as solvents, an end of shift sample is more appropriate. It should, however, be noted that the urinary concentration of a metabolite greatly depends on the rate of urine production and its measurement in either too dilute (large beverage intake) or too concentrated (low beverage intake, perspiration due to hard work or high environmental temperature) urine specimens can lead to misinterpretation.

Collection of urine specimens - Acceptability

The determination of urinary creatinine and/or density (specific gravity) is usually advisable to exclude over-diluted and over-concentrated samples. Correction of the results for the dilution of the urine may be necessary for some substances but needs to be considered on its merits for each individual substance.

Since creatinine excretion depends to a certain extent on urinary flow, it has been suggested to correct creatinine concentration in "spot" urine for the effect of varying hydration. Some BEIs®, for determinants whose concentration is dependent on urine output, are expressed relative to creatinine concentration. For other determinants, such as those excreted by diffusion, correction for urine output is not appropriate. In general, the best method may not be available. When the field data are only available adjusted for creatinine, the BEI will continue to be expressed relative to creatinine. In other circumstances, no correction is recommended, and the BEI will be expressed as concentration in urine.

Urine specimens that are highly diluted or highly concentrated are generally not suitable for monitoring. The World Health Organization has adopted guidelines for acceptable limits on urine specimens as follows:

Creatinine concentration: >0.3 g/L (2.5 mmol/l) and <3.0 g/L (26.5 mmol/l)

Or Specific gravity: >1.010 and <1.030

A specimen falling outside either of these ranges should be discarded and another collected. Workers who provide consistently unacceptable urine specimens should be referred for medical evaluation.

Collection of urine specimens - Sampling time
Although the measurement of the elimination rate of a chemical may better reflect the internal dose than its concentration, quantitative urine collection during a defined time interval is rarely done in industry and is difficult to achieve. Mainly for metals, urine contamination during collection may also represent an important source of errors. The renal excretion is governed by three mechanisms: glomerular filtration, tubular secretion and tubular reabsorption. The alteration of one of these mechanisms may greatly influence the elimination of a substance. Additional tests for kidney function integrity may be necessary, in addition to creatinine correction (similarly for organ abnormalities, such as lung, liver and blood cells may need to be excluded or noted). Because the concentration of some determinants can change rapidly, the specimen collection time (sampling time) is very important and must be observed and recorded carefully. The sampling time is specified in the BEI and is determined by the duration of retention of the determinant. Substances and determinants that accumulate may not require a specific sampling time (refer to page 14 for the BEI sampling time).

Sampling time	Recommended collection
Prior to shift	16 hours after exposure ceases
During shift	Any time after 2 hours of exposure
End of shift	As soon as possible after exposure ceases
End of the workweek	After four or five consecutive working days exposure
Discretionary	At any time

Guidelines on urine collection

Responsibility of the practitioner receiving the urine sample

- Ensure the worker showers prior to urine collection, or the worker produces a urine sample on entering the factory site and before changing out of personal clothing into uniform.
- The sample should be collected either as a pre-shift sample (collected on the morning of the first workday) or a post-shift sample (collected at the end of shift, at the end of the work day).
- The type of urine container requested should be ascertained from the laboratory analysing the sample.
- Provide a clean urine container for sample collection.

- Ensure correct labelling, specific container for substance and that all details are correct.

Procedure for collecting a urine sample

- If possible, ensure that the employee is well-hydrated by giving 200 ml of water 30 minutes before urine collection.
- Supply the correct urine container to the employee with advice on the necessity of an uncontaminated sample.
- The employee should produce between 20 and 30 ml of urine.
- The lid to the container must be closed tightly.
- The container must be clearly labelled with the employee's name and/or employee number and date of collection.

1.3 BIOLOGICAL EFFECT MONITORING

Biological effect monitoring determines the intensity of biochemical or physiological changes to exposure, e.g., red cell cholinesterase for exposure to organophosphate or pesticides. Several factors affect the dependability of monitoring for exposure. The following is a list of those that should be considered including their impact on testing:

- **Workplace issues:** Poor risk assessments, complex HEG, longer working hours, PPE issues etc.
- **The worker demographics**: Age; gender; weight; diet; genetics and presence of disease.
- **Presence of underlying disease and ill health**: Workers with a pre-existing condition are less tolerant of exposure which may either aggravate an existing condition or show symptoms of exposure earlier due to an existing condition such as diabetes.
- **The home/community/moonlighting environment**: Pollution of drinking water and food, air pollution, working with toxic substances (hobbies/sport etc.) and other work exposures.
- **Behavioural patterns**: Personal hygiene, smoking, substance abuse, failure to change uniform, poor eating habits and failure to wear protective equipment supplied for the job. The employer assumes that the employee is protected from exposure but due to poor personal hygiene the worker eats/smokes in the workplace and ingests/inhales chemicals through this route. The work exposure is then added to by the environmental exposure increasing the BM results.
- **Timing of specimen collection** (see above)
- **Inappropriate specimen collection**: Such as contamination of the sample and its container by the environment; inappropriate storage and preservation of the sample, utilisation of inappropriate sample collection equipment such stainless steel needles that may contaminate a blood sample for chrome or nickel analysis.
- **Laboratory analysis error**: The laboratory should practise a disciplined quality control procedure, utilising the appropriate equipment for the analysis of the substance.

2. QUALITY ASSURANCE AND MEDICAL SURVEILLANCE

Each aspect of medical surveillance should be conducted within an effective quality assurance (QA) program. The appropriate specimen must be collected, at the proper time, without contamination or loss, and with use of a suitable container. Participants of biological monitoring should be selected according to clear and agreed exposure profiles and it is best to ensure that a representative sample of no, low, moderate and high exposure scenarios is included in the group tested. Donor identification, time of exposure, source of exposure, and the sampling time must be recorded. The development of a predictive model as to what the BM outcome will be for the different scenarios will support clarification of outliers. The analytical method used by the laboratory must follow routine quality control rules and the laboratory should participate in an external proficiency program. The laboratory should provide regular reports on this.

Additionally, the occupational health professional should provide known blind challenges to the laboratory along with worker specimens (e.g. blanks, purchased or spiked specimens containing the determinant, or split specimens, non-exposed samples which may help identify background levels in a specific plant, site, community and region). When blind challenges are used, the spiked determinant should be in the same chemical form and matrix as that being analysed by the laboratory. These blind challenges will enable the occupational health professional to assess the ability of the laboratory to process, analyse, and report results properly and to have confidence in the laboratory's ability to report accurately and consistently.

3. ADDITIONAL CONSIDERATIONS FOR OCCUPATIONAL MEDICAL SURVEILLANCE AND WORKPLACE HEALTH PROGRAMS

The ICOH code of ethics is intended to guide those who carry out occupational health activities and to set a reference level on the basis of which their performance can be assessed.

The purpose of occupational health is to serve the health and social well-being of the workers individually and collectively. Good occupational health practice provides for individual employee health protection and prevention through anticipation, recognition, identification and monitoring of workplace stresses and employee health. Increasingly, risk assessment, occupational risk and exposure profiling within medical surveillance programs is becoming individualised rather than generalised (homogenous exposure group). However, in many instances it is worthwhile to consider an "occupation or risk-based" health program, in particular for specialised occupations/individuals or groups, such as drivers, food handlers and users of drugs of abuse are examples.

An occupation or risk-based occupational health program allows for both individual and group interaction; and provides opportunities to better understand the interaction between work and health as well as create new opportunities to improve health for individuals and the group alike.

The principle of occupational health practice does not differ from the occupational or risk-based approach, in fact, it strengthens it. The basic principle of occupational health is to protect and prevent occupational disease.

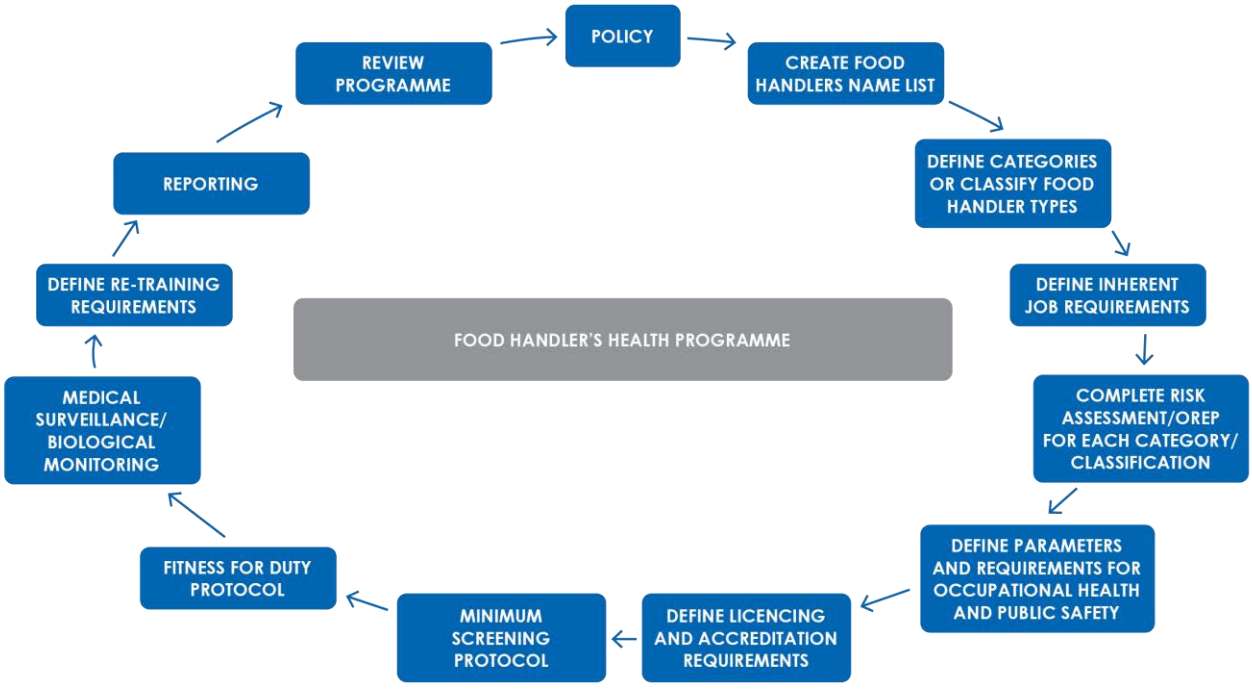
Occupational health practice must be performed according to the highest professional standards and ethical principles. Occupational health professionals must contribute to environmental and community health.

The duties of occupational health professionals include protecting the life and the health of the worker, respecting human dignity and promoting the highest ethical principles in occupational health policies and programs. Integrity in professional conduct, impartiality and the protection of the confidentiality of health data and of the privacy of workers are part of these duties.

Occupational health professionals are experts who must enjoy full professional independence in the execution of their functions. They must acquire and maintain the competence necessary for their duties and require conditions which allow them to carry out their tasks according to good practice and professional ethics.

WORKPLACE HEALTH PROGRAMS

A basic framework for workplace health program for food handlers is available below. All workplace health programs are evolutionary in nature, requiring regular review, consultation and update. This framework can be adapted for any occupation, risk, workplace and/or activity.



4. DESIGNING AND IMPLEMENTING A PROGRAM OF MEDICAL SURVEILLANCE AS PER HCA REGULATIONS

The following steps should be included in any program:

- **Risk assessment** to determine the potential exposure to and routes of absorption of any HCA, as required by Reg 5.
- **Identification of target-organ toxicity**, so as to direct medical screening.
- **Selection of appropriate tests and testing schedule:** Tests should have the desirable operating characteristics of high sensitivity, specificity, reliability and predictive value. Frequency of testing is laid down in Reg 7(2), but should be based on an understanding of the nature of the hazard and the natural history of any adverse effects.
- **Development of action criteria:** These are provided for some HCAs in the form of BEIs in Table IV of the Regulations.
- **Standardisation of testing process:** Quality control needs to be exercised both in the testing site and in the laboratory contracted to carry out analyses. Consistency over time should be sought so as to make longitudinal measurements comparable.
- **Ethical considerations:** Information and training of employees as required by Reg 3(1) should include the rationale for doing medical surveillance and the consequence of abnormal findings. An employee must be notified of the results and interpretation of his/her tests and any recommendations made. The confidentiality of personal medical records is laid down by Reg 9.
- **Determination of an employee's fitness to remain in that job (Reg 7(3)):** Results may be compared against the action criteria (BEI if relevant), and also the employee's previous results to determine whether individual action needs to be taken. Action may include repeating the test, further medical examination, removal of the employee from further exposure, and notification of the employer. Co-operation of employees can be best secured through a policy of protection of conditions of service in case of medical removal from a particular job.
- **Evaluation of control:** An abnormal finding in an employee, or a pattern of findings in a group of employees, may point to inadequate primary control of exposure. In such cases it is necessary to evaluate the workplace problem and take remedial action.
- **Recordkeeping:** Includes both medical records and exposure information for every employee. While the employer is responsible for recordkeeping in terms of Reg 9, the contents of personal medical records may be accessible to the OMP, the employee, and any person nominated by the employee in writing

5. DESIGNING EXPOSURE PROFILES FOR MEDICAL SURVEILLANCE

Exposure profiles are designed to assist with the understanding of the exposure, its chemical and physical characteristics, metabolism and effects on the body. It also outlines how to conduct the biological and biological effect monitoring of each exposure. Below are general references and information used to generate these profiles found in this medical surveillance guideline which can assist in compiling and or improving your own exposure profiles.

Topic	Reference & Information
Chemical Formula	MSDS/NIOSH/CAS/IUPAC
CAS number	Unique numerical identifier assigned by the Chemical Abstracts Service (CAS) to every chemical substance. This unique code assists with referencing on global databases.
Toxicokinetics and toxicodynamics	ILO/NIOSH/ ACGIH/MSDS/ CDC /ATSDR/Pubmed/published literature (use search engine)
Clinical manifestations of occupational exposure	ILO/NIOSH/ ACGIH/MSDS/ CDC /ATSDR/Pubmed/published literature (use search engine)
Metabolism (includes absorption & elimination)	ILO/NIOSH/ ACGIH/MSDS/ CDC /ATSDR/Pubmed/published literature (use search engine)
Occupational exposure	IDLH: CDC/NIOSH STEL: OHSAct OEL: OHSAct
OCCUPATIONAL EXPOSURE	
Biological Monitoring	Exposure-specific metabolites to be tested as well as BEI per exposure used as per OHSAct. If not specified in the Act, the company should decide on the BEI to be used. Ampath provides an BEI or equivalent from the following references: **International/USA** • Lauwerys R.R. et al. Industrial Chemical Exposure. Guidelines for Biological monitoring • NIOSH/ACGIH/ATSDR/Pubmed **Germany** • Biological Tolerance values (BAT)
Biological Effect Monitoring	Monitor possible effects of exposure, ensure that non occupational exposure and confounders are eliminated. Abnormal results should be followed up for diagnosis or enhanced protection/removal from the workplace. Make use of ILO/NIOSH/ ACGIH/MSDS/CDC/ATSDR/Pubmed/published literature (use search engine).

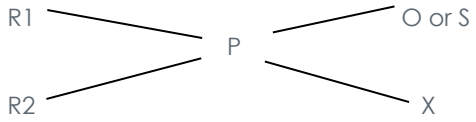
6. EXPOSURE PROFILES

ACETONE

Chemical Formula	**C_3H_6O**	
CAS number	**67-64-1**	
Occupational uses	It is primarily used as an industrial solvent and chemical intermediate. Acetone is also found in/used as a solvent for: • Paints • Varnishes • Lacquers • Cements • Leather and rubber industries • Fabric coating and dyeing process	
Toxicokinetics and toxicodynamics	**Route of entry: Inhalation, ingestion and dermal absorption.** Acetone is readily absorbed from the lungs and exhalation is the major route of elimination. Its terminal metabolite (carbon dioxide), and the fraction of administered acetone that is exhaled as unchanged acetone is dose related. Acetone, because of its solubility in water, is readily absorbed into the bloodstream and is thus transported rapidly throughout the body. Acetone is metabolised slowly and may accumulate in the body throughout a 40-hour work week. Can be metabolised through most tissues in the body, but the liver is the primary site. Exposure to acetone vapour results in an estimated retention of 45% up to a level of 80%. It is thought that these levels of retention may be lower in women. The half-life in alveolar air is about 4 hours, in venous blood 6 hours, and in arterial blood 4 hours. Urinary excretion of acetone and its metabolites occurs but this route of elimination is minor (1%). The highest concentration of acetone in urine is found 3-3.5 hours after exposure. Workload affects the mean levels of acetone in body fluids i.e., higher workloads leading to higher levels in body fluids. Skin absorption is possible. In healthy human subjects, acetone levels in blood covers a range of 0.15-15.4 mg/L with arithmetic means ranging from 0.29 - 1.59 mg/L. Urinary excretion of acetone in normal unexposed subjects is in the range of 0.127-9.350 mg/IL with an arithmetic mean of 0.842 mg/L. Acetone in blood, isopropanol in urine and formic acid in urine are also potential markers of exposure. Acetone appears in humans as an endogenous product of normal metabolism. With abnormal fat breakdown, ketones will appear in the urine before the serum as in diabetic ketoacidosis and increase in acetone, diabetes, fasting, pregnancy or genetic status.	
Clinical manifestations of occupational exposure	Short-term exposure usually results in eye irritation, dryness of the mouth and throat, nausea and vomiting, headache, sleepiness, dizziness, light-headedness, and fainting. Repeated exposure causes dermal inflammation, as well as inflammation of the gastrointestinal and respiratory tracts. Volunteers experienced slight irritation at 300 ppm. Eye irritation, headache, light-headedness, nasal irritation, and throat irritation were noted in workers exposed to concentrations considerably in excess of 1,000 ppm.	
Occupational exposure	IDLH STEL OEL	2500 ppm 1000 ppm 500 ppm
OCCUPATIONAL EXPOSURE		
Biological Monitoring	**Sample:** 1. Acetone in urine	**Sampling time:** ES (End of Shift)
	Acetone in urine: Notation	**BEI (Biological exposure index):** 25 mg/l ES Ns

Biological Effect Monitoring pathology based	Blood: urea, creatinine, eGFR and liver function.

ACETYLCHOLINESTERASE INHIBITORS

Chemical Formula	 (R1 and R2 represent alkoxy substituents, while X can be anything from a simple alkyl group to an aromatic ring, a derivative of either of the former, or a halogen or nitrile)	
Occupational uses	Pesticides INHIBITING ACETYLCHOLINESTERASE	
Toxicokinetics and toxicodynamics	**Route of entry: Inhalation, ingestion (food) and dermal absorption** The onset and severity of symptoms depend on the chemical structure of the compound being used, the amount of ACTIVE INGREDIENT to which an individual is exposed, route of exposure, rate of metabolic degradation, respiratory rate, ambient temperature, humidity and/or the use of personal protective equipment. Following oral or respiratory exposure, signs and symptoms appear IMMEDIATELY OR 3 hours later, while with dermal exposure, they are usually delayed to 12-hours post-exposure. These products inhibit cholinesterase enzymes, resulting in an accumulation of the neurotransmitter acetylcholine. This leads to the overstimulation of muscarinic receptors, i.e., excessive cholinergic activity. Most organophosphorus chemicals are eliminated in the urine in the form of metabolites (dialkyl phosphates). About 90% of the compound is eliminated between 6 and 24 hours after absorption.	
Clinical manifestations of occupational exposure	**Short term exposure**: Acute exposure may cause headache, dizziness, weakness, cramps, tightness of chest, wheezing, watering of mouth and blurring of vision. Convulsions and coma may occur. **Long term exposure:** Prolonged or repeated exposure makes an individual susceptible to systemic intoxication.	
Occupational exposures	IDLH STEL OEL	Product specific Product specific Product specific
OCCUPATIONAL EXPOSURE		
Biological Monitoring	**Sample:** 1. Whole blood cholinesterase activity	**Sampling Time:** Discretionary Pre-shift/Post exposure [generally baseline taken during season or peak application period] True Baseline Level = taken 4 weeks after non-exposure; ideally 2 baseline measurements must be done 3 to14 days apart and should agree between 15 and 20%.
	1. Pseudocholinesterase – serum (CHS)	After acute exposure
	1. Red cell cholinesterase 2. CHS	**Reference limits:** 70% of baseline, a reduction of 30% or more from a basal (pre-exposure) level may indicate organophosphate/cholinesterase inhibitor toxicity/exposure. M&F: 3167-6333 U/L

Biological Effect Monitoring pathology based	Blood	Full blood count, urea, creatinine, eGFR, electrolytes, ALT, AST, GGT, ALP
	Urine	Dipstick

ALUMINIUM

Chemical Formula	**Al**	
CAS Number	**7429-90-5**	
Occupational Uses	Occupational exposure occurs in refining the primary metal and in secondary industries that use aluminium. Aluminium is produced by the Hall-Heroult process, which involves the electrolytic reduction of alumina (Al2O3) in large carbon-lined steel vessels called pots ("potroom"). Aluminium as a pure metal or in alloys is used to make a range of products in the aircraft and automotive industries, and is also used in the manufacture of electrical conductors. The main sources of non-occupational exposure include the use of beverage and food containers (particularly when used for acidic foods), paints, explosives, fireworks, water treatment, aluminium-containing cosmetics and medicinal products.	
Toxicokinetics and toxicodynamics	**Routes of entry: Inhalation, ingestion and dermal absorption** Inhalation of airborne particles in dust and fumes (primary smelting, foundry work, production of aluminium flake powder, and welding of aluminium). The bioavailability of aluminium depends on the chemical form which affects the health risks. Inhaled soluble particles, i.e. aluminium sulphate/nitrate and hydrated aluminium chloride are rapidly absorbed from the lungs. The less soluble forms, i.e. aluminium metal, aluminium hydroxide/oxide/phosphate/silicate are retained in the lung and slowly released into the systemic circulation. Aluminium accumulates in blood if not filtered by the kidney, i.e. urine is the main route of excretion. Hence, in impaired renal function, aluminium accumulates. The main sites of deposition are the skeleton and lungs (to a lesser extent in the muscles, kidneys, liver and brain). The major proportion in the blood compartment is avidly bound to serum proteins such as transferrin and rapidly distributed throughout the body. The kinetics of aluminium excretion suggest two compartments, i.e. a relatively rapid elimination rate and a slower one most likely after redistribution from the major deposition sites. Accumulation in healthy workers, particularly in the skeleton, is from long term exposure (aluminium flakes and welding) with elimination occurring at different rates over many years.	
Clinical manifestations of occupational exposure	**Central nervous system:** Encephalopathy with abnormal speech, myoclonic jerks, seizures, dementia (long-term dialysis). **Respiratory system:** Aluminium-induced pulmonary diseases (aluminosis - Shaver's disease), chronic obstructive airways disease (multi-factorial confounders implicated i.e., silica, welding fume and fluorides), asthma and pulmonary fibrosis. **Musculoskeletal system**: Osteomalacia, aluminium-related bone disease **Skin**: sensitisation/dermatitis.	
Occupational exposures	IDLH STEL OEL	Not available Not available 2 mg/m^3 (Respirable fraction)
OCCUPATIONAL EXPOSURE		
Biological Monitoring	**Sample**: 1. Aluminium in serum 2. Aluminium in urine	**Sample time:** ES (End of Shift), EW (End of Work week) ES (End of shift), EW (End of Work week)

	1. Aluminium urine: 2. Aluminium serum:	***Tentative maximum permissible concentration:** 150.0 ug/g creatinine ES,EW Exposure to fumes seems to produce higher urinary levels of aluminium than exposure to dust. ***Reference range**: <0.57 umol/l (<16 ug/l) ***Patients on hemodialysis**: <1.49 umol/l (<41 ug/l) ***Toxic:** >1.85 umol/l (>50 ug/l)
Biological Effect Monitoring-pathology based	Blood	Full blood count, urea, creatinine and electrolytes, PTH
	Urine	Albumin or protein excretion (as per OMP discretion)

ANILINE

Chemical Formula	**$C_6H_5NH_2$**
CAS number	**62-53-3**
Occupational uses	Used as: • A chemical intermediate for the dye, agricultural, polymer and rubber industries • A solvent and antiknock compound for gasoline • For the production of MDI (methylene diphenyl diisocyanate) and PMPPI (Polymeric MDI)
Toxicokinetics and toxicodynamics	**Route of entry: Inhalation, ingestion and dermal absorption** Fifteen to sixty percent of inhaled aniline is oxidised to p-aminophenol, which is excreted by the kidney. As it is heavier than air, it may cause asphyxiation in poorly-ventilated areas. The toxic effects of aniline are probably due to the metabolite phenylhydroxyl-amine. Exposure due to burning of plastic and tobacco and smoking. Small amounts are found in food, such as apples, beans, rhubarb, corn and grains. Polymorphism in human N-acetyltransferase divides the human population in two groups, i.e. those with a high enzyme activity (fast acetylator) and those with a low enzyme activity (slow acetylators). Fifty percent of Europeans are particularly affected from aniline-induced hematotoxicity due to a genetically caused lower acitivity of N-acetyltransferase meaning that the reaction of aniline to acetanilide is retarded in favor to the formation of phenylhydroxylamine. As a result, MetHb levels are higher in slow acetylators (1.0-1.5%) than in fast acetylators (0.7-1.2%) after occupational exposure to airborne aniline below the OEL in Germany (Lewalter and Korallus, 1985).
Clinical manifestations of occupational exposure	Aniline is irritating to mucous membranes and affects the eyes, nose, skin and respiratory tract. Severe exposure can lead to inebriation-like symptoms (anilinism) and even coma. Short-term or acute exposure from inhalation can cause methemoglobinemia (causing formation of methaemoglobin, feroglobin and Heinz bodies), resulting in functional anaemia. Initial symptoms of cyanosis (15% methaemoglobin) and headache are followed by shortness of breath, nausea and vomiting, weakness, dizziness (40% methaemoglobin), tachycardia, arrhythmia and coma (75% methaemoglobin). Symptoms may occur 2 to 4 hours post exposure depending on the exposure level. Generally, an increase in MHb above the normal background level in blood (about 1.1%) to 15% will be without signs and symptoms. However, as known from CO-induced oxygen deficiency for sensitive risk groups (persons with latent restricted coronary or arterial function), much lower MHb-levels may be tolerable (Bolt et al 1985). Clinical cyanosis but no hypoxic symptoms except for a possible slight euphoria will develop at 15-20% MHb and higher. Fatigue, anxiety, headache, weakness, dizziness, tachycardia, dyspnoea, and syncope will occur at 30-45% MHb. Higher concentrations will cause a decreased level of consciousness and finally coma, heart failure and death at more than 60- 70% MHb. Symptoms of bladder cancer such as blood in urine, lumps in groin area, painful urination and lower back pain have been observed. Alanine is a potential carcinogen to bladders of humans but there is no information on its teratogenicity or reproductive toxicity in humans.

Occupational exposure	IDLH STEL OEL	100 ppm Not available 4 ppm
OCCUPATIONAL EXPOSURE		
Biological Monitoring	**Sample:** 1. Total p-aminophenol in urine 2. Methaemoglobin in blood	**Sampling time:** ES (End of Shift) During or ES
	1. Total p-aminophenol: creatinine 2. Methaemoglobin in blood Notation	**BEI (Biological exposure index):** 50 mg/l >1.5% of haemoglobin B, Ns, Sq
Biological Effect Monitoring pathology based	Blood	Full blood count, urea, creatinine, eGFR, GGT methaemoglobin, haemoglobin, ALT, AST
	Urine	Dipstick

ANTIMONY

Chemical Formula	**Sb**	
CAS Number	**7440-36-0**	
Occupational uses	Antimony is a brittle metal mixed with alloys. It is used in: • Lead storage batteries, solder, sheet and pipe metal, bearings, castings, and pewter. • Paints, ceramics, and fireworks, and as enamels for plastics, metal and glass. Antimony is aslo added to textiles and plastics to prevent them from catching fire. It is incorporated into thermoelectric materials used in nanoparticle technology.	
Toxicokinetics and toxicodynamics	**Routes of entry: Inhalation** is the primary route of occupational exposure. **Ingestion, dermal absorption and eye contact** are minor contributors. It is rapidly excreted in urine (pentavalent) and faeces (trivalent). It is suggested that complete excretion of absorbed antimony occurs after occupational exposure in the preceding week and without any exposure over the weekend. The elimination half-life is 95 hours. Some long-term retention in the lungs may occur with some antimony compounds. Antimony is found throughout the environment and in food in small amounts.	
Clinical manifestations of occupational exposure	**Non-specific symptoms:** Headache, sleepiness, dizziness, metallic taste, weight loss, nausea, diarrhoea, vomiting, impaired smell and tight chest. **Irritant:** Upper respiratory tract, ocular conjunctivitis and dermatitis (antimony spots). **Lungs:** Pneumoconiosis- related inflammation with fibrosis, chronic bronchitis, chronic emphysema and pleural adhesions. **Cardiac:** Arrhythmias, hypertension **Musculoskeletal:** Arthralgia, myalgia **Liver:** Elevated alanine transferase and aspartate transferase **Reproductive:** Spontaneous abortion and premature birth Not classified as a carcinogen	
Occupational exposures	IDLH STEL OEL	50mg Sb/m^3 Not available 1 mg/m^3 CARC

OCCUPATIONAL EXPOSURE		
Biological Monitoring	**Sample:** Antimony in urine	**Sample time:** Not critical
	Antimony: Creatinine	**Tentative maximum permissible concentration:** 35 ug/g creatinine
Biological Effect Monitoring pathology based	Blood	ALT, AST, ALP, GGT, full blood count, urea, creatinine

ARSENIC

Chemical Formula	**As**	
CAS Number	**7440-38-2**	
Occupational uses	Manufacture of insecticides, weed killers and fungicides. Used in: • Wood preservatives and in the manufacture and handling of calcium arsenate. • Manufacture of electrical semiconductors, diodes and solar batteries and as a bronzing or decolorizing addition in glass manufacturing. • Production of opal glass and enamels. Arsenic is also used as an addition to alloys to increase hardening and heat resistance/non-ferrous smelting and used for recycling of electronic waste.	
Toxicokinetics and toxicodynamics	**Routes of entry: Inhalation, ingestion, dermal and eye absorption** Toxicity associated with exposure to inorganic arsenic i.e. arsenite/As (III) and arsenate/As (V). Also of concern are the methylated metabolites i.e. Monomethylarsonic Acid (MMA) and Cacodylic Acid (DMA) that are used in pesticides and in feed additives for poultry and swine. The rate of absorption is highly dependent on the solubility and valence state. Blood levels are elevated for a short period of time after which they are rapidly incorporated into the body phosphate pool (the body treats As as phosphate). In acute exposures, As is detected only for a few hours post-exposure. Toxicity is due to the effects on energy transfer as (i) avidly binds to the cofactor for pyruvate dehydrogenase thereby inhibiting conversion of pyruvate to acetyl CoA for gluconeogenesis; and (ii) competes with phosphate for ATP production. Also binds to the sulfhydryl groups of proteins resulting in conformational change in the protein and loss of activity. It interferes with the activity of enzymes in haeme synthesis.	
Clinical manifestations of occupational exposure	**Acute:** Nausea, vomiting, diarrhoea, weakness, loss of appetite, colic, cough, chest pains, headache, dyspnoea and hemoglobinuria. Arsine gas is a haemolytic toxin. **Chronic Health Effects:** Peripheral nerve inflammation (neuritis) and degeneration (neuropathy), reduced peripheral circulation, anaemia, increased mortality due to cardiovascular failure and cancers of the skin, lungs and lymphatic system, hyper pigmentation, thickening of the palms and soles (hyperkeratosis), contact dermatitis, skin sensitization, warts, ulceration and perforation of the nasal septum. In addition, arsenic is a potential autotoxin and can cause hemolysis, gastrointestinal disturbances, mild jaundice and renal dysfunction. Inorganic arsenic is a known human carcinogen (Category 1). As arsenic can cross the placenta, there is a risk of reproductive disorders such as spontaneous abortion. Prolonged exposure may lead to hair loss.	
Occupational exposures	IDLH STEL RHCA-OEL	5mg As/m^3 Not available 0.02mg/m^3 CARC

OCCUPATIONAL EXPOSURE		
Biological Monitoring	**Sample:** Total arsenic in urine	**Sampling time**: EWW (End of Work Week)
	Arsenic in urine Notation	**BEI (Biological Exposure Index) :** 35 ug/l (total arsenic) EWW (In the absence of the consumption of seafood for 2 days prior to specimen collection.) B
Biological Effect Monitoring-pathology based	Blood	Full blood count differential, ALT, AST, GGT, ALP, bilirubin, urea, creatinine
	Urine	Dipstick (proteinuria, hematuria), albumin or protein excretion (as per OMP)

BENZENE

Chemical Formula	**C_6H_6**
CAS number	**71-43-2**
Occupational uses	As solvent and raw material for chemical synthesis, as impurity in chemical processes (petrochemicals, toluene, xylene, paints, varnishes, rubber cements and lacquers. Has also been used in the rubber and leather industries.
Toxicokinetics and toxicodynamics	**Route of entry: Inhalation, ingestion and dermal absorption** Inhalation is the most important route of exposure. Absorbed benzene is rapidly distributed throughout the body and tends to partition into fatty tissues. The liver serves an important function in benzene metabolism. The metabolism of benzene is inherently complex; most of the metabolism is performed by the liver and lungs, with secondary metabolism occurring in the bone marrow. Following inhalation exposure, the main route of elimination of unmetabolised benzene is through exhalation. About a third of benzene retained in the body is excreted in urine as conjugated phenol and dihydroxy phenols. The remainder is either absorbed in tissue (fat) or exhaled as CO_2. In the OH setting, dermal absorption contributes to 20 to 40% of the total dose of benzene absorbed. The urinary metabolites include phenol, S-phenylmercapturic acid (SPMA) and trans, trans muconic acid (TTMA). The choice of sampling times and sampling intervals has a marked effect on the findings as indicated in the table below:
Clinical manifestations of occupational exposure	High concentrations by inhalation or oral ingestion lead to central nervous system depression (benzol jag - type of drunkenness) and death. Benzene irritates the skin and mucous membranes. Long-term occupational exposures lead to bone marrow depression (thrombocytopenia, anaemia - more so in females, granulocytopenia and aplastic anaemia). Leukaemia of the myelogenous variety is most common. Benzene is a known carcinogen (IARC category 1) and a suspected human reproductive agent. Chronic exposure

Air		**Sampling time: End of exposure or end of shift**		
Benzene (ml/m^3)	**Benzene (mg/m^3)**	**Whole blood Benzene (ug/l)**	**SPMA (ug/g creatinine)**	**TTMA (ug/g creatinine)**
0.3	1	0.9	0.010	-
0.6	2	2.4	0.025	1.6
0.9	3	4.4	0.040	-
1.0	3.3	5	0.045	2
2.0	6.5	14	0.090	3
4.0	13	38	0.180	5
6.0	19.5	-	0.270	7

	can manifest with renal tubular dysfunction, hepatocellular damage and neurotoxicity (personality and mood changes, memory loss). In workers exposed to high levels of the mixture of organic solvents (much greater than the permissible levels), a linear dose-response relationship has been reported between the exposure level, risk of hearing loss, and hearing threshold at high frequencies, especially 8000 Hz.	
Occupational exposures	IDLH STEL OEL	500 ppm 5 ppm 1 ppm
OCCUPATIONAL EXPOSURE5		
Biological Monitoring	**Sample:** 1. Phenol in urine (total) (**not recommended**) 2. (t,t) Muconic acid in urine 3. Phenylmercapturic acid in urine (highly specific) 4. Benzene in blood (since unmetabolized higher ES)	**Sampling time:** ES (End of Shift)
	(t,t) Muconic acid in urine Phenylmercapturic acid in urine Benzene in blood Notation	**BEI (Biological exposure index):** 500 ug/g creatinine ES 25 ug/g creatinine ES 0.5 ug/100ml ES at OEL of 1ppm B
Biological Effect Monitoring-pathology based	Blood	Full blood count & differential counts, ALT, AST, ALP, GGT, Urea, creatinine, eGFR
	Urine	Dipstick

CADMIUM

Chemical Formula	**Cd**
CAS Number	**7440-43-9**
Occupational uses	Cadmium is a by-product of zinc and lead smelting. It is used in: • The electroplating industry and used in the production of rechargeable batteries (nickel cadmium). • In manufacturing plastics, coatings, and solar panels. • In alloys, cadmium vapour lamps, catalyst, ceramics, dyes, welding, engraving, glass colouring, metalizing, nuclear reactors, organic -based paints (spray painting), plantings, photometry, silver soldering, welding cadmium alloys. Cadmium is also present in tobacco products (smokers).
Toxicokinetics and toxicodynamics	**Route of entry: Inhalation, ingestion and dermal absorption** Inhalation is the primary route of Cd exposure. The absorbed amount is dependent on particle size and solubility of the Cd compound. Absorption from GIT occurs with clearance of Cd deposits in the lungs and from contaminated hands and food. With chronic low exposures half the body burden is stored in the liver and kidneys. Since urine remains the main elimination route, urinary Cd is used for biomonitoring of chronic exposures (reflects body burden). In newly-exposed workers, blood Cd (reflects recent exposure) is the biomonitoring test of choice as Cd is highly bound within the body and urinary Cd excretion is determined by the intensity of the integrated exposure (i.e., there is a lag time before urinary Cd correlates with exposure). Toxicity from Cd exposure occurs from protein-Cd adduct formation which results in conformational change in protein structure in sites of highest concentration i.e. alveoli of lungs and proximal tubules of kidney. Upon adsorption 80% of Cd is found in red blood cells. Cd is transported in plasma

	bound to albumin and to the renal tubules bound to metallothionein. The cellular effects include: 1. Disruption of cell cycle (includes DNA replication and repair and apoptosis) 2. Cd induced oxidative stress Cd and its compounds are classified as Group1 carcinogens in humans (lung and prostate).	
Clinical manifestations of occupational exposure	The renal damage induced by Cd typically results in slow onset proteinuria (develops over years). There is a loss of reabsorptive capacity for nutrients, vitamins, and minerals (such as zinc and copper bound to the metal binding protein metallothionein (MT), glucose, amino acids, phosphate, calcium, β2-MG, and retinol-binding protein (RBP). This is similar to Fanconi's syndrome. Inhalation of Cd fumes leads to nasal epithelial damage and pulmonary congestion that has a chronic emphysema-like presentation. Long-term exposure to high-dose cadmium causes Itai-itai disease (affecting mainly women) which is characterised by severely impaired tubular and glomerular function. In addition, there is generalised osteomalacia and osteoporosis that results in multiple bone fractures.	
Occupational exposures	IDLH STEL RHCA-OEL	9mg Cd/m^3 0.004 ppm (Respirable fraction) CARC
OCCUPATIONAL EXPOSURE.		
Biological Monitoring	**Sample:** 1. Cadmium in urine 2. Cadmium in blood	**Sample time:** NC (not critical)
	1. Cadmium urine: Notation 2. Cadmium blood: Notation	**BEI (Biological Exposure Index):** 5 ug/g creatinine sampling time NC B 5 ug/l collection time NC B
Biological Effect Monitoring pathology based	Blood	Liver, kidney, full blood count, urea, creatinine, EGFR
	Urine	Beta-2-microglobulin (to be interpreted with urine Cd levels): ≤300 ug/L is considered normal (annual biological monitoring recommended) >300 - ≤750 ug/L reflects an increased risk for renal tubular proteinuria (semiannual biological monitoring recommended until normalisation) >750 is considered a highly elevated risk for renal tubular proteinuria (quarterly biological monitoring recommended in addition to semiannual medical examinations and removal from workplace, either permanently or temporarily until normalisation of levels as per discretion of OMP). If urine/blood Cd within 90 days of follow-up remains elevated, and/or beta-2-microglobulin remains elevated, mandatory removal from work is recommended and the above recommendations will apply as per the discretion of the OMP i.e. temporary or permanent removal.

CARBON DISULPHIDE

Chemical Formula	**CS_2**	
CAS Number	**75-15-0**	
Occupational uses	Adhesives, chemical synthesis, disinfectants, extraction solvent, insecticides, lacquers and varnishes, perfumes, rayon, resins, rubber, fibres, cellophane, carbon tetrachloride and pesticides and to dissolve rubber in the production of tyres. Carbon disulphide is both a reagent and decomposition product in the manufacture of xanthates.	
Toxicokinetics and toxicodynamics	**Route of entry: Inhalation** Absorbed chiefly through the lungs, entering the bloodstream and being distributed through the body. CS_2 is rapidly and extensively absorbed upon inhalation. An equilibrium between exhaled air and inhaled air concentrations and a blood steady-state concentration is established within approximately 2 hours or less. Absorption after inhalation depends on various factors (exercise and body fat), but 80% has been measured initially, when the equilibrium is not reached. At an equilibrium 40% or less of the inhaled substance enters the circulation. It can also be absorbed through the skin and is absorbed from the gastrointestinal tract if swallowed. People not previously exposed absorb about 80% of inhaled vapour during the first 15 minutes, but the proportion falls to about 40% after 45 minutes and remains at that level for some time. In workers previously exposed, about 55% of inhaled vapour is absorbed during the first 15 minutes. Excretion through the lungs and urine (1%) is small. About 92% retained in the tissues and metabolised. The condensation product of the reaction of CS_2 with the amino acid cysteine, 2-thio-thiazolidine-4-carboxylic acid (TTCA), is excreted in the urine in concentrations directly related to the level of CS_2 exposure and so is a suitable parameter of internal exposure.	
Clinical manifestations of occupational exposure	**EXTREMELY TOXIC** **Acute:** Vesicant action on skin, headache, dizziness, nausea and vomiting, abdominal pains, flushing of skin, generalised pains, narcosis, conjunctivitis and keratitis. Can result in acute encephalopathy. **Chronic:** Slowing of pupillary light reaction blind spots and narrowing of vision, headache, dizziness, polyneuritis, peripheral neuropathy, motor and sensory, emotional disturbances, parkinsonism, vision, gastrointestinal, renal damage, anorexia, chronic gastritis, damage to liver, fatigue, anaemia, dermatitis, coronary artery disease (increases blood cholesterol levels). Other chronic manifestations include high blood pressure, spasmatogenic effect, menstrual disorders and spontaneous abortions, depression and suicidal tendencies.	
Occupational exposures	IDLH STEL OEL	500 ppm Not available 2 ppm
OCCUPATIONAL EXPOSURE		
Biological Monitoring	**Sample:** 2-thiothiazolidine – 4 carboxylic acid (TTCA) in urine	**Sample time:** ES (End of Shift)
	TTCA in urine Notation	**BEI (Biological exposure index) :** 0.5 mg/g creatinine ES B, Ns
Biological Effect Monitoring pathology based	Blood	Urea, creatinine, eGFR, electrolytes, ALT, AST, ALP, GGT, lipogram
	Urine	Dipstick

CARBON MONOXIDE

Chemical Formula	**CO**	
CAS number	**630-08-0**	
Occupational uses	Used in: • Liberation from emissions in enclosed places from exhaust fumes of internal combustion engines, metallurgic industry and foundries • Chemical industry for synthesis and emission as result of incomplete combustion • Liberation during acetylene welding; Carbon monoxide is also from enclosed areas as mines or tunnels as well as fire-damp explosions and liberation from industrial heating.	
Toxicokinetics and toxicodynamics	**Route of entry: Inhalation** Exposure mainly by inhalation. Pulmonary uptake of carbon monoxide (CO) accounts for all environmental CO absorption and occurs at the respiratory bronchioles, alveolar ducts and sacs. The exchange of CO between the air and the body depends on a number of physical (e.g. mass transfer and diffusion), as well as physiological factors (e.g. alveolar ventilation and cardiac output) which are controlled by environmental conditions, physical exertion and other processes. Absorbed and excreted unchanged via lungs. CO dissolved in blood is <1%. Carboxyhemoglobin is formed in blood and tissue anoxia. The blood is the largest reservoir for CO, where it reversibly binds to haemoglobin (Hb) to form carboxyhemoglobin (COHb). The affinity of CO for Hb in adult blood is ~218 times greater than of oxygen. CO is eliminated from tissues back into the blood (carried as COHb) and excreted predominantly via the lungs, unchanged, with a minor component undergoing oxidation to carbon dioxide. Exposure relationship: %HBCO = 0.16 x CO (ppm) x physical work. Non-smoker at CO exposure 30 and 50ppm x sedentary work = 5 to 8% HbCO. It is a non-cumulative toxin. Notation of B and Ns add text. Methylene chloride metabolite, ubiquitous pollutant and other diseases. Smoking up to 15% HbCO.	
Clinical manifestations of occupational exposure	Headache, nausea, dizziness, weakness, rapid breathing, unconsciousness and death (if in excess of 3500ppm). No cyanosis, usually a pink colour due to the presence of carboxyhemoglobin. Carboxyhemoglobin below 10%, with no signs or symptoms. Electrocardiograms may show sinus tachycardia and ST segment and T wave abnormalities. Electroencephalograms may show focal and diffuse epileptiform discharges, which later disappear. Exposure to CO can aggravate heart disease and arterial disease and lead to chest pain in pre-existing cardiac disease.	
Occupational exposures	IDLH STEL OEL	1200 ppm Not available 50 ppm
OCCUPATIONAL EXPOSURE		
Biological Monitoring	**Sample:** Carboxy Hb in blood* * Non smokers	**Sampling Time:** ES (End of Shift)
	Carboxy Hb in blood Notation	**BEI (Biological exposure index):** 3.5% haemoglobin B, Ns
Biological Effect Monitoring pathology based	Blood	Full blood count and differential

CHROME VI

Chemical Formula	**Cr (VI)**	
CAS Number	-	
Occupational uses	Cr (VI) is used for: • Manufacturing of stainless steel • In chrome plating; • In tanning leather • As a pigment in paints and dye for printing and textile manufacture • As a cleaning solution and as an anticorrosive in cooling systems.	
Toxicokinetics and toxicodynamics	**Inhalation** of dusts, mists or fumes, and dermal contact are the main routes of entry. The hexavalent form is the toxic form. The trivalent form requires exposure to high temperatures in an oxidising environment to convert to the hexavalent form (e.g. in electroplating). The form of Cr influences the distribution. Cr III is poorly absorbed by all routes as it is insoluble. Cr VI is lipid soluble and at physiologic pH, Cr VI forms CrO_4^{2-} (chromates) which readily crosses cell membranes. Absorption of inhaled Cr VI is dependent on the physical and chemical properties i.e. size (< 5um absorbed into blood) and solubility (the presence of the reduced Cr III state). Absorbed Cr is distributed in all tissues with highest levels found in kidney, liver and bone. When Cr VI enters the cell it is rapidly reduced to nontoxic Cr III. Hence, Cr VI is not used for biological monitoring (the presence of Cr in erythrocytes suggests exposure to Cr VI in the past 120 days as Cr VI crosses cell membranes; also provides information on the intensity of exposure). Urine Cr assesses exposure to total Cr, mainly exposure to water soluble hexavalent Cr compounds. Cr accumulates in the body. Urine Cr concentration can therefore reflect both recent and past exposures. Intracellularly, Cr VI is reduced to reactive intermediates. These produce free radicals and oxidise DNA and therefore apoptosis.	
Clinical manifestations of occupational exposure	The symptomatic presentation of Cr exposure is dependent on route and dose of exposure. These include dermatitis, burns and ulcers on contact with the skin. Inhalation of Cr (VI) vapours causes respiratory irritation, erosion of the nasal epithelium with ulceration, and tissue damage to the throat and lungs (including squamous cell carcinoma). IARC classified Cr VI as a human carcinogen (A1) causing lung, nose and nasal sinus cancers (suspected stomach and laryngeal cancers).	
Occupational exposures(MSDS)	IDLH RHCA-STEL Metallic Cr Trivalent Cr Hexavalent Cr RHCA-OEL Metallic Cr Trivalent Cr Hexavalent Cr	250mg Cr/m^3 Not available Not available 0.0004 mg/m^3 (Inhalable fraction) 1 mg/m^3 (Inhalable fraction) 0.006 mg/m^3 (Inhalable fraction), CARC, RSEN 0.0004 mg/m^3 (Inhalable fraction), CARC, RSEN
OCCUPATIONAL EXPOSURE		
Biological Monitoring	**Sample:** 1. Total Chromium in urine 2. Chromium whole blood	**Sampling time:** ES (End of Shift), EW(End of Work week)
	1. Total Cr in urine: 2. Cr in whole blood:	**BEI (Biological exposure index):** 25 ug/l at ES,EW * Toxic levels not established
Biological Effect Monitoring pathology based	Blood	Full blood count, liver function, urea, creatinine, eGFR
	Urine	Albumin and protein excretion

COBALT

Chemical Formula	**Co**	
CAS Number	**7440-48-4**	
Occupational uses	Cobalt is used in hard heat resistant metal alloys, magnets, pigments, paints, grinding and cutting tools, surgical implants, batteries, catalysts, batteries, welding and in radioactive isotopes.	
Toxicokinetics and toxicodynamics	**Route of entry**: **Inhalation, skin and gastro-intestinal tract** Cobalt in the form of cyanocobalamin (an essential micronutrient and cofactor in vitamin B12) is nontoxic. The co-exposure with tungsten (and other metal exposures) is considered more toxic than Co exposure alone. Cobalt is mainly absorbed by inhalation of dust, fume, ingestion and dermal absorption. With chronic exposure the thyroid gland, lungs, immune system, and kidneys are affected. The mechanisms of toxicity include: ● Binding of sulfhydryl groups resulting in enzyme inhibition ● Intracellular calcium homeostatic disruption ● Generation of reactive oxygen species Inhaled small particle sized Co partitions to the lower respiratory tract where they are dissolved into the bloodstream or phagocytosed and translocated. Insoluble particles are cleared by phagocytosis or mucociliary transport and thus have a low systemic absorption. Co is eliminated by (a) rapid phase lasting a few hours to a few days and (b) slow phase with half-lives ranging from months to years. Soluble cobalt that is ingested is mainly transported via blood to the liver and kidneys and excreted in urine. Faecal elimination is the primary method of excretion of insoluble cobalt. Excretion after dermal exposure is in urine.	
Clinical manifestations of occupational exposure	Chronic exposure can result in (a) pulmonary syndrome i.e. cough, shortness of breath, respiratory hypersensitivity, dyspnoea, decreased pulmonary function (parenchymal lesions known as " hard metal disease" that can progress to severe alveolitis and end-stage pulmonary fibrosis), (b) skin irritation and contact dermatitis, (c) GIT irritation with nausea and vomiting, (d) cardiomyopathy due to accumulation of Co in the myocardium, (e) haematologic disorders, and (f) thyroid abnormalities. Cobalt has an effect on the thyroid function thought to result from Co blocking iodine uptake into the thyroid, causing functional iodine deficiency with increased TSH stimulation and thyroid hyperplasia.	
Occupational exposures(MSDS)	IDLH STEL OEL	20mg Co/m^3 0.04 ppm (Inhalable fraction) CARC,RSEN Not available
OCCUPATIONAL EXPOSURE		
Biological Monitoring	**Sample:** 1. Total Cobalt in urine 2. Cobalt whole blood	**Sampling time:** ES (End of Shift), EWW (End of Work week)
	1. Total Cobalt in urine Notation 2. Cobalt whole blood	**BEI (Biological Exposure Index) :** 15 ug/l at ES, EWW Ns When the average exposure levels are 0.1 and 0.5 mg/m3, the estimated blood levels are 10 and 25ug/L respectively. *Biological Exposure Index (BEI) is UNKNOWN
Biological Effect Monitoring pathology based	Blood	Full blood count, Creatinine, Urea, eGFR, thyroid function test
	Urine	Albumin and protein excretion

CYANIDE

Chemical Formula	**CN-(cyano functional group)**	
CAS number	**57-12-5**	
Occupational uses	Mining (extracting gold and silver ores), photo developing, electroplating, plastic manufacturing. Used in pesticides and fumigants. Some industrial processes such as iron and steel production, chemical industries and wastewater treatment can create cyanides.	
Toxicokinetics and toxicodynamics	**Route of entry: Inhalation, ingestion and slow dermal absorption** Potassium cyanide and sodium cyanide can be considered as a chemical category, along with hydrogen cyanide (HCN) and acetone cyanohydrin (ACH, also known as 2-hydroxy-2-methylpropanenitrile), based on structural similarity, similar physico-chemical properties and common breakdown/metabolic products (CN- anion) in physical and biological systems. Particular attention is paid to the dissociation constant of HCN, 9.36 at 20°C; in normal environmental and physiological conditions, the CN- anion will be hydrated to HCN, regardless of its origin in NaCN, KCN, ACH or HCN. Cyanide gas and salts are rapidly absorbed following ingestion or inhalation. Skin absorption is much slower. Absorbed cyanide is rapidly distributed throughout the body. In small doses, cyanide is metabolised to thiocyanate, which is less harmful and excreted in urine. In large doses the body's ability to convert cyanide to thiocyanate is overwhelmed, as cyanide combines with the ferric ion, preventing electron transport in the cytochrome system. This brings ATP production and oxidative metabolism to a halt, affecting the heart and CNS. Hydrogen cyanide will distribute in the body to blood (erythrocytes), muscles and other organs. Metabolism occurs in muscles and organs mainly via rhodanese, forming thiocyanate which is excreted in the urine. Saturation of the enzyme results in build-up of HCN and acute toxicity. Chronic toxicity involves competition by thiocyanate with iodine transfer into the thyroid, with the consequence of increased secretion of thyroid stimulating hormone (TSH) and development of goitre. Smokers and non-smokers should be differentiated by normal values. Toxic levels: - conscious, flushed + tachycardia : 500 - 1000 ug/l - stupor + agitation : 1000 - 2500 ug/l - coma + potentially lethal : >2500 ug/l	
Clinical manifestations of occupational exposure	Cyanide is extremely toxic and high exposure (100mg/m^3) can cause death. Exposure to lower concentrations (6-49mg/m^3) will cause weakness, headache, nausea, increased rate of respiration and eye and skin irritation. Primarily affects the central nervous system. Cardiovascular and respiratory effects are also noted.	
Occupational exposures	IDLH STEL OEL	25mg/m^3 as CN 4.7ppm HCNM, 5mg/m^3 CYANIDE SALTS Not available
OCCUPATIONAL EXPOSURE		
Biological Monitoring	**Sample:** 1. Thiocyanate in urine 2. Thiocyanate in serum 3. Cyanide in blood	**Sampling Time:** ES (End of Shift) ES (End of Shift) After acute exposure
	1. Thiocyanate in urine 2. Thiocyanate in serum 3. Cyanide in blood	**Reference limits:** Non-smokers : 0.66- 2.7 mg/l Smokers : 4.70 - 11.3 mg/l Non-smokers : 1 - 4 mg/l Smokers : 3 - 12 mg/l Usually asymptomatic : <200 ug/l
	Blood	TSH, FT4, urea, creatinine and eGFR

Biological Effect Monitoring pathology based	Urine	Dipstick, albumin or protein excretion (as per OMP)

DICHLOROMETHANE/METHYLENE CHLORIDE

Chemical Formula	**CH_2Cl_2**	
CAS Number	**75-09-2**	
Occupational uses	Solvent and extracting agent, paint stripper, blowing agent for polyurethane foams and degreasing, production of polycarbonate resins. Also used in film processing and in ink formulations. Dichloromethane can decompose and emit highly toxic fumes of phosgene and chlorine.	
Toxicokinetics and toxicodynamics	**Route of entry**: Highly volatile, is rapidly absorbed following **inhalation, skin absorption, eye contact and ingestion.** Dichloromethane is first metabolised to carbon monoxide. This can result in elevated levels of Carboxyhemoglobin (COHb) and potentially lead to carbon monoxide poisoning. The combined effect of smoking and exposure to Dichloromethane can produce an additive increase in COHb levels.	
Clinical manifestations of occupational exposure	Dichloromethane is a highly volatile liquid for which vapour inhalation is the most likely exposure route in occupational settings. It is characterised by moderate acute toxicity by oral route (Acute oral toxicity, Category 4) and low acute toxicity via inhalation and dermal exposures. The effects of acute toxicity of dichloromethane include CNS depression, formation of carboxyhemoglobin (CO-Hb), as well as effects on liver, kidney and haematological parameters.	
Occupational exposure	IDLH STEL OEL 8 hour TLV	100 ppm Not available 4 ppm/50ppm
OCCUPATIONAL EXPOSURE		
Biological Monitoring	**Sample:** Urine	**Sampling time:** ES (End of Shift)
	Dichloromethane urine Notation	**BEI (Biological exposure index):** 0,3 mg/L ES Sq
Biological Effect Monitoring pathology based	Blood	COHb, liver function, urea, creatinine, eGFR
	Urine	Dipstick

nn-DIMETHYLFORMAMIDE

Chemical Formula	**$HCON(CH_3)$ /C_3H_7NO**	
CAS Number	**68-12-2**	
Occupational uses	Solvents for liquids and gases, including those used in artificial leather production. Used in the synthesis of organic compounds, manufacture of polyacrylic fibres, butadiene, pharmaceuticals, dyes, petroleum products and other organic chemicals. Also used in adhesives, pesticides, epoxy formulations, perfumes, fragrances and nonmetallic mineral products.	
Toxicokinetics and toxicodynamics	**Route of entry**: **Inhalation, Ingestion and dermal absorption** The following metabolites of DMF are excreted in urine: N-methyl-N-hydroxyethyl formamide (DMF-OH or, alternatively, HMMF), N-methylformamide, formamide, mercapturic acid. Elimination through urine is biphasic with half-lives of 3-hours and 7-hours respectively. Monophasic elimination with a 4-hour half-life has also been reported. Notation: Sq	
Clinical manifestations of occupational exposure	Inhalation of vapour may cause colicky abdominal pain, appetite loss, nausea, vomiting, constipation, diarrhoea, nervous agitation, increased blood pressure, liver and kidney injury. Results in liver toxicity, presenting with jaundice, altered liver enzymes and alcohol intolerance. Skin contact may cause similar effects as inhalation. In addition, mild skin irritation, drying and cracking may occur. Exposure followed by ingestion of alcohol may cause facial flushing and alcohol intolerance. (Porphyric symptomatology)	
Occupational exposures	IDLH STEL OEL	50 ppm Not available 20 ppm/5ppm CARC,SKIN
OCCUPATIONAL EXPOSURE		
Biological Monitoring	**Sample:** N-Methylformamide in urine	**Sampling Time:** ES (End of Shift)
	N-Methylformamide in urine Acetyl-S-(N-methylcarbamoyl) cysteine urine Notation	**Biological exposure index (BEI):** 15 mg/L ES Prior to last shift of work week 40 mg/L Sq
Biological Effect Monitoring pathology based	Blood	ALT, AST, GGT, ALP, full blood count, urea, creatinine and eGFR
	Urine	Dipstick

ETHYL BENZENE

Chemical Formula	$C_6H_5C_2H_5$ /C_8H_{10}	
CAS Number	**100-41-4**	
Occupational uses	Chemical intermediate in manufacture of styrene and starting product for a wide variety of plastics, synthetic rubber and latex products based on styrene. Used as a solvent, polymerisation agent and cross linking and raw material for production of cellulose acetate, acetophenone, diethyl benzene and anthraquinones. Ethyl benzene is a minor component of gasoline and aviation fuels and is used in electroplating aluminium.	
Toxicokinetics and toxicodynamics	**Route of entry: Inhalation, ingestion and dermal absorption** Ethylbenzene is rapidly distributed through the body. In humans exposed via inhalation, the major metabolites are mandelic acid (approximately 70 % of the absorbed dose) and phenylglyoxylic acid (approximately 59 % of the absorbed dose), which are excreted in the urine. Excretion is almost complete within 24 hrs after exposure, with only about 0.2% of absorbed dose remaining in the body within 42 hours after inhalation exposure. In man, highest urinary concentrations of mandelic acid and phenylglyoxylic acid occurred 7 hours after exposure, the biological half-life of both metabolites is 4-7 hours.	
Clinical manifestations of occupational exposure	Vapour or mist can irritate the nose and throat. Inhaled ethyl benzene may cause nausea, headache, vomiting and other symptoms of central nervous system depression. Human volunteers exposed at 85 ppm for 8 hours reported no adverse health effects. At a level of 100 ppm mild vertigo sleepiness and headache were reported. Exposure to 1000-2000 ppm for 6 minutes caused fatigue and increasing vertigo, chest constriction and dizziness. Slight skin irritation may occur in contact with the liquid. At 200 ppm level a transient eye irritation occurs and, at 1000 ppm, there is irritation with tearing but some eye tolerance develops. At level 2000 ppm immediate irritation and tearing occurs. No human information is available on ingestion. Long-term exposure may cause kidney, blood and testicular effects. Similarly, to other hydrocarbons, ethyl benzene vapour may cause central nervous system effects such as headache, memory loss, fatigue, etc. Skin prolonged and repeated contact may cause dermatitis, reddening of skin, hair loss and chapped appearance due to its de-fattening action.	
Occupational exposures	IDLH STEL OEL	800 ppm Not available 40 ppm
OCCUPATIONAL EXPOSURE:		
Biological Monitoring	**Sample:** 1. Mandelic acid (MA) and Phenylglyoxylic acid (PGA) in urine 2. Ethyl benzene in blood	**Sampling time:** 1. ES (End of Shift) 2. DS (During Shift)
	1. Sum of MA and PGA in urine 2. Ethyl benzene blood Notation	**BEI (Biological exposure index):** 150 mg/g creatinine ES 0.150 mg/100ml DS Ns
Biological Effect Monitoring pathology based	Blood	Full blood count, liver function tests, urea, creatinine and eGFR
	Urine	Dipstick

FLUORIDE

Chemical Formula	**F**	
CAS Number	**16984-48-8**	
Occupational uses	Mining of minerals, production of aluminium and steel, brick and refractory, fluxes in welding, hydrofluoric acid and fluorine production and uses.	
Toxicokinetics and toxicodynamics	Metallic fluorides are solids with variable solubilities in water i.e. the salts of monovalent metals are fairly soluble, the salts of divalent metals are sparingly soluble, and hydrogen fluoride (HF) is a reactive gas that readily dissolves in water, reacts with glass and is corrosive. Pulmonary and GIT absorption depends on the solubility of the compound and particle size. Soluble fluorides are readily absorbed following inhalation (.90%) and insoluble fluorides are absorbed to a lesser extent. Following absorption by the inhalation route, urinary fluoride levels increase within two hours of exposure and remain elevated for 2-4 hours after exposure has ceased. Excretion of fluoride from the body is predominantly via the urine (40% of absorbed fraction). The remainder is deposited in the mineral matrix of bone. The half-life of fluoride in skeletal tissues is long (8-20 years) compared with the half-life in soft tissue and plasma (2-9 hours). Dietary sources of fluoride and the long-term retention of fluoride in skeletal tissues cause elevated, background levels of fluoride in urine, There is significant inter-individual variability, particularly in those subjects who have worked previously with fluoride compounds. Pre-shift urine sampling is therefore recommended.	
Clinical manifestations of occupational exposure	The primary route of fluoride exposure is via inhalation. Chronic effects from inhalation of fluorides may include fluorosis and osteosclerosis, brittle bones, joint stiffness and weakness, weight loss, malaise, anaemia, and discolouration of teeth and dental mottling. Chronic exposure to high levels of either hydrogen fluoride or fluorine may result in pulmonary oedema, tracheobronchitis, haemorrhagic alveolitis, adult respiratory disease syndrome pulmonary fibrosis. Skin Irritation can occur i.e. itchiness and rash, and burns. Eye contact causes severe burning and/or irritation. Hydrogen fluoride may cause deep seated burns of the eyes. Cardiac arrhythmias occur from hyperkalaemia and hypocalaemia i.e. fluorides may inhibit oxygen binding and blood clotting, decreases erythrocyte glycolysis, and results in the efflux of potassium from red blood cells. This often leads to hypocalcemia (fluoride has a high affinity for calcium). The CNS effect may include headaches and tremor. Renal injury, thyroid injury, anaemia, hypersensitivity and menstrual irregularities may occur.	
Occupational exposures (MSDS)	IDLH STEL OEL 8hr TWA	250 mg F/m^3 5 mg/m^3
OCCUPATIONAL EXPOSURE		
Biological Monitoring	**Sample:** Fluoride in urine	**Sampling time:** PS (Prior to shift) or ES (End of Shift)
	Fluoride in urine Notation	**BEI (Biological Exposure Index):** 2 mg/l at PS and 3 mg/l at ES B, Ns
Biological Effect Monitoring pathology based	Blood	Serum urea, creatinine, eGFR and electrolytes, calcium, magnesium, phosphate
	Urine	Albumin or Proteinuria

FORMALDEHYDE

Chemical Formula	**CH_2O**
CAS Number	**50-00-0**
Occupational uses	Formaldehyde is synthesised by the oxidation of methanol. Commonly used as: • A preservative in medical laboratories and mortuaries for embalming, formaldehyde is also found in many products such as chemicals, particle board, household products, glues, permanent press fabrics, paper product and coatings. • For manufacturing of plastics, resins, and urea-formaldehyde foam insulation. • An industrial fungicide, germicide and disinfectant. • Other industries such as photography, dyeing, rubber, agriculture, fertiliser manufacture, construction (plywood adhesives), artificial silk, explosives, tanning, precious metal recovery, sewage treatment, pharmaceuticals, food, and textiles. Paraformaldehyde is the most common commercial polymer of formaldehyde containing a mixture of products with varying degrees of polymerisation. Formalin/formol is an aqueous formaldehyde preparation.
Toxicokinetics and toxicodynamics	**Route of entry: Inhalation, ingestion and dermal absorption** Formaldehyde is an essential metabolic intermediate in both humans and animals. It is endogenously formed from serine, glycine, methionine and choline and is produced in the demethylation of N-, O-, and S-methyl compounds. Formaldehyde is an essential intermediate in the biosynthesis of purines, thymidine and several amino acids (IARC, 1995; summary reviews). The mean endogenous concentration of free and reversible bound formaldehyde in blood of unexposed humans was 2.61 µg/g blood (range 2.05- 3.09 µg/g). Formaldehyde is rapidly metabolised and storage is not a factor in its toxicity. It is quickly broken down in the air, generally within hours. Formaldehyde also occurs naturally in the environment. Formaldehyde can be reduced to methanol or oxidised in the cytosol or mitochondria to formate/formic acid. Further oxidation of formate to CO2 occurs. Most inhaled formaldehyde is broken down by the cells lining the mouth, nose, throat, and airways, so that less than a third is absorbed into the blood. Given the rapid conversion of formaldehyde to formate/formic acid and subsequent incorporation into naturally occurring cellular constituents, excretion does not appear to be a factor in the toxicity of formaldehyde. The metabolism of formaldehyde to formate/formic acid takes place in all of the body's tissues as a consequence of endogenous formation of formaldehyde. Exogenous formaldehyde enters this pathway and is eliminated from the body as metabolites, primarily CO2 Formaldehyde is also a component of tobacco smoke and both smokers and those breathing second-hand smoke are exposed to higher levels.
Clinical manifestations of occupational exposure	Formaldehyde is highly irritating to the upper respiratory tract and eyes. It is readily absorbed from the lungs. Concentrations of 0.5 to 2.0 ppm may irritate the eyes, nose, and throat of some individuals. Concentrations of 3 to 5 ppm may also cause tearing of the eyes. Concentrations of 10 to 20 ppm cause difficulty in breathing, burning of the nose and throat, cough, and heavy tearing of the eyes, while 25 to 30 ppm causes severe respiratory tract injury leading to pulmonary edema and pneumonitis. Asthmatic symptoms may occur due to allergic sensitivity to formaldehyde, even at very low concentrations. A concentration of 100 ppm is immediately dangerous to life and health, producing a feeling of restricted chest, headache, and palpitations and, in extreme cases, death due to oedema or spasm of the glottis. Formalin is a severe skin irritant (reacting readily with tissue proteins and promotes allergic reactions) and a sensitiser. Once sensitised, the allergic response may follow contact with only very small quantities. Contact with formalin causes white discolouration, smarting, drying, cracking (allergic contact dermatitis), and scaling.

	Prolonged and repeated contact can cause numbness and a hardening or tanning of the skin. There have been reports of both inflammatory and allergic dermatitis, including nail dystrophy due to direct contact with solutions, solids or resins containing free formaldehyde. Kidney injury may occur in excessive and repeated exposure. Formaldehyde solutions splashed in the eye can cause injuries ranging from transient discomfort to severe, permanent corneal clouding and loss of vision. Ingestion of formaldehyde causes severe irritation and inflammation of the mouth, throat, and stomach. Severe stomach pains will follow ingestion with possible loss of consciousness and death. Ingestion of diluted formaldehyde solutions (0.03-0.04%) may cause discomfort in the stomach and pharynx. Carcinogenicity: has been associated with nasopharynx and nasal sinuses (IARC-Group 1B i.e. probable human carcinogen and ACGIH-A2); epidemiological studies suggests an increased risk of myeloid leukaemia.	
Occupational exposures	IDLH STEL OEL	20 ppm 0.6 ppm 0.2 ppm CARC, DSEN, RSEN
OCCUPATIONAL EXPOSURE		
Biological Monitoring	**Sample:** Formic acid in urine	**Sampling time:**
	Formic acid: creatinine	**Biological exposure index (BEI):** Cannot be adequately assessed-too many variables **Not industrial exposed (NIE):** 23 mg/g creatinine
Biological Effect Monitoring pathology based	Blood	Full blood count

FURFURAL/2-furaldehyde

Chemical Formula	$C_5H_4O_2$	
CAS Number	**98-01-1**	
Occupational uses	Used as: • Solvents for oils, synthetic and natural resins • Cellulose, derivatives, dyes, polymers and other organic chemicals • Intermediates in the production of plastics and insecticides/pesticides and vulcanisation accelerators in the rubber industry.	
Toxicokinetics and toxicodynamics	**Route of entry: Inhalation and dermal absorption** Furfural is rapidly absorbed after inhalation or dermal absorption. It is detoxified by oxidation and conjugates to amino acids. The biological half-life of absorbed furfural is 2-2.5 h. Furfural is metabolised such that approximately 97% (range, 93-100%) is oxidised to 2-furoic acid and excreted as the glycine conjugate, 0.5-5% is excreted as furanacrylic acid and less than 1% is exhaled unchanged.	
Clinical manifestations of occupational exposure	Exposure is usually to furfural vapours, but the hazard of poisoning by furfural and its derivatives is limited in view of the low volatility of these products at low temperatures. Furfural vapours are strong skin, eye and mucous membrane irritants, and can lead to sensitisation and pulmonary oedema. Chronic exposure can cause congestion in the liver, kidney, lungs and brain and be associated with hepatic and renal lesions. Prolonged exposure may further present with nervous disorders such as tremors and dizziness. Dermatitis is caused by skin sensitisation with chronic exposure. Also, presents with loss of sense of taste, and numbness of the tongue. Deaths have occurred due to respiratory paralysis and a depressant action on the CNS and heart has been observed.	
Occupational exposures	IDLH STEL OEL	100 ppm Not available 0.4 ppm
OCCUPATIONAL EXPOSURE		
Biological Monitoring	**Sample:** Total furoic acid in urine	**Sampling time:** ES (End of Shift)
	Total furoic acid in urine Notation	**(BEI) Biological Exposure Index:** 200 mg/L ES Ns
Biological Effect Monitoring pathology based	Blood	Kidney function: Urea, creatinine, eGFR Liver function: ALT, AST, GGT, ALP
	Urine	Dipstick

n-HEXANE

Chemical Formula	**$CH_3 (CH_2)_4 CH_3$**	
CAS Number	**110-54-3**	
Occupational uses	It is a solvent used in: • The chemical and food industries • In glues, cements and adhesives for production of footwear and furniture • Car tyre retreads • Extraction of vegetable oils • Food additive Common in paints and thinners, as well as being a component of petroleum and petroleum distillates (like solvents and grease removers).	
Toxicokinetics and toxicodynamics	**Route of entry**: **Mainly inhalation** It accumulates in fat tissue, decreasing with a half-life of 64 hours after exposure has ended. Skin absorption may raise biological levels significantly above those reached during inhalation exposure to the occupational exposure value. Absorption through ingestion is likely to be rapid and complete. Hexane is rapidly eliminated in exhaled air, 10% of inhaled n-hexane is immediately eliminated unchanged through the lungs. The remainder of absorbed n-hexane is metabolised in the liver. Absorbed hexane is metabolised to 2,5 hexanedione, 2,5-dimethylfuran and gamma-valerolactone in urine. The urinary elimination half-life of 2,5-HD is 14 hours. Elimination from blood is biphasic, with half-lives of 12 minutes and 120 minutes respectively.	
Clinical manifestations of occupational exposure	At high levels of exposure, hexane acts as a narcotic. It is an eye irritant, and may be irritating to the respiratory tract. Inhalation of acute doses can cause drowsiness, fatigue, vertigo, loss of appetite, muscle weakness, paraesthesias, cold pulsation in extremities, polyneuropathy and blurred vision.	
Occupational exposures	IDLH STEL OEL	1100 ppm Not available 100 ppm
OCCUPATIONAL EXPOSURE		
Biological Monitoring	**Sample:** 2.5 Hexanedione	**Sampling time:** ES, EWW (end of shift,end of workweek)
	Reference limits: 2.5 Hexanedione Notation	**BEI (Biological Exposure Index):** 0.4 mg/l ES, EWW None
Biological Effect Monitoring pathology based	Blood	Urea, creatinine and eGFR Confirmation of exposure – n-hexane in blood
	Urine	Dipstick

ISOCYANATES: 1,6-Hexamethylene diisocyanate (HDI) and Toluene diisocyanate-2,4 (TDI) or as a mixture of isomers

1,6-Hexamethylene diisocyanate (HDI)

Chemical formula	**$OCN(CH_2)_6NCO$**	
CAS Number	**822-060-0**	
Occupational uses	The most commonly use is in alcohol–containing hydroxyl groups of compounds used to produce polyurethane polymers. Used in: • The manufacture of surface diisocyanate polyol surface coatings and finishes • Polyurethane paints • Thermal and electrical insulation • Polyurethane form, elastoplastics, adhesives and sealants.	
Toxicokinetics and toxicodynamics	**Route of entry: Inhalation and dermal absorption** HDI is a clear colourless liquid with a low vapour pressure under normal ambient conditions (0.007hPa at 20°C), therefore inhalation exposure to the vapour is expected to be low. In controlled studies in human volunteers 1,6-hexamethylene diamine (HDA) could be detected in the urine of HDI exposed persons (inhalation exposure) after acid hydrolysis as a biomarker for excretion of HDI or HDI-metabolites. In a study with three volunteers each exposed to 0.012, 0.020 and 0.022 mg/m^3 for 2 hours (2 days each between the exposures), the average urinary elimination half-time for HDA in hydrolysed urine was 2.5 hours. No HDA could be found in hydrolysed plasma during the exposure days (before and half an hour after exposure. Under physiological conditions it is expected that HDI decomposes in the GI tract mainly into HDA and carbon dioxide. Therefore intestinal absorption of HDI subsequent to oral ingestion may be limited. Due to a molecular weight of 168.2 g/mol and a calculated log Pow of 3.2, dermal absorption is conceivable. Furthermore, after contact of HDI with the surface moisture of the skin, hydrolysis to HDA and carbon dioxide can be expected as well as reaction with nucleophiles like NH- or SH-groups. HDI revealed corrosive properties to the skin. Damage to the skin surface may enhance penetration of HDI and/or HDA. The assumption of a dermal absorption is confirmed by the data on acute dermal toxicity and skin sensitisation.	
Clinical manifestations of occupational exposure	The health effects include occupational asthma, skin irritation (dermatitis), irritation to the mucous membranes, eyes, nose, and throat, gastrointestinal irritation, chemical bronchitis and pneumonitis. Dermal sensitivity may result in a rash, itching, hives, blistering and swelling of the extremities. Continued over-exposure may lead to pulmonary sensitisation/"isocyanate asthma" which may include coughing, tightness of the chest, and shortness of breath. Although symptoms may improve after removal of exposure, acute asthma attacks may occur after renewed exposure even when exposure is small and brief.	
Occupational exposure	IDLH STEL OEL 8 hour TLV	Not available Not available 0.01ppm
OCCUPATIONAL EXPOSURE		
Biological Monitoring	**Sample:** 1.1.6-hexamethylene diisocyanate (HDI) urine	**Sampling time:** ES (End of Shift)
	1. HDI Notation 2. Serum IgE HDI	**BEI (Biological exposure index):** 15 ug/g creatinine ES Ns <0.35 IU/ml

Biological Effect Monitoring pathology based	Serum IgE HDI

2,4-and 2,6-Toluene diisocyanate(TDI) & 4,4-Methylene diphenyl isocyanate (MDI)

Chemical Formula	**$CH_3C_6H(NCO)_2$ & $CH_2(C_6H_4NCO)_2$**	
CAS Number	**584-84-9 & 91-08-7**	
Occupational uses	The most common use is in alcohol-containing hydroxyl groups of compounds used to produce polyurethane polymers. Used in: • The manufacture of surface diisocyanate polyol surface coatings and finishes • Polyurethane paints • Thermal and electrical insulation • Polyurethane form, elasto plastics, adhesives and sealants.	
Toxicokinetics and toxicodynamics	**Route of entry: Inhalation ingestion and dermal absorption** After oral administration of TDI, physicochemical properties of the substance leads to the hydrolysis of TDA or formation of polyurea in the stomach. It is the TDA which is subsequently absorbed and metabolised. This does not happen by inhalation as supported by the data from several studies. While the chemical reactivity of TDI precludes the free isocyanate entering the systemic circulation from the lung, it has been postulated that TDI will conjugate or react with biological molecules in the lung which then enter the systemic circulation. In humans there are several reports measuring either plasma or haemoglobin adducts of TDI, or urinary metabolites. For urinary biomarkers, methodology uses acid or base hydrolysis to release TDA which is subsequently quantified. Free TDA has not been detected in urine of humans exposed to atmospheric TDI. While a precise relationship between inhalation exposure and biomarker is not established, it is clear that urinary excretion reflects very recent exposures to TDI, while blood biomarkers may reflect exposures over the proceeding few weeks. The details of MDI metabolism in man are unknown.	
Clinical manifestations of occupational exposure	The health effects of TDI exposure include occupational asthma, skin irritation (dermatitis), irritation to the mucous membranes, eyes, nose, and throat, gastrointestinal irritation, chemical bronchitis and pneumonitis. Dermal sensitivity may result in a rash, itching, hives, blistering and swelling of the extremities. Continued over-exposure may lead to pulmonary sensitisation/"isocyanate asthma" which may include coughing, tightness of the chest and shortness of breath. Although symptoms may improve after removal of exposure, acute asthma attacks may occur after renewed exposure even when exposure is small and brief. TDI can cause severe eye irritation with permanent damage if untreated. Occupational exposure to 4,4-methylene diphenyl isocyanate (MDI) affects mainly the respiratory tract; the substance causes irritation of the eyes and respiratory passages and has adverse effects on lung function. This must be distinguished from the bronchial or alveolar hypersensitivity caused by the substance – with or without demonstrated effects on immunological parameters (sensitisation). Dermal sensitisation is unusual.	
Occupational exposure	**IDLH** TDI MDI **STEL** TDI MDI **OEL** TDI	 2.5 ppm 0.075 0,0 ppm (Inhalable Fraction and Vapour) 0,002 ppm (Inhalable Fraction and Vapour) 0.01ppm (Inhalable Fraction and Vapour)

	MDI	Not available
OCCUPATIONAL EXPOSURE		
Biological Monitoring	**Sample:** 1. Toluenediamine: creatinine 2. Serum IgE TDI 3. Serum IgE MDI	**Sampling time:** ES (End of Shift)
	1. Toluenediamine: creatinine 2. Serum IgE TDI 3. Serum IgE MDI	**BEI (Biological exposure index):** 5 ug/g creatinine (ES) <0.35 IU/ml <0.35 IU/ml
Biological Effect Monitoring pathology based	Blood Serum IgE TDI Serum IgE MDI	

LEAD

Chemical Formula	**Pb**
CAS Number	**7439-92-1**
Occupational uses	Used in: • Battery manufacturing • Chemical industry • Construction workers • Demolition workers • Foundry workers • Jewellers • Lead miners • Lead smelters and refiners • Pigment manufacturing • Pipe fitters • Plastics industry • Pottery workers • Printers • Radiator repair • Rubber industry • Soldering of lead products • Solid waste production • Stained-glass makers • Welders • Ammunition procedures and Firing-range instructors • Anti-knock additives in petrol (tetra alkyl lead) [Note that there are several non-occupational exposures to Pb such as pottery, working with lead windows, renovations to old houses, household soldering or welding - especially without appropriate personal protection, and environmental exposure i.e. contaminated foods, air, water, soil, smoking, home distilled alcohol, etc.]
Toxicokinetics and toxicodynamics	**Routes of entry: Inhalation, ingestion and dermal absorption** Inorganic lead is the most common form of lead found in the workplace. It can be absorbed as a fume or as dust. Fumes are formed when lead is heated to a temperature greater than 560°C. Tetra alkyl lead and tetra methyl lead are liquid compounds of lead and in some countries are still used as anti-knock agents. The primary route of occupational exposure is by inhalation of fumes and dust. Absorption is dependent on particle size and solubility. The larger particles (>2.5um) that are deposited in the airways are transported by mucociliary action to the GIT and excreted in the stool. The degree of lead absorption is increased considerably with fasting or in persons whose diet is deficient in calcium, iron, phosphorus or zinc. Pb in the blood is

	bound to the erythrocytes and has several erythropoietic effects i.e. decreased haeme biosynthesis by inhibiting delta-aminolevulinic acid dehydratase and ferrochelatase activity. Erythrocyte protoporphyrin and serum delta-aminolevulinic acid dehydratase concentration provide a good indication of Pb exposure. Pb readily forms covalent bonds with the sulfhydryl group of cysteine in protein molecules (hence hair is a good biological matrix for Pb exposure). Within the nervous system, Pb binding to proteins leads to tertiary structural changes resulting in the proteins becoming labile. This change can also result in proteins becoming antigenic - renal tubular cells are susceptible to this effect as they are exposed to high Pb concentrations during clearance. Blood Pb levels reflect soft tissue Pb in a steady state i.e. is an indicator of recent exposure. Most of the Pb body burden is in bone (with chronic Pb exposure). The lead that accumulates in the bone ultimately provides a source for remobilisation and continued toxicity after exposure ceases. The total bodily content of lead is called the body burden; in a steady state, about 90 percent of the body burden is bound to bone. Lead in blood has an estimated half-life of 35 days, in soft tissue 40 days and in bone 20 to 30 years. Inorganic lead does not undergo any metabolic transformation or digestion in the intestines or detoxification in the liver. Lead is excreted quite slowly from the body (with the biological half-life estimated at 10 years).
Clinical manifestations of occupational exposure	Symptoms may include arthralgias, headache, weakness, depression, loss of libido, impotence and vague gastrointestinal difficulties. Exposures to Pb cause the following late effects: • Encephalopathy • Peripheral neuropathy • Neurological and neurobehaviqoural effects • Renal effects • Hypertension • Reduced fertility • Anaemia • Gout • EPA classified lead as a probable human carcinogen (group B2). • Tetra

Occupational exposures (MSDS)	IDLH STEL OEL	100mg Pb/m^3 0.15mg Pb/m^3 CARC

OCCUPATIONAL EXPOSURE		
Biological Monitoring	**Sample type:** 1. Tetra alkyl lead: Urine Pb 2. Inorganic lead: Blood Pb	**Sampling time:** 1. DS (During Shift) – the 1st sample to be taken within 6 months of commencement of employment) 2. DS (During Shift) – the 1st sample to be taken within 14 days of commencement of employment

Reference ranges: Lead Regulations 2002 OHS Act 1993

1. Tetra alkyl lead: Urine Lead

Men:	
Urinary lead (ug/L)	Maximum Intervals between tests
<120.0	6 weeks
120-149.0	1 week
>150.0	Removal from workplace
Women not capable of procreation:	
Urinary lead (ug/L)	Maximum Intervals between tests
<120	6 weeks
120 – 149.0	1 week
>150.0	Removal from workplace

	Women capable of procreation:	
	Urinary lead (ug/L)	Maximum Intervals between tests
	<65	3 monthly
	>75	Removal from workplace
	<65	Reinstatement in workplace

2. Inorganic lead: Blood Lead

Men:	
Blood Lead (ug/100 ml)	Maximum Intervals between tests
<20	12 months
20-39	6 months
40-59	3 months
60 - 70	According to the discretion of the Occupational Health Practitioner
>60	Remove from workplace
<50	Reinstatement in workplace
Women not capable of procreation:	
Blood Lead (ug/100 ml)	Maximum Intervals between tests
<20	12 months
20 – 39	6 months
40 – 59	3 months
60 - 70	According to the discretion of the Occupational Health Practitioner
65 and over	Remove from workplace
<55	Reinstatement in workplace
Women capable of procreation:	
Blood Lead (ug/100 ml)	Maximum Intervals between tests
40 and less	3 months
>40	Removal from workplace
<30	Reinstatement in workplace

Biological Effect Monitoring pathology based	Blood	Serum urea and creatinine, eGFR, Zinc protoporphyrin or free erythrocyte protoporphyrin level, haemoglobin, haematocrit and peripheral smear
	Urine	Microscopy

MANGANESE

Chemical Formula	**Mn**	
CAS Number	**7439-96-5**	
Occupational uses	Used in the following: • Alloys with steel (main use), aluminium and copper • Anticorrosive in most steel alloys • Dry cell batteries • Glass manufacturing and cleaning agent for glassware • Binding and colouring agent in red bricks and pottery • Manufacture of fireworks and matches • Fertilisers and fungicides • Additive for gasoline	
Toxicokinetics and toxicodynamics	**Route of entry**: **Inhalation and ingestion** Absorption depends on particle size and solubility with a significant amount cleared by mucociliary action and swallowed. However, • Smaller particles deposit in the lower respiratory tract and absorb into the blood and lymph nodes • Nano- and larger sized particles are trapped in the nasal mucosa which may be absorbed into the brain via the olfactory and trigeminal nerves and accumulating in the globus pallidus. It is proposed to be mediated by divalent metal transporter proteins and transferrin receptors. • Soluble forms such as manganese chloride are more readily absorbed than less soluble oxides. Absorption in the GIT is via the divalent metal transporters and is influenced by iron, calcium, phosphorus, fibre, and phytates. Mn is eliminated mainly in the faeces with small amounts eliminated in the urine (Mn in urine do not correlate with exposure or their adverse effects). Mn is bound to erythrocytes, transferrin if in the trivalent state, and alpha-microglobulin if in the divalent state. Elimination of absorbed Mn from blood is rapid. It concentrates in the liver, and kidneys with minor amounts transported to the brain and bone. The neurotoxicity is associated with Mn-induced oxidative stress, and disruption of neurotransmitter synthesis and metabolism of the GABA and glutamate systems.	
Clinical manifestations of occupational exposure	Inhalation of Mn oxide fumes may cause flu-like symptoms and, during severe exposure to fumes or dust of various manganese salts, a severe chemical pneumonia (Mn pneumonia) may occur. The primary target organ of Mn toxicity is the central nervous system, particularly the extra- pyramidal system and manifests with chronic manganism. Exposure to heavy concentrations of dust or fumes for as little as three months may produce the condition, but usually cases develop after 1-3 years of exposure. The symptoms may simulate progressive bulbar paralysis, post encephalitic Parkinsonism and multiple sclerosis. Male reproductive effects such as decreased libido, impotence and decreased fertility may occur. Acute intoxication by ingestion rarely occurs and is caused by accidental or voluntary ingestion of a manganese salt which causes massive burns in the digestive tract, oedema of the upper respiratory tract and circulatory collapse.	
Occupational exposures(MSDS)	IDLH STEL OEL 8hr TWA	500mg Mn/m^3 0.2 mg/m^3
OCCUPATIONAL EXPOSURE		
Biological Monitoring	**Sample:** 1. Manganese in urine 2. Manganese in blood	**Sampling time:** Not specific (NS)
	1. Manganese urine	***BEI (Biological Exposure Index):** Unknown *Normal values: <4.5 ug/g creatinine

	2. Manganese in blood	Urine Mn results do not correlate with signs or symptoms of Mn toxicity, and are only indicative of exposure. * **BEI (Biological Exposure index)**: Not established * Biological Tolerance value (BAT, Germany 2007) : 20 ug/l
Biological Effect Monitoring pathology based	Blood	Full blood count & diff, urea, creatinine and eGFR electrolytes, iron profile, ALT, AST and Gamma GT
	Urine	proteinuria

MERCURY (INORGANIC)

Chemical Formula	**Hg**
CAS Number	**7439-97-6**
Occupational uses	Used in: • Mining • Smelting • Refining, batteries, gold ore extraction • Chlor alkali industries • Laboratories, dental amalgams, as part of instrumentation (pressure, mechanisms, vacuum pumps, etc.)
Toxicokinetics and toxicodynamics	**Route of entry: Inhalation skin and ingestion** Elemental Hg is non-toxic and is present in the air as vapours. The inorganic salts are present as aerosols. Once ionised to Hg^{2+} it becomes toxic. Further bioconversion to alkyl Hg (methylHg) yields a highly toxic form of Hg. Hg toxicity occurs in the following ways: • Reacts with sulfhydryl groups of proteins (albumin, glutathione, cysteine, and metallothionein) causing a conformational change in tertiary structure that results in loss of biological activity. For example the kidney is a target organ for toxicity since inorganic Hg is taken up and accumulates in the kidney. The resulting toxic effects lead to renal damage and increased elimination of enzymes and proteins in the urine (detected through measurement of urinary excretion of low and high molecular weight proteins and renal tubular enzymes (N-acetyl-β-D- glucosaminidase (NAG), β-galactosidase). • With tertiary structural change, some proteins become immunogenic, causing proliferation of beta-lymphocytes that produce immunoglobulins. These bind to new antigens such as collagen. • Alkyl Hg is highly lipophilic and is selective for lipid-rich tissue such as the neurons and myelin. When absorbed, some elemental Hg is oxidised to its divalent form and is mainly distributed to the kidney and brain. Hg salts are accumulated in the kidney. Elimination of Hg is in faeces and urine where the half-life is 40 days. This therefore does not reflect new or recent exposures. Hg in urine reflects long-term exposure to elemental Hg and its inorganic salts. Urinary Hg is preferred for biological monitoring, as exposures remain fairly constant over sufficiently long period of time and sampling is standardised. With urine Hg where exposures remain fairly constant over a sufficiently long period of time and sampling is standardised. There is a latent period before steady state is reached and urine Hg levels reflect exposure. Workers would, therefore, need to be occupationally exposed to Hg for a minimum of 6 months. Air borne exposure levels can be correlated. Blood levels of Hg are affected by the presence of organic Hg from dietary fish intake.
Clinical manifestations of occupational exposure	The neurological system and kidneys are the main target organs. Acute intense exposure to elemental Hg vapour results in bronchial irritations, erosive bronchitis and diffuse interstitial pneumonitis. Gastrointestinal and renal tubular necrosis occur after ingestion of mercuric mercury. Renal effects of long-term chronic exposure include renal tubular damage and immunological-based glomerulonephritis. The neurological effects manifest in

	a characteristic tremor. Other neuro- psychomotor effects include cognitive effects, impaired motor coordination, delays in reaction time, deficits in memory and attention and tremors (may begin in the fingers, eyelids, lips or tongue. This is considered as an early sign of metallic Hg vapour exposure and can be associated with severe behavioural and personality changes, memory loss, increased excitability, and in severe cases, delirium and hallucinations. This constellation of symptoms is called mercurial erethism.	
Occupational exposures (MSDS)	IDLH STEL OEL 8hr TWA	10mg Hg/m^3 0.05mg/m^3 SKIN
OCCUPATIONAL EXPOSURE		
Biological Monitoring	**Sample:** 1. Total inorganic Hg in urine 2. Hg in blood (Methyl Hg)	**Sampling time:** 1. Prior to shift (PS) 2. ES (End of shift) EW(end of work week)
	1. Hg in urine: 2. Hg in blood:	***BEI (Biological Exposure Index):** 20.0 ug/g creatinine PS ***Tentative maximum permissible concentration:** 20 ug/l
Biological Effect Monitoring pathology based	Blood	Urea, creatinine and eGFR
	Urine	Microscopy, proteinuria i.e. low and high molecular weight proteins, and renal tubular enzymes.

METHANOL

Chemical Formula	**CH_3OH**
CAS Number	**67-56-1**
Occupational uses	Used as: ● Starting material in the manufacture of many chemical products approximately 40 % in wood for the production of formaldehyde, synthesis of methacrylates, methylamines, methyl halides, ● Solvent for inks, dyes, resins and adhesives, manufacture of photographic film, plastics, textile soaps, wood stains, shatterproof glass and waterproofing formulations ● Ingredient of paint and varnish removers, embalming fluids and antifreeze mixtures, extractant in a number of processes ● Anti detonant fuel-injection fluid for aircraft. A major use is in the production of methyl tertiary butyl ether (MTBE).
Toxicokinetics and toxicodynamics	**Routes of entry: Inhalation, ingestion and dermal absorption** In the body products are formed by the oxidation of methanol. These products are formaldehyde and formic acid, both of which are toxic. Methanol is absorbed via the respiratory, dermal or gastrointestinal routes, respiratory being the major route of absorption in the workplace through exposure to vapours. Occupational intoxication has occurred as a result of extensive dermal exposure to liquid methanol. Methanol is eliminated unchanged in urine (<10%) and exhaled air. It is also excreted as metabolites. The elimination is fast and complete. The major elimination pathway is metabolism. The total amount excreted this way accounts for about 70% to 80% of the absorbed amount. The elimination is a saturable process with elimination half-lives of about 1.5- 2.0 hours). The major metabolite in humans is formic acid, which is responsible for the unique manifestations of methanol poisoning, metabolic acidosis and optic neuropathy. **Normal:** <0.05 mmol/l **Toxic:** >6.24 mmol/l

	Indication for haemodialysis: >14 mmol/l	
Clinical manifestations of occupational exposure	Mildly toxic by inhalation. Systemic effects by ingestion and inhalation including optic neuropathy, headache, cough, nausea and vomiting. It is also an eye and skin irritant. Methanol should be regarded as a cumulative poison. The main toxic effect of methanol is exerted upon the nervous system, particularly the optic nerves and possibly the retina which can progress to permanent blindness. Coma resulting from massive exposures may last as long as 2-4 days. Methanol is a narcotic. Although single exposures to fumes may cause no harmful effect, daily exposure may result in the accumulation of sufficient methanol in the body to cause illness.	
Occupational exposures	IDLH STEL OEL	6000 ppm 500 ppm 400 ppm SKIN
OCCUPATIONAL EXPOSURE		
Biological Monitoring	**Sample:** 1. Methanol in urine 2. Formic acid in urine 3. Methanol in blood	**Sampling Time:** 1. ES (End of Shift) 2. BS (Before Shift) and EWW 3. EW (End of Work)
	1. Methanol Notation 2. Formic acid : creatinine 3. Methanol in blood	**BEI (Biological Exposure Index):** 15 mg/l (ES) B, Ns 80 mg/g creatinine (BS, EWW) Normal: <0.05 mmol/l Toxic: >6.24 mmol/l Indication for haemodialysis: >14 mmol/l
Biological Effect Monitoring pathology based	Urine	Dipstick (pH)
	Blood	Urea, creatinine, eGFR, ALT, AST GGT and ALP

Methyl n-butyl ketone (2.5 Hexanedione)

Chemical Formula	**$CH_3CO(CH_2)_3CH_3$**	
CAS Number	**123-86-4**	
Occupational uses	Used as a volatile organic compound/solvent. It is formed as a waste product resulting from industrial activities such as making wood pulp and producing gas from coal, and in oil shale operations. In the past, 2-hexanone was used in paint and paint thinners, to make other chemical substances and to dissolve oils and waxes.	
Toxicokinetics and toxicodynamics	**Routes of entry: Inhalation, ingestion and dermal absorption** Absorption of Respiratory tract: 2-Hexanone is well absorbed from the respiratory tract. A small study in humans estimated that approximately 75–92% of the inhaled dose was absorbed. Gastrointestinal tract: 2-Hexanone is well absorbed from the gastrointestinal tract. A small study in humans estimated that approximately 66% of the oral dose was absorbed. 2-Hexanone is absorbed following dermal exposure; however, quantitative estimates of the absorption fraction are not available. In humans, 2-hexanone was detected in serum. 2-Hexanone undergoes metabolism through reduction and oxidation reactions. The metabolite, 2,5-hexanedione, is toxicologically active. Expired breath and urine appear to be the main routes of excretion for 2-hexanone and its metabolites. Analysis of serum showed that 2-hexanone was present in serum in subjects exposed to 100 ppm, but not to 10 or 50 ppm.	
Clinical manifestations of occupational exposure	Breathing or swallowing a high dose of 2-hexanone may harm your nervous system. Workers exposed to 2-hexanone in the air for almost a year felt weakness, numbness, and tingling of the hands and feet; both neuropathy and muscular atrophy are significant effects.	
Occupational exposure	IDLH STEL OEL 8-hour TLV	Not available 20 ppm 10 ppm SKIN
OCCUPATIONAL EXPOSURE		
Biological Monitoring	**Sample:** 1.2.5 Hexanedione in urine	**Sampling time:** ES (End of Shift), EWW (End of Work week)
	2,5-Hexanedione urine	**BEI (Biological exposure index):** 0,4 mg/L ES EWW
Biological Effect Monitoring pathology based	None	

METHYL CHLOROFORM (1,1,1 Trichloroethane)

Chemical Formula	**CH_3CCl_3 or $C_2H_3Cl_3$**	
CAS Number	**71-55-6**	
Occupational uses	It is a synthetic chemical used: • As a solvent for metal degreasing, dry cleaning, natural and synthetic resins, oils, waxes, tar and alkaloids • In textile processing and in various formulations including adhesives aerosols, coatings, printing inks, typewriter correction fluid, drain cleaners, shoe polish and as a carrier of aerosols • For cleaning, degreasing and as an extraction solvent.	
Toxicokinetics and toxicodynamics	**Routes of entry: Inhalation** is a major route of absorption. Methyl chloroform is excreted almost entirely unchanged through the lungs. A small fraction of methyl chloroform is oxidised to trichloroethanol (TCOH) and then to trichloroacetic acid (TCAA).	
Clinical manifestations of occupational exposure	If you breathe air containing high levels of 1,1,1-trichloroethane for a short time, you may become dizzy and lightheaded and possibly lose your coordination. These effects rapidly disappear after you stop breathing contaminated air. If you breathe in much higher levels, you may become unconscious, your blood pressure may decrease, and your heart may stop beating.	
Occupational exposures	IDLH RHCA-STEL RHCA-OEL	700 ppm 900 ppm 700 ppm
OCCUPATIONAL EXPOSURE		
Biological Monitoring	**Sample:** 1. Trichloroacetic Acid (TCAA) in urine 2. Total trichloroethanol in urine 3. Trichloroethanol in blood 4. Trichloroethane in blood	**Sampling Time:** 1. EWW (End of Work week) 2. ES, EWW (End of Shift, End of Work week) 3. ES, EWW (End of Shift, End of Work Week) 4. ES, EWW (End of Shift, End of Work Week) or PS, EWW (Pre Shift, End of Work Week)
	1. Trichloroacetic acid Notation 2. Total Trichloroethanol urine Notation 3. Trichloroethanol blood Notation 4. Trichlorethane blood	**BEI (Biological exposure index):** 10 mg/l EWW Ns, Sq 30 mg/l ES, EWW Ns, Sq 1 mg/l ES, EWW Ns 4.0 mg/l at end of shift ES, EWW 0.7 mg/l PS, EWW
Biological Effect Monitoring pathology based	Blood	Urea, creatinine, eGFR, ALT, ASTL ALP and GGT
	Urine	Dipstick

METHYL ETHYL KETONE (MEK)

Chemical Formula	**$CH_3\ COCH_2\ CH_3$**	
CAS Number	**78-93-3**	
Occupational uses	MEK is one of the most widely used solvents in lacquers, paints, adhesives and coatings containing synthetic resins, plastics or rubber. MEK is also a solvent used in many different industrial and artisan types of work. It is one of the main solvents in the mixture used in leather glues. Because of this, MEK is (together with n-hexane and its isomers) an environmental pollutant in shoe factories.	
Toxicokinetics and toxicodynamics	**Routes of entry: Inhalation, ingestion and dermal absorption** The main part of inhaled MEK is supposedly metabolised in the intermediary metabolism. Pulmonary retention accounts for 53% of the inhaled amount. 3% of total uptake is excreted unchanged in expired air. Dermal absorption occurs rapidly. Elimination of MEK in blood appears to exhibit two phases: the initial alpha-phase (half-life = 30 min) over the first post-exposure hour, followed by the terminal beta-phase (half-life + 81 min). In man, the urinary excretion of MEK and 3 hydroxy-2 butanone together accounts for not more than 0,1% of the absorbed dose. Excretion over 24 hours is little more than 2% of total MEK absorbed. Inhalation is the primary route of absorption in human industrial exposure to MEK because of the chemical's high volatility at room temperature, but skin absorption and ingestion are also possible routes.	
Clinical manifestations of occupational exposure	It causes irritation of the nose and throat, and dermatitis of the face. MEK used in solvent mixtures can result in a decrease in nerve conduction, memory and motor alterations and vomiting. Effects reported in humans due to acute inhalation exposure to methyl chloroform include hypotension, mild hepatic effects and CNS depression. Mild motor impairment (e.g. increased reaction time), lightheadedness, impaired balance, and ataxia have been reported in acutely exposed humans. Cardiac arrhythmia and respiratory arrest may result from the depression of the CNS. Symptoms of acute inhalation exposure include dizziness, nausea, vomiting, diarrhoea, loss of consciousness and decreased blood pressure. Methyl chloroform is mildly irritating when applied to the skin of humans.	
Occupational exposures	IDLH STEL OEL	Not available 600 ppm 400 ppm SKIN
OCCUPATIONAL EXPOSURE		
Biological Monitoring	**Sample:** MEK in urine	**Sample Time:** ES (End of Shift)
	Methyl ethyl ketone	**BEI (Biological Exposure Index):** 2 mg/l ES
Biological Effect Monitoring pathology based	Blood	Urea, creatinine, eGFR, ALT, AST, ALP and GGT
	Urine	Dipstick

METHYL ISOBUTYL KETONE (MIBK)

Chemical Formula	**$CH_2COCH_2CH(CH_3)_2$**	
CAS Number	**108-10-1**	
Occupational uses	Used as: • A solvent for protective coatings, lacquers and varnishes. • A raw material in the production of antioxidants • An extraction solvent for metals and pharmaceuticals and in the production of paints and pesticide formulations • As a solvent for adhesives • A denaturant in cosmetic products. MIBK is a synthetic flavouring adjuvant. MIBK occurs naturally in plant and animal oils.	
Toxicokinetics and toxicodynamics	**Route of entry: Inhalation, ingestion and dermal absorption** After inhalation (major route of exposure), elimination of MIBK from blood is biphasic (elimination half-times of 12 minutes and 70 minutes respectively). Only about 0.04% is eliminated unchanged through the kidneys, within 3 hours. Absorbed MIBK is essentially completely cleared out of the system within 90 minutes of exposure. MIBK is metabolised in the liver to water-soluble excretory products. Therefore, urine is the major excretory route for MIBK excretion.	
Clinical manifestations of occupational exposure	Inhalation causes irritation of the eyes and nose. Also results in weakness, headache, nausea and vomiting, dizziness, and in coordination. High concentrations bring about anaesthesia and CNS depression. Chronic exposure: Skin contact dries out skin and may cause dermatitis. Causes: burning eyes, nausea, headache, weakness, insomnia, gastrointestinal pain, enlargement of the liver. May also cause renal effects.	
Occupational exposures	IDLH STEL OEL	Not available 150 ppm 40 ppm CARC, SKIN
OCCUPATIONAL EXPOSURE		
Biological Monitoring	**Sample:** MIBK in urine	**Sampling Time:** ES (End of Shift)
	Methyl isobutyl ketone	**BEI (Biological Exposure Index):** 1 mg/l
Biological Effect Monitoring pathology based	Blood	Urea, creatinine, eGFR, ALT, AST, GGT and ALP
	Urine	Dipstick

NICKEL

Chemical Formula	**Ni**	
CAS Number	**7440-02-0**	
Occupational uses	Sparingly soluble Ni compounds (sulfides, oxides, carbonates, and sulfidic ores): • mining of Ni ores • Smelting and refining processes • Grinding and welding of Ni-containing alloys Soluble Ni acetate, chloride, hydroxide, and sulfate: • Electroplating industry • Ni carbonyl/tetracarbonyl is a highly toxic form of Ni: • Ni coatings • Glass plating • Catalyst in chemical reactions Other industries uses: • Manufacture of products containing Ni (NiCad) batteries, coins, wires, electronics, computer equipment, watches, eyeglass frames, cooking utensils, dental braces, orthopaedic implants and circulatory stents and pigments for paints or ceramics. • Manufacture of products from stainless steel • Recycling, handling or using the abovementioned products	
Toxicokinetics and toxicodynamics	**Routes of Entry: Inhalation, skin, ingestion** Inhalation is the main route of occupational exposure and occurs by inhalation of: • Dust (relatively insoluble nickel compounds) • Aerosols derived from solutions (soluble nickel) • Gaseous Ni (usually nickel carbonyl) The solubility of Ni compounds affects the toxicokinetics of Ni in the body, i.e. the more soluble are more rapidly absorbed and eliminated mainly via urine. Inhaled less soluble particles of Ni compounds are retained and accumulate in the lung and regional lymph nodes. There is gradual release over time. Ni oxides and sulfides and aqueous solutions of Ni in the oxidation state i.e. 1^+ 2^+ 3^+ are considered group 1 carcinogens. Tissue inflammation results from the excretion of Ni-protein complexes. Inhaled Ni carbonyl is absorbed and crosses all biological membranes. In blood, Ni binds to proteins such as albumin, L-histidine, and alpha-2-macroglobulin. Ni binds to DNA and can lead to gene silencing and inhibition of DNA repair by various mechanisms.	
Clinical manifestations of occupational exposure	The carcinogenic and allergenic effects of Ni are most significant. These result in skin (contact dermatitis/ "nickel itch") and respiratory sensitisation. Inhalation of soluble Ni can cause irritation of the nose and sinuses and could also lead to loss of the sense of smell or perforation of the nasal septum. This mainly occurs in electroplating. Long-term exposure may lead to asthma, bronchitis or other respiratory diseases. Inhalation of Ni can cause cancer of the lungs, nose and sinuses. Cancers of the larynx (throat) and stomach have also been attributed to inhalation of Ni. Ni carbonyl and insoluble Ni compounds are the forms of Ni responsible for cancer. IARC classifies Ni compounds as group 1 human carcinogens.	
Occupational exposures (MSDS)	IDLH RHCA STEL as soluble /insoluble inorganic compound (NOS) OEL	10mg Ni/m^3 0.1 mg/m^3 (Inhalable fraction) CARC 0.02 mg/m^3 (Respirable fraction) CARC None
OCCUPATIONAL EXPOSURE		

<table>
<tr><td>Biological Monitoring</td><td>Sample:
1. Nickel in urine
2. Nickel in serum</td><td>Sampling time:
End of Shift (ES), End of Work week(EW)
Not critical (NC)</td></tr>
<tr><td></td><td>1. Nickel in urine
2. Nickel in serum</td><td>*BEI(Biological Exposure Index):
30 ug/l ES, EW
Tentative Scheme for Administrative Action Levels based on Atmospheric and Biological Monitoring of Nickel Refinery Workers:

<table>
<tr><th>Category</th><th>Air Ni (mg/m^3)</th><th>Serum Ni (ug/l)</th><th>Frequency</th><th>Action</th></tr>
<tr><td>I</td><td><0.1</td><td><4.0</td><td>2years</td><td>None</td></tr>
<tr><td>II</td><td>0.1-0.49</td><td>4.0-7.9</td><td>1year</td><td>None</td></tr>
<tr><td>III</td><td>0.5-0.99</td><td>8.0-9.9</td><td>6months</td><td>Review of work processes and protection</td></tr>
<tr><td>IV</td><td>>/= 1.0</td><td>>/= 10</td><td>3months</td><td>Same as III plus mandatory respiratory protection</td></tr>
</table>
</td></tr>
<tr><td>Biological Effect Monitoring pathology based</td><td>Blood</td><td>Creatinine, Urea and eGFR</td></tr>
</table>

NITROBENZENE

Chemical Formula	**$C_6H_5NO_2$**	
CAS Number	**98-95-3**	
Occupational uses	Nitrobenzene is mainly used in the production of Aniline. It is also used to manufacture dyes, oils, drugs, pesticides and synthetic rubber.	
Toxicokinetics and toxicodynamics	**Route of entry: Inhalation and dermal absorption** It is oxidised to p-aminophenol, which is excreted by the kidney. Exposure causes the formation of methaemoglobin resulting in functional anaemia. As it is heavier than air it may cause asphyxiation in poorly ventilated areas.	
Clinical manifestations of occupational exposure	Short term or acute exposure from inhalation can cause methaemoglobinaemia. Initial symptoms of cyanosis (15% methaemoglobin) and headache are followed by shortness of breath, nausea and vomiting, weakness, dizziness (40% methaemoglobin), tachycardia, arrhythmia and coma (75% methaemoglobin). Symptoms may occur 2-4 hours post exposure depending on the exposure level. No evidence available for carcinogenicity or reproductive and developmental effect. There are limited studies suggesting liver and CNS effects.	
Occupational exposures	IDLH STEL OEL	200 ppm Not available 2 ppm CARC, SKIN
OCCUPATIONAL EXPOSURE		
Biological Monitoring	**Sample:** Methaemoglobin in blood	**Sampling time:** DS/ES (During or End of Shift)

	Methaemoglobin in blood	**BEI (Biological exposure index):** 1.5 % of haemoglobin DS/ES
Biological Effect Monitoring pathology based	Blood	Full blood count, ALT, ALP, AST, GGT, urea, creatinine and eGFR
	Urine	Dipstick

PARATHION

Chemical Formula	**$PSOC_6H_4NO_2$ $(C_2H_5O)_2$**	
CAS Number	**56-38-2**	
Occupational uses	Primarily used as an insecticide on fruit, cotton, wheat, vegetables and nut crops.	
Toxicokinetics and toxicodynamics	**Route of entry**: **Inhalation, ingestion, dermal and eye absorption** Exposure is generally during manufacture, formulation, and in field application. The hepatic mixed function oxidases metabolise Parathion to Paraoxon. The latter is an active metabolite which inhibits cholinesterase. The esterases found in plasma and tissue hydrolyse Parathion and paraoxon to alkyl phosphates (diethyl thiophosphoric acid or dithiophosphate (DETP) and diethyl phosphoric acid or diethyl phosphate (DEP) and the main excretory metabolite of Parathion, p-nitrophenol. Elimination is via urine, with 80-90% of absorbed dose eliminated within 48 hours.	
Clinical manifestations of occupational exposure	The onset and severity of symptoms depend on the chemical structure of the compound being used, the amount of toxin to which an individual is exposed, route of exposure, rate of metabolic degradation, respiratory rate, ambient temperature, humidity and the use of personal protective equipment. **Short term:** Acute exposure to Parathion may cause tightness of chest, wheezing, watering of mouth, nausea, blurring of vision and twitching of skin in the area of contact. Severe intoxication may lead to convulsions and coma. **Long term:** Prolonged or repeated exposure to small amounts of Parathion makes the individual susceptible to systemic intoxication.	
Occupational exposures	IDLH STEL OEL	10 mg/m^3 Not available 0.1 mg/m^3 (Inhalable fraction and vapour) CARC,SKIN
OCCUPATIONAL EXPOSURE		
Biological Monitoring	**Sample:** 1. p-Aminophenol in urine 2. Whole blood cholinesterase activity 3. Pseudocholinesterase –serum (CHS)	**Sampling Time:** 1. ES (End of Shift) 2. Discretionary Pre-shift/Post exposure [generally baseline taken during season or peak application period] True Baseline Level = taken 4 weeks after non-exposure; ideally 2 baseline measurements are to be done 3-14 days apart; should agree to 15-20%. 3.After acute exposure

	1. p-Aminophenol:creatinine Notation 2.Whole blood cholinesterase 3.CHS	**BEI (Biological exposure index):** 0.5 mg/g creatinine ES Ns A reduction of 30% or more from a basal (pre-exposure) level may indicate organophosphate toxicity/exposure. **Reference limits:** M&F: 3167-6333 U/L
Biological Effect Monitoring Pathology based	Blood	Full blood count, urea, creatinine, eGFR, electrolytes, ALT, AST ALP and GGT
	Urine	Dipstick

PARAQUAT

Chemical Formula	**$C_{12}H_{14}Cl_2N_2$**	
CAS Number	**1910-42-5**	
Occupational uses	Herbicide-inhibits photosynthesis	
Toxicokinetics and toxicodynamics	**Route of entry**: **Inhalation, ingestion, skin and eye contact** The most likely route of exposure that would lead to poisoning is ingestion. Exposure is generally during manufacture, formulation, and in field application. Paraquat is largely non metabolised and excreted unchanged in urine.	
Clinical manifestations of occupational exposure	Paraquat is toxic; it causes direct damage when it comes in contact with the lining of the mouth, stomach and intestines when ingested. Death is likely if swallowed. Inhalation, skin and eye contact's effect depends on the severity of the exposure. It may cause heart-, kidney-, liver-, lung and oesophagus damage. Chronic exposure may cause pulmonary fibrosis - so called Paraquat lung.	
Occupational exposures	IDLH STEL OEL	1 mg/m^3 Not available Not available
OCCUPATIONAL EXPOSURE		
Biological Monitoring	**Sample:** 1. Urine	**Sampling Time:** 1. After acute exposure
	Reference limits: 1. Qualitative result should be negative to exclude Paraquat exposure	
Biological Effect Monitoring-pathology based	Blood	Arterial blood gas, kidney, liver, function
	Urine	Urinalysis

PENTACHLOROPHENOL

Chemical Formula	**C_6 Cl_5 OH**	
CAS Number	**87-86-5**	
Occupational uses	PCP is used as a wood, leather and paper preservative, a pesticide, a disinfectant, a mildew retardant, a fungicide and a contact herbicide [chlorinated hydrocarbon]. Persons may come into contact with it during manufacturing processes and application procedures.	
Toxicokinetics and toxicodynamics	**Routes of entry: Inhalation, ingestion and dermal absorption** It is oxidised to tetrachloro-hydroquinone and conjugated with glucuronic acid in the liver. PCP is primarily excreted in the urine in the free and (mostly) conjugated forms. Blood and urine elimination half-lives can range from 16 to 20 days. The slow elimination is due to high protein binding in the plasma (>96%) and tubular reabsorption.	
Clinical manifestations of occupational exposure	**Short term:** Acute exposure to PCP may cause irritation to the eyes, skin and respiratory tract. May also cause visual damage. **Long term exposure**: Prolonged or repeated exposure to PCP may cause systemic effects. The symptoms are weakness, loss of appetite, nausea, vomiting, shortness of breath, chest pain, excessive sweating, delirium, weakness, flushing, headache and dizziness. In severe cases, the body temperature is very high and death may occur within hours of the onset of symptoms. The risk of serious intoxication is greater in hot weather and in the presence of impaired liver and renal functions. Other effects include inflammation of the respiratory tract and bronchitis, aplastic anaemia, liver damage, renal damage, cardiovascular and central nervous system effects.	
Occupational exposures	IDLH STEL OEL	2.5 mg/m^3 2 mg/m^3 1 mg/m^3 (Inhalable fraction and vapour) CARC, SKIN
OCCUPATIONAL EXPOSURE		
Biological Monitoring	**Sample:** Total PCP in urine	**Sampling time:** 1. PS, EWW (prior to the last shift, End of Work week)
	PCP: creatinine	**Tentative maximum permissible concentration:** 1 mg/g creatinine PS, EWW
Biological Effect Monitoring pathology based	Blood	Full blood count and diff, urea, creatinine, eGFR, ALT, AST, GGT, ALP and Bilirubin
	Urine	Dipstick

PERCHLOROETHYLENE/Tetrachlorethylene

Chemical Formula	**$CCl_2 = CCl_2$**	
CAS Number	**127-18-4**	
Occupational uses	Cold cleaning and degreasing of metals, as a solvent for dry cleaning and for textile finishing and dyeing. Transformer insulating fluid for chemical muskant formulations. Process solvent for desulphurising coal.	
Toxicokinetics and toxicodynamics	**Route of entry: Inhalation, dermal and eye contact** After absorption, a fraction of perchlorethylene is oxidised to trichloroacetic acid (TCAA). Human ability to metabolise perchlorethylene is limited and the compound is mainly excreted unchanged in exhaled air. At rest, alveolar retention of perchlorethylene decreases from about 90% at the onset of exposure to 47% after 8 hours of exposure. Tests indicate that the alveolar retention drops to about 4%, 16 hours after a single 8-hour exposure period has ended. Between 1 and 3% is excreted in urine as Trichloroacetic acid (TCAA). The small value of this fraction, coupled with its variability, means that TCAA levels should only be used as a screening test. Elimination from the body is slow due to its progressive release from adipose tissue. The concentration of TCAA in blood increases up to 20 hours after a single exposure, thereafter it decreases with a half-life of about 80 hours.	
Clinical manifestations of occupational exposure	**Short term:** May cause headache, nausea, dizziness and coma. It may also cause irritation of the eyes, nose and throat. Liver damage may result after several weeks of exposure. **Long term:** Prolonged or repeated exposure to liquid perchlorethylene may lead to skin irritation or liver damage. May cause neuropathies.	
Occupational exposures	IDLH STEL OEL	150 ppm 200 ppm 50 ppm
OCCUPATIONAL EXPOSURE		
Biological Monitoring	**Sample:** 1. Trichloracetic acid in urine 2. Tetrachloroethylene in blood	**Sampling Time:** End of Shift (ES), Prior to last Shift (PS)
	1. Trichloroacetic Acid: creatinine 2. Tetrachloroethylene in blood	**Tentative maximum permissible concentration:** 3 mg/g creatinine ES **BEI (Biological exposure index):** 0.5 mg/l PS
Biological Effect Monitoring pathology based	Blood	ALT, AST, ALP, GGT, urea, creatinine, eGFR and electrolytes
	Urine	Dipstick

PHENOL

Chemical Formula	**C_6H_5OH**	
CAS Number	**108-95-2**	
Occupational uses	Commercially used as a disinfectant and as an intermediate in chemical syntheses such as nylon and other man-made fibres. Phenol is also used in the manufacturing of pesticides. Other exposures to phenol may occur through the use of phenol-containing medicinal products (including mouthwashes, toothache drops, throat lozenges, analgesic rubs, and antiseptic lotions) or smoking tobacco.	
Toxicokinetics and toxicodynamics	**Route of entry: Inhalation and skin absorption** It is rapidly excreted in the urine within 24 hours of exposure, in the form of conjugates with glucuronide and sulphate. Excretion is monophasic with a half-life of 3,5 hours.	
Clinical manifestations of occupational exposure	Phenol is a corrosive and acutely toxic chemical. Death has resulted from phenol absorption through a skin area as small as 400 cm^2. Because of the analgesic properties of phenol, the sensation of pain may be diminished leading to less awareness of contact with the chemical, resulting in higher degrees of local damage. Prolonged exposure may result in dark skin pigmentation (ochronosis). Burns eyes and skin affects tissue. Absorption may produce cyanosis, shock, weakness, collapse, convulsions, liver and kidney damage (mainly), coma and death. Phenol exposure increases the risk of coronary artery disease.	
Occupational exposures	IDLH STEL OEL	250 ppm Not available 10 ppm SKIN
OCCUPATIONAL EXPOSURE		
Biological Monitoring	**Sample:** Total phenol in urine	**Sampling Time:** ES (End of Shift)
	Phenol: creatinine	**BEI (Biological exposure index):** 250 mg/g creatinine ES
Biological Effect Monitoring Pathology based	Blood	Urea, creatinine, eGFR, ALT, AST, ALP and GGT
	Urine	Dipstick and microscopy

POLYACROMATIC HYDROCARBONS

Chemical Formula	**Benzo-(a)-pyrene ($C_{20}H_{12}$) & Naphthalene ($C_{10}H_8$)**	
CAS Number	**192-97-2 & 91-20-3**	
Occupational uses	Polycyclic Aromatic Hydrocarbons (PAH) are formed in the incomplete combustion of organic materials. They exist almost always as mixtures of several different compounds, except single compounds such as naphthalene. Coal tar pitch volatiles (CTPVs) are composed of chemical vapours that become airborne during the heating of coal tar pitch. Synonyms for CTPVs vary depending upon the specific compound (e.g. pyrene, phenanthrene, acridine, chrysene, anthracene and benzo(a)pyrene). The National Institute for Occupational Safety and Health (NIOSH) considers coal tar, coal tar pitch, and creosote to be coal tar products. Natural sources of PAHs include forest and grass fires, oil seeps, volcanoes, chlorophyllous plants and fungi. High concentrations of PAHs are found in coke ovens, aluminium production, steel industry, asphalt industry, creosote impregnating plants, coal tar roofing, bitumen used for road paving, heating oils, diesel oils, gas and petroleum industries and smokehouses(for smoked meat and fish). Naphthalene is used for the production of moth balls and found in JP-8 jet fuel.	
Toxicokinetics and toxicodynamics	**Routes of entry: The major route of exposure is via the lungs, skin or eye contact** The most studied of the PAHs is benzo-[a]-pyrene. The PAH particles may dissolve, may be removed by bronchial-mucociliary action, or may remain in the lung for a long time. The major depots for PAHs are adipose tissue and mammary gland. PAH's metabolised by the CYP-450 enzyme complex in the liver resulting in hydroxylated metabolites. These reactive metabolites then react with DNA to form DNA adducts, which contributes to key gene mutations. PAHs are transformed initially to epoxides, which are converted to dihydrodiol derivatives and phenols. Glucuronide and sulphate conjugates of these metabolites are excreted in the bile and urine. Glutathione conjugates are further metabolised to mercapturic acids in the kidney and are excreted in the urine. The hydroxylated metabolites of the PAHs are excreted in human urine both as free hydroxylated metabolites and as hydroxylated metabolites conjugated to glucuronic acid and sulphate. 1-Hydroxypyrene is commonly used as a biological marker for exposure to PAH and CTPV compounds. 1-Naphthol is used as a marker for Naphthalene exposure.	
Clinical manifestations of occupational exposure	The acute toxicity from PAHs varies from moderate to low. Systemic toxicity in humans is only caused by Naphthalene but it is very rare. Acute exposure of humans to Naphthalene by inhalation, ingestion, and dermal contact is associated with hemolytic anaemia, damage to the liver, and neurological damage. Cataracts have also been reported in workers acutely exposed to Naphthalene by inhalation and ingestion. PAHs are photosensitizers i.e., an abnormally high reactivity in the skin and eyes to ultraviolet radiation or natural sunlight. The dermal toxic effects are enhanced by exposure to ultraviolet light. Progression to skin cancer may occur. Cough, chronic bronchitis, and haematuria are effects noted. There is sufficient information from experimental animals that PAHs are carcinogenic. These include lung [main site], kidney, bladder, gastrointestinal, and skin. The well-known carcinogenic PAHs include benzo-[a]-pyrene, benz-[a]-anthracene, and dibenz-[a,h]-anthracene. These are classified as Carcinogenicity Category 1B. In addition, benzo-[a]-pyrene is classified as a Germ Cell Mutagenicity category 1B (may cause genetic defects) and Reproductive Toxicity Category 1B (may damage fertility or unborn child).	
Occupational exposures	IDLH Naphthalene IDLH Benzo-(a)-pyrene STEL Naphthalene, Benzo-(a)-pyrene	250 ppm 80 mg/m^3 - 20 ppm CARC, SKIN

	OEL 8hr TWA Naphthalene OEL 8hr TWA Benzo-(a)-pyrene	0.2 mg/m3
OCCUPATIONAL EXPOSURE		
Biological Monitoring	**Sample:** Hydroxypyrene in urine 1-Naphthol in urine	**Sampling time:** ES (End of Work week) NS (Not specific)
	Hydroxypyrene: creatinine 1-Naphthol in urine	***BEI (Biological exposure index):** 2.7 ug/g creatinine ES ***Industrial exposed:** 106-4109 ug/g (Range, NB not BEI/ref range)
Biological Effect Monitoring pathology based	Blood	Serum U&E, full blood count, liver function urea, creatinine and eGFR
	Urine	Dipstick - proteinuria, haematuria

STYRENE

Chemical Formula	**$C_6H_5CH = CH_2$**	
CAS Number	**100-42-5**	
Occupational uses	Liberation during spray-up manufacture of glass fibre, reinforced styrene-polyester articles, during spray application of styrene polyester surface coatings, during hand lay-up of glass fibres, during moulding of articles or potting electrical components with polystyrene, during manufacture of tires and other rubber goods using styrene-butadiene elastomers (SBR), in manufacture of concretes, during bag lay-up manufacture of glass fibre, reinforced styrene-polyester articles, during use of surface coatings containing styrene-butadiene copolymer resins, liberation during die moulding of articles made from styrene polyester resins, during brush application of surface coatings, in process operations for production of polystyrene, acrylonitrile-butadiene styrene (ABS), styrene-acrylonitrile (SAN) and styrene-butadiene copolymers, in manufacture of surface coatings, use in miscellaneous processes as an elastomer, intermediate, or starting material, during manufacture of ion-exchange resins (styrene-divinylbenzene copolymer).	
Toxicokinetics and toxicodynamics	**Route of entry: Inhalation** (main absorption) and **dermal** (liquid/ vapour form) 1-2% of inhaled styrene is exhaled unchanged. Styrene is metabolised to mandelic acid (MA) and phenylglyoxylic acid (PhGA). These are excreted in the urine with a half-life of 5-10 hours. Elimination of styrene (less than 1% of the absorbed amount is eliminated as styrene) from the lungs is biphasic with half-lives of 13 to 52 minutes and 4 to 20 hours respectively. Elimination of MA (the major metabolite) from urine is biphasic with a half-life of 3 to 4 hours and 25 to 40 hours respectively. The biological half-life of PhGA in urine is greater than that of mandelic acid and is a function of the intensity of exposure.	
Clinical manifestations of occupational exposure	Inhalation causes irritation of the mucous membranes (@ 300 ppm) (eyes, nose and throat and can cause dizziness and loss of consciousness. Skin contact can burn the skin and eyes and cause dermatitis. Can also result in chest burning, wheezing, and dyspnoea. Heavy styrene exposure results in "styrene sickness" as manifested by muscle weakness, a feeling of drunkenness, etc. Possible reproductive hazard (spermatogenesis).	
Occupational exposures	IDLH	700 ppm

	STEL OEL	80 ppm 40 ppm CARC
OCCUPATIONAL EXPOSURE		
Biological Monitoring	**Sample:** 1. Mandelic acid + Phenylglyoxylic acid in urine 2. Styrene in urine 3. Styrene in blood	**Sampling Time:** 1. ES (End of Shift) 2. ES (End of Shift) 3. Not specified
	Reference Limits: 1. Mandelic acid + Phenylglyoxylic acid: creatinine Notation 3. Styrene in urine 4. Styrene blood	**BEI (Biological Exposure Index):** 400 mg/g creatinine ES Ns 40 ug/l **Tentative maximum permissible concentration:** 0.3 mg/l
Biological Effect Monitoring pathology based	Blood	Urea, creatinine, eGFR, ALT, AST, ALP and GGT
	Urine	Dipstick

TOLUENE

Chemical Formula	**$C_6H_5CH_3$**
CAS Number	**108-88-3**
Occupational uses	Major use of toluene is as a mixture added to gasoline to improve octane ratings, to produce benzene as a solvent in paints, chemicals, rubber, coatings, adhesives, inks and cleaning agents. Also found in glues and paint thinners. Occupations exposed to toluene include paint workers, dye makers, chemicals and petrochemical industries.
Toxicokinetics and toxicodynamics	**Routes of entry: Inhalation** (primary route), **ingestion and dermal contact** (liquid form) Pulmonary retention is about 50%. Toluene is eliminated unchanged in exhaled air. Pulmonary elimination accounts for 15-20% of the absorbed dose and is triphasic with half-lives of 1.5 minutes, 26 minutes and (tentatively) 3.7 hours. The remainder is excreted in the urine after being metabolised to hippuric acid (mainly) and o-cresol (less than 1% of the absorbed amount). Elimination of hippuric acid has a half-life of 1 to 2 hours according to American sources, but 7 to 8 hours according to WHO sources (Intake of alcohol speeds up the process of elimination of toluene from the blood, but inhibits the elimination of the metabolites (hippuric acid). Smoking also affects the metabolism of toluene (smokers have higher urinary excretion of o-cresol than non-smokers). The formation of hippuric acid and 0-cresol is decreased by co-exposure to benzene.
Clinical manifestations of occupational exposure	Inhalation can cause irritation to mucous membranes (eye, nose and throat) and can cause nausea, vomiting, headaches, dizziness and loss of consciousness. Skin contact can cause irritation of the skin and eyes and ingestion can bring about nausea, vomiting or loss of consciousness. Peculiar skin sensation may be produced such as "pins and needles" feeling of numbness. Very high concentrations may cause unconsciousness and death (IDHL is 500ppm [OSHA]). The liquid splashed in the eye may cause irritation and temporary damage. Inhalation may also cause difficulty in seeing in bright light. Skin contact

	will cause skin to crack and peel. Toluene has been implicated in the causation of cardiac arrhythmias, renal tubular damage, damage to the optic nerves and permanent neuropsychiatric effects. Chronic exposure results in bronchial asthma with an accelerated decrease in lung function (FEV1). In workers exposed to high levels of the mixture of organic solvents (much greater than the permissible levels), a linear dose-response relationship has been reported between the exposure level, risk of hearing loss and hearing threshold at high frequencies, especially 8000 Hz.	
Occupational exposures (MSDS)	IDLH STEL OEL	500 ppm Not available 40 ppm
OCCUPATIONAL EXPOSURE		
Biological Monitoring	**Sample:** 1. o-Cresol in urine 2. Toluene in venous blood 3. Toluene in urine 4. Hippuric acid in urine	**Sampling Time:** 1. ES (End of Shift) 2. PLSWW (Prior to Last Shift of Work week) 3. ES (End of Shift) 4. ES (End of Shift)
	1. o-Cresol:creatinine Notation 2. Toluene in venous blood 3. Toluene in urine 4. Hippuric acid:creatinine	**BEI (Biological exposure index):** 0.3 mg/g creatinine ES B 0.02 mg/l PLSWW 0.03 mg/l ES 1.5 g/g creatinine ES
Biological Effect Monitoring pathology based	Blood	Urea, creatinine ,eGFR, ALT, AST, GGT and ALP
	Urine	Dipstick

TRICHLOROETHYLENE

Chemical Formula	**CCl_2=CHCl**
CAS Number	**79-01-6**
Occupational uses	Solvent to remove grease from metal parts and extraction solvents for greases, oils, fats, waxes and tars. Can be found in some household products such as typewriter correction fluid, paint and spot removers and adhesives.
Toxicokinetics and toxicodynamics	**Routes of entry: Inhalation and dermal absorption** TCE enters the body mainly through inhalation, with an absorption rate of 60%. Extended skin contact may lead to significant dermal absorption. It is eliminated unchanged in exhaled air, and through the urine in the form of metabolites. It is metabolised by hepatic mixed function oxidases to chloral hydrate. The latter is rapidly oxidised to trichloroacetic acid (TCAA) or reduced to trichloroethanol (TCOH, sometimes also called TCE). Alcohol dehydrogenase catalyses the oxidation process. Individuals who are exposed to TCE may be intolerant to alcohol. Only small amounts are excreted in the form of metabolites in the urine, with half-lives ranging from 20 to 50 hours. Alcohol, caffeine and drug effects.
Clinical manifestations of occupational exposure	**Short term:** Inhalation of TCE can cause drowsiness, dizziness, headache, blurred vision, flushed skin, nausea, vomiting and cardiac arrhythmia. **Long term:** Prolonged or repeated exposure can cause headache, double vision, impaired co- ordination and senses of touch and smell, respiratory, liver and kidney function and intolerance to alcohol. The skin may be dry, have blisters or develop dermatitis. Flushing of skin also occurs and is referred to "degreaser's flush". TCE has been linked to mutagenic effects on humans. In workers exposed to high levels of the

	mixture of organic solvents (much greater than the permissible levels), a linear dose-response relationship has been reported between the exposure level, risk of hearing loss, and hearing threshold at high frequencies, especially 8000 Hz.	
Occupational exposures	IDLH STEL OEL	1000 ppm 50 ppm 20 ppm CARC, RSEN
OCCUPATIONAL EXPOSURE		
Biological Monitoring	**Sample:** 1. Trichloroacetic Acid (TCAA) 2. Trichloroethanol blood(TCE) 3. Total Trichloro Compounds (TTCC) 4. Trichloroethylene in blood	**Sampling Time:** 1. ES (End of Shift), EWW (End of Work week) 2. ES (End of Shift), EWW (End of Work week) 3. ES (End of Shift), EWW (End of Work week) 4. ES (End of Shift), EWW (End of Work week)
	1. Trichloroacetic Acid (TCAA) Notation 2. Trichloroethanol blood(TCE) Notation 3. Total Trichloro Compounds (TTCC) 4. Trichloroethylene in blood	**BEI (Biological exposure index):** 15 mg/l ES, EWW Ns 0.5 mg/l ES, EWW Ns Not established 1 mg/l ES.EWW
Biological Effect Monitoring pathology based	Blood	ALT, AST, GGT, ALP, urea, creatinine and eGFR
	Urine	Dipstick

VANADIUM

Chemical Formula	**V**
CAS Number	**1314-62-1**
Occupational uses	About 80% of the V now produced is used as ferrovanadium or as a steel additive.The following operations may involve VO and lead to worker exposures to the dust of this substance: • Use as a catalyst in the preparation of V alloys and compounds • Use as an oxidation catalyst in automobile catalytic converters and in organic synthesis • Use as a component of special ferrovanadium steels and in electric furnace steels, welding rods, and permanent magnet • Manufacture of pigments and glasses for ceramics production • Use as a catalyst in the textile industry to yield intensive black dyes and in the printing industry to make resinous black pigments from tar oils • Manufacture of ultraviolet filter glass to prevent radiation injury and fading of fabrics • Use in photographic developers and depolarisers • Mining and processing of V-containing ores; extraction from slag • Cleaning and maintenance of furnaces, boilers, and gas turbines
Toxicokinetics and toxicodynamics	**Route of entry: Inhalation** exposure poses greatest risk.

	V is poorly absorbed (2%) from the gastrointestinal tract. Dermal absorption of V compounds is likely to be extremely small. About 25% of soluble V is absorbed. Body burden of 100 – 200 micrograms. V is found in all body tissues. V is transported by transferrin, metabolised to vanadyl and bound to albumin to be transported likely, via phosphate-transport mechanisms. Hence bone is a site of accumulation. The main route of excretion is urine with a half-life of 15-40 hours. Urinary V is preferred for biological monitoring.	
Clinical manifestations of occupational exposure	The exposure to V causes irritation to the respiratory tract, leading to cough, wheezing, chest pain, rhinitis and sore throat. The green discoloration of the tongue is associated with V pentaoxide exposure. These are reversible clinical effects after cessation of exposure. V pentaoxide exposure has been shown to cause bronchial hyperreactivity and occupational asthma. Exposure to V dust can result in eye irritation, conjunctivitis, and skin rashes. IARC classifies V pentaoxide as possibly carcinogenic to humans i.e group 2B.	
Occupational exposures (MSDS)	IDLH STEL OEL 8 Hr TWA-Vanadium pentoxide	35mg V/mg^3 0.1 mg/m^3 (Inhalable fraction) CARC
OCCUPATIONAL EXPOSURE		
Biological Monitoring	**Sample:** Vanadium in urine	**Sample time:** Post shift (PS)
	Vanadium in urine	***Tentative maximum allowable concentration:** 50 ug/g creatinine PS
Biological Effect Monitoring pathology based	Blood	Urea, creatinine, eGFR
	Urine	proteinuria

VINYL CHLORIDE

Chemical Formula	**C_2H_3Cl**
CAS Number	**75-01-4**
Occupational uses	Vinyl chloride (VC) is a colourless, flammable gas with a slightly sweet odour and was previously used as an anaesthetic agent. It is now mainly used for the production of polyvinyl (PVC) pipes in closed systems. Forms highly toxic combustion products such as hydrogen chloride, phosgene, and carbon monoxide.
Toxicokinetics and toxicodynamics	Metabolism is believed to proceed via different pathways, the extent of which is dependent on vinyl chloride concentrations. At low concentrations, vinyl chloride is oxidised sequentially to 2-chloroethanol, 2-chloroacetaldehyde and 2-chloroacetic acid by alcohol dehydrogenase, while at higher concentrations it is metabolised by liver cytochrome P-450 IIE1 to the reactive oxirane, 2 -chloroethylene oxide and its rearrangement product 2-chloroacetaldehyde. Chloroethylene oxide and chloroacetaldehyde react with nucleic acid bases, forming DNA adducts, which are thought to play a role in carcinogenicity of vinyl chloride. Vinyl chloride carcinogenicity occurs via a genotoxic pathway and is understood in some detail. Vinyl chloride is metabolised to a reactive metabolite, probably chloroethylene oxide, which is believed to be the ultimate carcinogenic metabolite of vinyl chloride. Cytochrome P450 2E1

	and glutathione transferase genetic polymorphism have been associated with liver damage susceptibility. The mechanisms of liver cancer include: • Metabolism activation to form CEO • DNA binding of CEO to form exocyclic ethanol adducts • Adduct cause base mutations • Mutations have an effect on proto-oncogenes/tumour suppressor genes at the gene and gene product level. The elimination of vinyl chloride follows first-order kinetics. At low exposure levels, the majority is excreted into the urine, while at higher exposure levels, the proportion of exhaled unmetabolised vinyl chloride increases. Urinary levels of thiodiglycolic acid were correlated with levels of vinyl chloride in the air at concentrations of > 5 ppm. Thiodiglycolic acid levels of 0.3 to 4.0 mg/l have been associated with exposure to air levels of vinyl chloride of 0.14 to 7.0 ppm. Pathologic porphyrinuria, especially secondary coproporphyrin urine with transition to subclinical chronic hepatic porphyria, is a consistent pathobiochemical parameter for the recognition of vinyl chloride hepatic lesions.	
Clinical manifestations of occupational exposure	A severe irritant to skin, eyes, and mucous membranes. Direct skin contact with compressed gas or liquid vinyl chloride can cause frostbite injury. Localised burns or irritation of the conjunctiva and cornea from VC gas has been observed. Exposure to this substance affects the central and peripheral nervous system and causes liver damage. Prolonged exposure to vinyl chloride can cause a set of symptoms that is characterized by Raynaud's phenomenon, joint and muscle pain and scleroderma-like skin changes. The odour of VC becomes detectable at around 3,000 ppm and the OSHA OEL is 2 ppm. As a result, workers can be overexposed to vinyl chloride without being aware of its presence. At 1000 ppm the "VC illness" was described in workers. Symptoms include headache, dizziness, earache, blurred vision, fatigue, nausea, pain in the liver/spleen area, pain and tingling sensation in the arms and legs, loss of appetite and weight loss. Exposure to VC is also associated with hepatomegaly and or splenomegaly. The IARC concluded in 2009 that VC causes angiosarcoma of the liver and hepatocellular carcinoma.	
Occupational exposures (MSDS)	IDLH STEL OEL 8 Hr TWA	Not determined Not determined/5 ppm 1(2) ppm CARC
OCCUPATIONAL EXPOSURE		
Biological Monitoring	**Sample:** Thiodiglycolic acid in urine	**Sample Time:** ES (End of Shift), EWW (End of Work week)
	Thiodiglycolic acid in urine	***BEI (Biological exposure index):** *Not determined ***Not industrial exposed**: 0.0-2.0 mg/l ES, EWW
Biological Effect Monitoring pathology based	Blood	Liver Function, DNA adducts, FBC
	Urine	Urea, creatinine, eGFR and porphyrinuria

XYLENE

Chemical Formula	$C_6H_4 (CH_3)_2$	
CAS Number	**95-47-6**	
Occupational uses	Xylene is used as an industrial solvent, and as raw material in the manufacturing of plasticizers, resins and other products. It occurs in motor car fuel, especially unleaded fuel. Some examples of workers at riks of exposure: • Painters and furniture refinishers who use paint thinners, solvents, lacquers and paint removers • Biomedical laboratory workers who use it as a solvent to fix tissue specimens and rinse stains • Workers involved in distillation and purification of xylene • Workers employed in industries who use xylene as a raw material • Gas station and automobile garage workers through exposure to petroleum products	
Toxicokinetics and toxicodynamics	**Routes of entry: Inhalation(main route), inhalation and dermal absorption** About 60% of the inhaled amount is retained after 8 hours of exposure. Dermal absorption through skin contact with the liquid is also significant. Gastro-intestinal absorption (through ingestion) is rapid. An amount of 3% to 6% of the absorbed Xylene is exhaled in unchanged form. This elimination route is biphasic with half-lives of 1 hour and 20 hours. About 95% of elimination occurs through the urine, after being metabolised to o-, m- and p-methylhippuric acid. This route is also biphasic with half-lives of 3.6 hours and 30 hours. (Alcohol intake or the use of aspirin inhibits this metabolic path by 50%). Xylenes are also deposited in adipose tissue, from whence elimination proceeds slowly.	
Clinical manifestations of occupational exposure	Suppression of the central nervous system, causing nausea, vomiting, dizziness, incoordination, loss of consciousness and even death. Irritation of the mucous membranes (eyes, nose and throat). In workers exposed to high levels of the mixture of organic solvents (much greater than the permissible levels), a linear dose-response relationship has been reported between the exposure level, risk of hearing loss, and hearing threshold at high frequencies, especially 8000 Hz.	
Occupational exposures (MSDS)	IDLH STEL OEL	900 ppm 300 ppm SKIN 200 ppm
OCCUPATIONAL EXPOSURE		
Biological Monitoring	**Sample:** 1. Methylhippuric acid in urine 2. Xylene in blood	**Sampling time:** 1. ES (End of Shift) 2. DS (During Shift)
	1. Methylhippuric acid: creatinine 2. Xylene in blood	**BEI (Biological exposure index):** 1.5 g/g creatinine ES 0.15 mg/100ml DS
Biological Effect Monitoring pathology based	Blood	Full blood count, liver function
	Urine	Combur 9 + creatinine

7. AMPATH TEAM

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8. USEFUL WEBSITES AND LINKS

https://www.ampath.co.za	Ampath
http://www.asosh.org/	ASOSH Website
http://www.cdc.gov/niosh/homepage.html	NIOSH Website
http://www.cdc.gov/	CDC Website
http://www.acgih.org/home.htm	ACGIH Website
http://www.ilo.org/	ILO Website
https://www.icohweb.org/site/code-of-ethics.asp	ICOH code of ethics
http://www.epa.gov/	EPA Website
http://www.hse.gov.uk/	HSE Website
http://www.wcomp.gov.za/	Compensation Commissioner website
http://www.ccohs.com/	CCOHS Website
http://www.fda.gov/	FDA Website
http://www.who.org/	WHO Website
http://www.travelclinic.co.za	Info on travel medicine
http://www.cdc.gov/niosh/nmed/medstart.html	Medical Test selection
http://www.redribbon.co.za	HIV / AIDS site
http://www.epa.gov/iaq/pubs/index.html	Indoor Air Quality
http://www.pp.okstate.edu/ehs/modules/home.htm	On-line SHE training system
http://www.cfia.agr.ca	Food Handlers – WHO
http://www.enviroderm.co.uk	Info on Skin and Occupational Risk
http://www.iarc.fr	International agency for research on cancer
https://monographs.iarc.who.int/list-of-classifications	Monographs IARC
http://www.inchem.org	INCHEM home page
https://www.nlm.nih.gov	US National library of medicine
https://www.nlm.nih.gov/medlineplus	Medline Plus Health information from the National Library of Medicine
https://hazmap.nlm.nih.gov	US National library of medicine
https://www.ncadd.org	National council on alcoholism and drug dependence.INC
https://www.ccohs.ca	Canadian Centre for Occupational Health and Safety
http://www.labourguide.co.za/	The South African Labour Guide
https://oshwiki.eu/wiki/Health_screening_and_surveillance	OSH WIKI
https://www.cdc.gov/niosh/idlh/intridl4.html	IDLH values
https://pubmed.ncbi.nlm.nih.gov/?term=Loukzadeh%20Z%5BAuthor%5D	Effect of Exposure to a Mixture of Organic Solvents on Hearing Thresholds in Petrochemical Industry Workers
https://www.cancer.gov/about-cancer/	National cancer institute
https://www.researchgate.net/publication/287272856_Heavy_metal_contaminants_and_male_reproductive_health	Metals and reproductive health
https://www.cdc.gov/niosh/idlh/65996932.html	Coal tar pitch volatiles (IDLH)
https://www.cdc.gov/niosh/npg/nengapdxc.html	CTPV TWA
https://monographs.iarc.who.int/wp-content/uploads/2019/07/Classifications_by_cancer_site.pdf	List of classifications by cancer sites with sufficient or limited evidence in humans, IARC Monographs Volumes 1–132a

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ANNEXURES
ANNEXURE A

1. CHEMICAL EXPOSURE: BIOLOGICAL MONITORING TESTS

EXPOSURE with METABOLITE	SPECIMEN	MNEMONIC	CONTAINERS
Acetone exposure:			
Acetone	urine	ACET	25ml urine frozen on ice
Aniline exposure:			
Methaemoglobin	blood	MHB	Heparin
Benzene exposure:			
Phenol	urine	BEN	25ml urine frozen on ice
tt Muconic acid	urine	BENT	25ml urine frozen on ice
Phenylmercapturic acid	urine	BENP	25ml urine frozen on ice
Benzene	blood	BENB	EDTA 1 on ice
Carbaryl exposure:			
1-Naphthol	urine	NAPHT	25ml urine frozen on ice
Carbon Disulfide exposure (2-thiothiazolidine):			
TTCA	urine	CDE	25ml urine frozen on ice
Coal tar pitch volatile exposure (Polycyclic aromatic hydrocarbon exposure):			
1-Hydroxypyrene	urine	PAH	25ml urine frozen on ice
Cyanide exposure:			
Cyanide (acute exposure)	blood	CYAN	Fluoride 2 on ice Alternatively EDTA 1 on ice
Thiocyanate	urine	THIOU	25ml urine frozen on ice
Dichloromethane exposure:			
Dichloromethane	blood	DCMB	EDTA 1
Dichloromethane	urine	DCMU	25ml urine frozen on ice
Dimethylformamide exposure:			
N-Methylformamide	urine	DMF	25ml urine frozen on ice. Avoid Alcohol >24hrs
Ethyl benzene exposure:			
Mandelic acid + Phenylglyoxylic acid	urine	EB	25ml urine frozen on ice
Ethyl benzene	blood	EBB	EDTA 1
Ethylene Glycol exposure:			

EXPOSURE with METABOLITE	SPECIMEN	MNEMONIC	CONTAINERS
Oxalic acid	urine	EGE	25ml urine on ice with 20ml HCL
Formaldehyde exposure:			
Formic acid	urine	FOR	25ml urine frozen on ice
Furfural exposure:			
Furoic acid	urine	FUR	25ml urine frozen on ice
Glue and thinners sniffing:			
Toluene	urine	TOLC	25ml urine frozen on ice
Phenylmercapturic acid	urine	BENP	25ml urine frozen on ice
Xylene	urine	XYL	25ml urine frozen on ice
Hexane	urine	HEXAN	25ml urine frozen on ice
Ethylbenzene	urine	EB	25ml urine frozen on ice
Herbicide exposure:			
Paraquat	urine	PARAQ	25ml urine frozen on ice/ Gastric Juice
Hexane exposure:			
2,5 Hexanedione	urine	HEXAN	25ml urine frozen on ice
Isocyanate exposure:			
MDI (methylene diphenyl isocyanate) IgE	serum	ISOM	SST 1
HDI (hexamethylene diisocyanate) IgE	serum	ISOH	SST1
TDI (toluene diisocyanate) IgE	serum	ISOT	SST1
TDI (toluene diisocyanate)	urine	TDI	25ml urine frozen on ice
Isopropanol exposure:			
Acetone	urine	ISOP	25ml urine frozen on ice
Methanol exposure:			
Formic acid	urine	METHE	25ml urine frozen on ice
Methanol	urine	METHU	25ml urine frozen on ice
Methanol	blood	METHB	FLUORIDE 2 Alternative EDTA 1
Methylene chloride(Dichloromethane) exposure:			
Dichloromethane	blood	DCMB	EDTA 2
Carboxyhemoglobin	blood	CHB	HEP 1
Methyl Ethyl ketone (MEK) exposure:			

EXPOSURE with METABOLITE	SPECIMEN	MNEMONIC	CONTAINERS
Methyl Ethyl ketone (MEK)	urine	MEK	25ml urine frozen on ice
Methyl Isobutyl Ketone (MIBK) exposure:			
Methyl Isobutyl Ketone (MIBK)	urine	MIBK	25ml urine frozen on ice
Methyl-n-butyl ketone(MBK) exposure:			
2,5 Hexanedione	urine	MBK	25ml urine frozen on ice
Mono Bromomethane (Methylbromide) exposure:			
Bromide	serum	BR	SST 1
Naphthalene exposure:			
1-Naphthol	urine	NAPHTN	25ml urine frozen on ice
Parathion	blood	CHEWB	EDTA 1
Pesticide/Insecticide exposure:			
Organophosphates, Carbamates			
ChE Whole blood	blood	CHEWB	EDTA 1
WB ChE/Hb Ratio	blood	CHEWB + HB	EDTA 2
Pseudocholinesterase – serum (ChE)	serum	CHS	SST 1
Pentachlorophenol exposure:			
Pentachlorophenol	urine	PCPE	25ml urine frozen on ice
Phenol exposure:			
Phenol	urine	PHENOL	25ml urine frozen on ice
Polycyclic aromatic hydrocarbon exposure (Coal tar pitch volatile exposure):			
1-Hydroxypyrene	urine	PAH	25ml urine frozen on ice
Styrene exposure:			
Styrene	blood	STYRB	EDTA 1
Mandelic acid & Phenylglyoxylic acid	urine	STYRU	25ml urine frozen on ice
Tetrachlorethylene (perchloroethylene) exposure:			
Trichloroacetic acid	urine	TETRA	25ml urine frozen on ice
Tetrachloroethylene	blood	TETRAB	EDTA 1
Thiocyanate exposure:			
Thiocyanate	urine	THIOU	25ml urine frozen on ice
Trichloroethylene exposure:			

EXPOSURE with METABOLITE	SPECIMEN	MNEMONIC	CONTAINERS
Total trichloro compounds & Trichloroacetic Acid & Trichloroethanol	urine	TRIC	25ml urine frozen on ice
Trichloroacetic acid	urine	TRIA	25ml urine frozen on ice
Total trichloro compounds	urine	TRITTC	25ml urine frozen on ice
Trichloroethylene	blood	TRIB	EDTA 1
Trichloroethane exposure (Methyl chloroform):			
Trichloroacetic acid	urine	TRIMCU	25ml urine frozen on ice
Trichloroethane & Trichloroethanol	blood	TRIMCB	EDTA 2
Trinitrotoluene exposure			
Total Amino - dinitrotoluene	urine	TNTE	25ml urine frozen on ice
Toluene Diisocyanate exposure:			
Isocyanates - Toluenediamine	urine	TDI	25ml urine frozen on ice
TDI (toluene diisocyanate) IgE	serum	ISOT	SST1
Toluene exposure (Hippuric acid & O-Cresol):	urine	TOLCH	25ml urine frozen on ice
Hippuric acid	urine	TOLH	25ml urine frozen on ice
Ortho cresol	urine	TOLC	25ml urine frozen on ice
Toluene	blood	TOLB	EDTA 1
Vinyl Chloride exposure:			
Thiodiglycolic acid	urine	VCE	25ml urine on ice
Xylene exposure:			
Methylhippuric acid	urine	XYL	25ml urine frozen on ice
Xylene	blood	XYLB	EDTA 1

2. METAL EXPOSURE: BIOLOGICAL MONITORING TESTS

METAL	SPECIMEN	MNEMONIC	CONTAINER
Aluminium (Al)	urine	ALU	25ml urine
	serum	AL	A04 - Royal Blue Trace Metal Free Serum Tube & Metal Free Plastic Transport Tube
Antimony (Sb)	urine	SBU	25ml urine
Arsenic (As)	urine	ASU	25ml urine
	blood	ASB	EDTA 1
Bromide(B)	blood	BR	SST 1
Cadmium (Cd)	urine	CDU	25ml urine
	blood	CD	Heparin 1
Chromium (Cr)	urine	CHU	25ml urine
	blood	CRB	EDTA 1
Cobalt (Co)	urine	COU	25ml urine
	blood	CO	EDTA 1
Copper (Cu)	urine	CUEX	25ml urine
	blood	CU	SST 1
Fluoride (Fl) random	urine	FLU	25ml urine
Fluoride (Fl) pre shift	urine	FLPREU	25ml urine
Fluoride (Fl) post shift	urine	FLPOSTU	25ml urine
Lead (Pb)	urine	PBU	25ml urine
	blood	PBB	EDTA 1
Manganese (Mn)	urine	MNU	25ml urine
	blood	MNB	EDTA 1
Mercury (Hg)	blood	HGB	EDTA 1
	urine	HGU	25ml urine
Molybdenum (Mo)	urine	MOU	25ml urine
Nickel (Ni)	urine	NIU	25ml urine
	blood	NI	EDTA 1
Platinum (Pt)	urine	PTU	25ml urine
	blood	PTB	EDTA 1
Selenium (Se)	urine	SEU	25ml urine
	serum	SE	A04 - Royal Blue Trace Metal Free Serum Tube & Metal Free Plastic Transport Tube

METAL	SPECIMEN	MNEMONIC	CONTAINER
Thallium (Tl)	urine	TLU	25ml urine
Uranium(U)	urine	URANU	25ml urine
Titanium (Ti)	urine	TIU	25ml urine
	blood	TI	K02 Royal Blue Trace Metal Free EDTA Tube
Vanadium (V)	urine	VU	25ml urine
Zinc (Zn)	urine	ZNU	25ml urine
	plasma	ZN	Heparin 1
Heavy Metal profile (includes: As, Hg, Cd, Co, Pb, Cr)	urine	HMPIND	25ml urine

3. BIOLOGICAL EFFECT MONITORING TESTS

TEST	SPECIMEN	MNEMONIC	CONTAINER
Creatinine	serum	CR	SST 1
AST (SGOT)	serum	AST	SST 1
ALT (SGPT)	serum	ALT	SST 1
ALP (Alk. Phosphatase)	serum	ALP	SST 1
Gamma GT	serum	GGT	SST 1
Liver enzymes only	serum	LEN	SST 1
Liver functions	serum	LF	SST 1
Full Blood count & PLT	blood	FBC	EDTA 1
Haemoglobin	blood	HB	EDTA 1
Urea & Electrolytes	serum	UE	SST 1
Dipstick	urine	URCHEM	Random urine 25ml
Creatinine	urine	CRU	Random urine 25ml
Total Protein & Creatinine	urine	TPU	Random urine 25ml
Total Protein & Creatinine Excretion	urine	TPU24	24hr urine
Albumin excretion	urine	MAU24	24hr urine

4. FOOD HANDLERS SCREENING

TEST	SPECIMEN	MNEMONIC
Staphylococcus Aureus Screening	Nose/hand swab	SAUR
Salmonella/Shigella Culture	Rectal swab	STSS
Hepatitis A IgG	Serum	HEPAG

5. ANTIBODY TESTING

TEST	SPECIMEN	MNEMONIC
COVID19 Antibody	Nasal swab	COVID19AB
Hepatitis A Antibody	Serum	HEPA
Hepatitis B Antibody	Serum	HEPBSAB
Hepatitis C Antibody	Serum	HEPC
Hepatitis Immunity	Serum	HEPIM

ANNEXURE B
APPLICATION OF BIOLOGICAL EXPOSURE INDEX (BEI)

BEIs are intended as guidelines to be used in the evaluation of potential health hazards in the practice of occupational hygiene. BEIs do not indicate a sharp distinction between hazardous and non-hazardous exposures. For example, it is possible for an individual's determinant concentration to exceed the BEI without incurring an increased health risk. If measurements in specimens obtained from a worker on different occasions persistently exceed the BEI, the cause of the excessive value should be investigated and action taken to reduce the exposure. An investigation is also warranted if the majority of the measurements in specimens obtained from a group of workers at the same workplace and work shift exceed the BEI. It is desirable that relevant information on related operations in the workplace be recorded.

Due to the variable nature of concentrations in biological specimens, dependance should not be placed on the results of one single specimen. Administrative action should not normally be based on a single isolated measurement, but on measurements of multiple sampling, or an analysis of a repeat specimen. It may be appropriate to remove the worker from exposure following a single high result if there is a reason to believe that significant exposure may have occurred. Conversely, observations below the BEI do not necessarily indicate a lack of health risk.

BEIs apply to 8 hour exposures, five days per week. Although modified work schedules are sometimes used in various occupations, the BEI Committee does not recommend that any adjustment or correction factor be applied to the BEIs (i.e. the BEIs should be used as listed, regardless of the work schedule).
The BEIs should be applied by a knowledgeable occupational health professional. Toxicokinetic and toxicodynamic information is taken into account when establishing the BEI; thus, some knowledge of the metabolism, distribution, accumulation, excretion, and effect(s) is helpful in using the BEI effectively. The BEI is a guideline for the control of potential health hazards to the worker and should not be used for other purposes. The values are inappropriate to use for the general population or for no occupational exposures. The BEI values are neither rigid lines between safe and dangerous concentrations nor an index of toxicity. The BEI values are available in the Regulations for Hazardous Chemical Agents GNR.780 of 29 March 2021 in Table 4 of the regulation.

Where no BEI is available from the regulation, reference values are sourced from publications and reported but companies should, in these cases, decide which values will be used to action possible exposure.

TABLE 4: BIOLOGICAL EXPOSURE INDICES FOR HAZARDOUS CHEMICAL AGENTS

CHEMICAL	METABOLITE/SUBSTANCE MEASURED	SAMPLING TIME	VALUE	UNIT	NOTATION
Acetone	Acetone in urine	ES	25	mg/l	Ns
Acetylcholinester-ase inhibitors	Cholinesterase activity in whole blood	D	70	% of baseline	Ns
Aniline	Total p-Aminophenol in urine	ES	50	mg/l	B, Ns, Sq
Arsenic and soluble inorganic compounds	Inorganic arsenic metabolites in urine	EWW	35	ug/l	B
Benzene	S-Phenylmercapturic acid (SPMA)in urine	ES	25	ug/g creatinine	B
	tt-Muconic acid (TTMA)in urine	ES	500	ug/g creatinine	B
1,3 **B**utadiene	1,2 Dihydroxy-4-(N-acetylcysteinyl)-butane urine	ES	2.5	mg/l	B, Sq
	Mixture of N-1-and N-2-(hydroxybutenyl) valine haemoglobin adducts	NC	2.5	pmol/g Hb	Sq
2-**B**utoxyethanol	Butoxyacetic acid (BAA)	ES	200	mg/g creatinine	
Cadmium and inorganic compounds	Cadmium in urine	NC	5	ug/g creatinine	B
	Cadmium in blood	NC	5	ug/l	B
Carbon disulphide	2-Thiothiazolidine-4-carboxylic acid in urine (TTCA)	ES	0.5	mg/g creatinine	B,Ns
Carbon monoxide	Carboxyhemoglobin in blood	ES	3.5	% haemo-globin	B, Ns
	Carbon monoxide air	ES	20	ppm	B, Ns
Chlorobenzene	4-Chlorocatechol in urine	ES, EWW	100	mg/g creatinine	
	p-Clorophenol in urine	ES, EWW	20	mg/g creatinine	

CHEMICAL	METABOLITE/SUBSTANCE MEASURED	SAMPLING TIME	VALUE	UNIT	NOTATION
Cobalt and inorganic compounds, including cobalt oxides but not combined with tungsten carbide	Cobalt	ES, EWW	15	ug/l	Ns
Cyclohexanone	1,2 Cyclohexanediol in urine Cyclohexanol in urine	ES, EWW ES	80 8	mg/l mg/l	Ns, Sq Ns, Sq
Dichloromethane	Dichloromethane urine	ES	0.3	mg/l	Sq
N,N-**D**imethyl-acetamide	N-Methylacetamide in urine	ES, EWW	30	mg/g creatinine	
N,N-**D**imethylformamide (DMF)	N-Methylformamide in urine N-Acetyl-S-(N-methylcarbamoyl) cysteine in urine	ES PLSWW	15 40	mg/l mg/l	Sq
2-**E**thoxyethanol (EGEE) and 2-Ethoxyethyl acetate (EGEEA)	2-Ethoxyacetic acid in urine	ES, EWW	100	mg/g creatinine	
Ethyl benzene	Sum of mandelic acid and phenylglyoxylic acid in urine	ES	0.15	g/g creatinine	Ns
Fluorides	Fluorides in urine	PS ES	2 3	mg/l mg/l	B, Ns B, Ns
Furfural	Furoic acid in urine	ES	200	mg/l	Ns
1,6-**H**examethylene diisocyanate	1,6-Hexamethylene diamine in urine	ES	15	ug/g creatinine	
n-**H**exane	2,5-Hexanedione in urine	ES, EWW	0.4	mg/l	
Lead	Lead Blood	NC	See Lead regulations		
Mercury (Elemental)	Mercury in urine	PS	20	ug/g creatinine	
Methanol	Methanol in urine	ES	15	mg/l	B, Ns
Methemoglobin inducers	Methemoglobin in blood	During/ES	1.5	% haemo-globin	B, Ns, Sq
2-**M**ethoxyethanol and 2-**M**ethoxyethyl Acetate	2-Methoxyacetic acid in urine	ES, EWW	1	mg/g creatinine	
Methyl n-butyl ketone	2,5 Hexanedione in urine	ES, EWW	0.4	mg/l	

CHEMICAL	METABOLITE/SUBSTANCE MEASURED	SAMPLING TIME	VALUE	UNIT	NOTATION
Methyl chloroform (Trichloroethane)	Methyl chloroform in end-exhaled air	PLSWW	40	ppm	
	Trichloroacetic acid in urine	EWW	10	mg/l	Ns, Sq
	Total trichloro-ethanol in urine	ES, EWW	30	mg/l	Ns, Sq
	Total trichloroethanol in blood	ES, EWW	1	mg/l	Ns
Methyl ethyl ketone (MEK)	Methyl ethyl ketone (MEK) in urine	ES	2	mg/l	Ns
Methyl isobutyl ketone (MIBK)	Methyl isobutyl ketone (MIBK) in urine	ES	1	mg/l	
Nitrobenzene	Methaemoglobin in blood	During /ES	1.5	% haemo-globin	B, Ns, Sq
Parathion	Total p-Nitrophenol in urine	ES	0.5	mg/g creatinine	Ns
	Cholinesterase activity in red cells	D	70	% of baseline	B, Ns, Sq
Phenol	Phenol in urine	ES	250	mg/g creatinine	B,Ns
2-**P**ropanol	Acetone in urine	ES,EWW	40	mg/l	B, Ns
Styrene	Mandelic acid in urine AND Phenylglyoxylic acid in urine	ES	400	mg/g creatinine	Ns
	Styrene in venous blood	ES	40	mg/l	
Tetrachloroethylene (Perchloroethylene)	Tetrachloroethylene end exhaled	PS	3	Ppm	
	Tetrachloroethylene in urine	PS	0.5	mg/l	
Tetrahydrofuran	Tetrahydrofuran in urine	ES	2	mg/l	
Toluene	Toluene in urine	ES	0.03	mg/l	
	Toluene in venous blood	PLSWW	0.02	mg/l	
	o-Cresol in urine	ES	0.3	mg/g creatinine	B
Toluene diisocyanate-2,4 or as a mixture of isomers	Toluene diamine	ES	5	ug/g creatinine	Ns
Trichloroethylene	Trichloroacetic acid in urine	ES, EWW	15	mg/l	Ns
	Free trichloroethanol in blood	ES, EWW	0.5	mg/l	Ns
Uranium	Uranium in urine	ES	200	ug/l	
Xylene	Methylhippuric acid in urine	ES	1.5	g/g creatinine	

INTERPRETATION OF THE RESULTS

The biological monitoring test must be interpreted according to our current knowledge of the relationships between external exposure, internal exposure and the risk of adverse health effects and on which basis the biological reference values (BEI's) have been established. The finding of a biological level above the reference value may only be a qualitative indication of exposure to a substance. If the quantitative relationship between external exposure and the internal dose is known, the biological parameter can be used as an index of exposure but provides little information on the health risk. In other words, biological monitoring performed under these conditions is much more an assessment of the exposure intensity than of the potential health risk. In some situations, a quantitative relationship has been identified between internal dose and adverse health effects. The biological parameter can, in these cases, be considered an indicator of health risk. It is also possible to derive a biological permissible value from this dose-effect relationship. When the internal dose is quantitatively related to adverse effects and external exposure, the biological parameter provides information on both exposure and health risk. Sometimes, the relationship between internal dose and effect is unknown, but the internal dose can be related to external exposure and indirectly to the adverse effects. A biological permissible value can be estimated indirectly from the exposure limit in air. It is clear, however, that this method of deriving the biological limit value is much less reliable than a direct estimation based on the relationship between internal dose and adverse effect. Finally, if all the parameters are quantitatively related, both the biological and environmental exposure limits can be directly estimated.

So far, the majority of published works have focused on the internal dose-external exposure relationships established in volunteers or in industrial workers. The relationships between internal dose and early adverse effects, which are essential for deriving meaningful biological limit values, are comparatively less well documented.

In cases where there is currently no known relation between the biological index and exposure (e.g., when the main route of exposure is through the skin) or health effect, it could be appropriate to set a biological monitoring guideline that is related to what level is being currently achieved across industry. A possible approach would be to set a guideline that was being achieved in 90% of employees. This approach may sometimes be supplemented by animal pharmacokinetic and effects data which are more easily generated. The relationship between internal concentration and adverse health effects may be known in the future only if biological monitoring is conducted in the present. In the future at least, epidemiological studies could be carried out to assess whether the present levels of exposure were low enough.

The results of a biological monitoring program can be interpreted on an individual basis. This is usually performed by the occupational health physician (occupational medical practitioner) who must also take into account several possible individual compounding factors. For instance, liver function impairment may be associated with a decrease in xenobiotics (chemical) biotransformation. Several drugs may either increase or decrease liver microsomal enzyme activity and hence influence xenobiotics biotransformation. Likewise, alcohol consumption may interfere with the metabolism of various substances (e.g., methanol, toluene, xylene, and styrene) in two opposite ways. Moderate chronic intake of ethanol usually stimulates drug-metabolising enzymes and hence the biotransformation of other absorbed chemical agents, whereas during or shortly after a large alcohol intake entailing a

high concentration of alcohol in the body, there appears to be an inhibitory effect on the metabolism of xenobiotics. Perturbation of renal clearance, large or restricted beverage intake, may be responsible for misinterpretation of urinary results. Tobacco smoke containing many substances (e.g., cadmium, carbon monoxide) can also be a serious con- founding factor. For example, smoking influences the concentration of thioethers in urine as well as the mutagenic activity thereof. Exposure from diet, environment and leisure activities may sometimes be of importance.

In the occupational setting there is often exposure to a mixture of substances. This may entail variations in terms of toxicokinetic and toxicodynamic processes. When interpreting the results, one must consider the possible physicochemical interactions between the substances, the effect that one agent may have on the absorption, metabolism, excretion of the other, and the possibility of interactions between the parent compound and the metabolites. The effect may be (i) independent, where the substances exert their own toxicity independent of each other, (ii) additive, where the combined effect of the two chemicals is equal to the sum of the effects produced by the individual agents, (iii) synergistic, where the combined effect of the two chemicals is much greater than the sum of the effects of each agent given separately, (iv) antagonistic, where two chemicals administered together interfere with each other, or (v) potentiating, where a substance of low or no toxicity enhances the toxicity of another chemical.

Results are generally interpreted through comparison to adequate reference values. However, because of the difference in individual susceptibility, the threshold values above which an adverse effect will occur, will differ between the subjects. A biological reference value for occupationally exposed people is not, therefore, an assurance that it will protect all the exposed persons from adverse health effects. In some susceptible individuals, a biological response may occur even with exposure below these reference values. When there is considerable inter-individual variability for a certain parameter, the post-exposure level may be better interpreted through comparison to the individual pre-exposure level (the baseline value). For example, the cholinesterase activity of red blood cells used as an index of exposure to organophosphates or carbamates should preferably be expressed as a percentage of the individual baseline activity. Similarly, for cumulative industrial chemicals it is recommended that the baseline internal dose be established before the subjects are exposed to these substances.

The results can also be interpreted on a group basis by considering their distribution. If all the observed values are below the biological permissible value, the working conditions are satisfactory. If all or the majority of the results are above the biological permissible value, the overall exposure conditions must certainly be corrected. A third situation may also occur: the majority of the workers may have values below the biological permissible level but a few of them have abnormally high values. Several interpretations can be put forward. One interpretation is that the subjects exhibiting the high values perform activities exposing them to higher levels of the pollutant, in which case the biological monitoring program has identified job categories for which work conditions need to be improved. Another interpretation is that these workers do not perform different activities and, in this case, their higher internal dose must result from different hygiene habits; non-occupational exposure or genetic polymorphism.

REFERENCE

- Regulations for Hazardous Chemical Agents GNR.280 dated 29 March 2021
- American Conference of Governmental Industrial Hygienists. TLVs® and BEIs®. Based on the Documentation of the Threshold Limit values for chemical substances and Physical agents & Biological exposure indices 2021

ANNEXURE C

EFFECT MONITORING TEST INTERPRETATION

General notes

- The reference ranges provided are applicable to adults only.
- The reference ranges provided may vary according to instrument and methodology.
- Only the most common causes are listed in the interpretation of analytes.

BIOCHEMISTRY

Electrolytes and renal function

Analyte	Ref. Range	Units	Interpretation
Sodium	136 - 145	mmol/l	↑ Diabetes insipidus, dehydration (water loss in excess of salt loss). ↓ Drugs (e.g. diuretics, indapamide), vomiting, diarrhoea, acute renal failure, congestive heart failure, Addison's disease, syndrome of inappropriate ADH secretion, falsely decreased (due to ↑ protein, ↑triglycerides).
Potassium	3.5 - 5.1	mmol/l	↑ Acute renal failure, falsely elevated (haemolysed blood, aged blood, contamination with FBC tube anticoagulant), drugs (e.g. ACE inhibitors, spironolactone, amiloride), Addison's disease, acidosis, untreated diabetic ketoacidosis. ↓ Vomiting, diarrhoea, drugs (e.g., diuretics, indapamide, laxatives).
Chloride	98 - 107	mmol/l	↑ Diuretics, vomiting. ↓ Diarrhoea, dehydration.
Bicarbonate (TCO_2)	22 - 29	mmol/l	↑ Potassium depletion, vomiting, diuretics, emphysema. ↓ Acute renal failure, diabetic ketoacidosis, diabetic hyperosmolar coma, diarrhoea, renal tubular acidosis, lactic acidosis, toxins.
Urea	<8.4	mmol/l	↑ Acute or chronic renal failure, dehydration (due to vomiting, diarrhoea, sweating), intestinal bleeding, shock. ↓ Hepatic failure, pregnancy, cachexia.
Creatinine	M 64 - 104 F 49 - 90	µmol/l	↑ Acute or chronic renal failure, acromegaly, meat meals, hyperthyroidism. ↓ Pregnancy, chronic muscle wasting, immobilisation.
Urate	M 0.21- 0.43 F 0.16 - 0.36	mmol/l	↑ Gout, renal failure, insulin resistance syndrome, alcoholism, malignancies (e.g. leukaemia, lymphoma, multiple myeloma), ↓ Psoriasis, drugs (e.g. diuretics, salicylates, etc). Syndrome of inappropriate ADH secretion, pregnancy.
eGFR	>90	ml/min/ 1.73m²	↓ eGFR = estimated (calculated) Glomerular Filtration Rate: Renal impairment

Analyte	Ref. Range	Units	Interpretation
Urine protein (24-hour urine)	<0.15	g/24 h	↓ Cystitis, pyelonephritis, glomerular disease, tubular renal disease, nephrotic syndrome, Diabetes Mellitus, fever, strenuous exercise, orthostatic changes, rhabdomyolysis.
Urine Creatinine	M 3.5 - 22.9 F 2.5 - 19.2	mmol/l	↓ Urine creatinine <2.5 mmol/ in females and <3.5 mmol/l in males indicates dilute urine, which may decrease detection of analyte/s.

Liver function tests

Analyte	Ref. Range	Units	Interpretation
Total Protein	60-83	g/l	↑ Multiple myeloma, autoimmune disease, chronic liver disease, chronic infection (e.g., AIDS, TB). ↓ Nephrotic syndrome, chronic liver failure, malnutrition, pregnancy.
Albumin	35-52	g/l	↑ Dehydration, prolonged tourniquet during venipuncture. ↓ Acute and chronic liver disease, malnutrition, malabsorption, nephrotic syndrome, acute and chronic inflammation, systemic infections, autoimmune disease, congestive cardiac failure, pregnancy.
Total Bilirubin	0-21	µmol/l	↑ Hepatocellular damage (e.g., hepatitis, toxic damage due to drugs or toxins), intrahepatic biliary tree obstruction (e.g., primary biliary cirrhosis), extrahepatic biliary tree obstruction (e.g., gallstones, carcinoma of the head of the pancreas), haemolytic diseases, Gilbert's disease.
Conjugated Bilirubin	0-5	µmol/l	↑ Hepatocellular damage (e.g., hepatitis, toxic damage due to drugs or toxins), intrahepatic biliary tree obstruction (e.g., primary biliary cirrhosis), extrahepatic biliary tree obstruction (e.g., gallstones, carcinoma of the head of the Pancreas).
Unconjugated Bilirubin	0-18	µmol/l	Mainly increased unconjugated bilirubin: ↑ Gilbert's disease, haemolytic diseases. Increased unconjugated as well as conjugated bilirubin: Hepatocellular damage, intrahepatic and extrahepatic biliary tree obstruction.
Alkaline Phosphatase (ALP)	M: 40-130 F: 35-105	U/l	↑ Primary and secondary hyperparathyroidism, extrahepatic biliary tree obstruction (e.g., gallstones, carcinoma of the head of the pancreas), intrahepatic biliary tree obstruction (e.g., primary biliary cirrhosis), hepatocellular disease (e.g., hepatitis), space occupying lesions in the liver (e.g., liver metastases), bone metastases, Paget's disease of bone, uremic osteodystrophy, thyrotoxicosis, during healing of a fracture, pregnancy.
Gamma Glutamyl-transferase (GGT)	M <60 F <40	U/l	↑ Extrahepatic biliary tree obstruction (e.g., gallstones, carcinoma of the head of the pancreas), intrahepatic biliary tree obstruction (e.g., primary biliary cirrhosis), hepatocellular disease (e.g., hepatitis), fatty liver, space occupying lesions in the liver (e.g., liver metastases), induction by alcohol or medication.

Analyte	Ref. Range	Units	Interpretation
Alanine amino Transferase (ALT)	M <50 F <35	U/l	↑ Acute hepatitis, chronic hepatitis, liver cirrhosis, liver cell necrosis (e.g., hypoxic shock, paracetamol overdose), viraemia, chronic alcohol abuse, liver cirrhosis, fatty liver.
Aspartate amino-Transferase (AST)	M <38 F <32	U/l	↑ Acute hepatitis, acute liver cell necrosis (e.g., hypoxic shock, paracetamol overdose), chronic hepatitis, liver cirrhosis, chronic alcohol abuse (AST:ALT ratio >2), intrahepatic neoplasms, viraemia, fatty liver, haemolytic anaemia, megaloblastic anaemia, rhabdomyolysis, vigorous exercise, muscular dystrophy.
Lactate dehydrogenase (LD)	100-250	U/l	↑ Megaloblastic anaemia, haemolytic anaemia, leukaemia, acute hepatitis, acute liver cell necrosis, liver cirrhosis, skeletomuscular disease, vigorous exercise, rhabdomyolysis, neoplastic disease, myocardial infarction.

HAEMATOLOGY

Full blood count (FBC)

Analyte	Ref. Range	Units	Interpretation
Haemoglobin	Inland: M 14.3 - 18.3 Sea level: M 13.0 - 17.0	g/dl	↑ Polycythaemia. ↓ Anaemia (bleeding, dietary deficiencies, malabsorption, chronic illness, haemolysis and bone marrow failure (inherited or acquired).
	Inland: F 12.1 - 16.3 Sea level: F 12.0 - 15.0	g/dl	
Red cell count	Inland: M 4.89 - 6.11 Sea level: M 4.50 - 5.50	10^{12}/l	↑ Polycythaemia, thalassemia. ↓ Anaemia.
	Inland: F 4.13 - 5.67 Sea level: F 3.80 - 4.80	10^{12}/l	
Haematocrit	Inland: M 43.0 - 55.0 Sea level: M 40.0 - 50.0	%	↑ Polycythaemia. ↓ Anaemia.
	Inland: F 37.0 - 49.0 Sea level: F 36.0 - 46.0	%	
MCV (mean corpuscular volume)	79.1 - 98	fl	↑ Macrocytic red cells. Check peripheral blood smear for round or oval macrocytes. Oval macrocytes are associated with megaloblastic anaemia (vit B12 / folate deficiency). Round macrocytes are associated with liver disease, ↓ Hypothyroidism, antiretroviral therapy, alcohol, chemotherapy, reticulocytosis and myelodysplasia. Microcytic red cells

Analyte	Ref. Range	Units	Interpretation
			Iron deficiency, thalassaemia, other haemoglobin defects, anaemia of chronic disease, lead poisoning, sideroblastic anaemia.
MCH (mean corpuscular haemoglobin)	27.0 - 32.0	pg.	↑ Hyperchromatic red cells e.g. spherocytes. ↓ Hypochromic (pale) red cells (causes as for microcytic cells).
MCHC (mean corpuscular haemoglobin concentration)	32.0 - 36.0	g/dl	↑ Spherocytes, Bilirubinemia, auto-agglutination, lipaemic sample.
Red cell distribution width (RDW)	12.0 - 14.5	%	If raised, indicates red cells of different sizes. Often the earliest sign of iron deficiency.
White cell count	3.92 - 9.88	10^9/l	If abnormal, evaluate the differential white cell count.
Neutrophils	2.0 - 7.5	10^9/l	↑ Neutrophil leucocytosis: Bacterial infection, inflammation, trauma/surgery, neoplasia, haemorrhage, haemolysis, pregnancy, metabolic e.g. diabetic ketoacidosis, drugs e.g., steroids, growth factor therapy e.g., G-CSF. Neutropenia: Decreased production: ↓ General bone marrow failure e.g., aplastic anaemia, acute leukaemia. Specific failure of neutrophil production e.g. congenital, cyclical, drug induced, peripheral loss e.g. hypersplenism, autoimmune, severe infection.
Lymphocytes	1.0 - 4.0	10^9/l	↑ Lymphocytosis: Primary causes: Lymphoproliferative disorders. e.g. CLL, lymphoma. Reactive causes: viral infections, *Bordetella pertussis*, stress lymphocytosis (e.g., myocardial infarction, surgery, trauma), smoking, post splenectomy and auto-immune disorders. ↓ Lymphopenia: Inherited: congenital immunodeficiencies. Acquired: e.g., viral infections, TB, lymphoma, aplastic anaemia, immunosuppressive therapy, radiation, renal failure, auto-immune diseases.
Monocytes	0.18 – 1.00	10^9/l	↑ Monocytosis: Infections e.g., TB, CMV, subacute bacterial endocarditis, syphilis Inflammatory and immune disorders e.g. SLE, RA, ulcerative colitis, sarcoidosis. Haematological malignancies e.g., CMML, AML. Non haematological malignancies. Chronic neutropenias. Monocytopenia: ↓ Haematological disorders e.g., Aplasia, hairy cell leukaemia, Auto-immune disorders e.g. RA, SLE, HIV.
Eosinophils	0.0 - 0.45	10^9/l	↑ Eosinophilia: Allergy e.g. asthma, parasites, skin disease, drug sensitivity, connective tissue disease, Hodgkin lymphoma, chronic myeloproliferative disorders, hypereosinophilic syndrome.
Basophils	0.0 - 0.2	10^9/l	Usually increased in chronic myeloproliferative disorders e.g. chronic myeloid leukaemia.

ANNEXURE D
DIAGNOSIS AND INVESTIGATION OF OCCUPATIONAL EXPOSURE: A GENERAL REVIEW

Exposure in the workplace presents serious and significant health risks. The hazards that chemicals and metals present are a function of their toxic properties and include the duration, dose and route of exposure, and health history of the individuals exposed to them. Controlling and preventing exposures often involves a multidisciplinary team, usually beginning with the primary health care provider. Many strategies exist to this end and include screening and surveillance of exposures, public education and awareness programs, environmental control of exposures, availability of adequate and accessible employee health services, worker safety programs and medical programs. In general, the clinical suspicion of occupationally related diseases is very low. These are frequently undiagnosed as a result of poor occupational history.

A. OCCUPATIONAL HISTORY

Obtaining such history does not require detailed knowledge of Toxicology.
In seeking history, the health worker should consider all possible exposures that may have occurred in occupational activities and in the community where the patient lived/s and/or worked/s.

Taking an exposure history involves gathering information about the individual's work activities. Below is an approach to good occupational history recording.

(a) Current job of the patient—job title, type or nature of work, and any protective equipment on the job.
(b) Patient's perception whether or not their presenting symptoms are related to their work or the environment they live in.
(c) Information on whether others at home or work present with similar problems,
(d) Employment history and chronology of jobs held; temporal relationship is explored.
(e) Relationship between work and health problems.
(f) Environmental (non-occupational) exposures: hobbies, smoking, household, herbal products and community.
(g) Specific environmental and/or occupational exposures: Fumes, dust, metals and chemicals.
(h) History of any comorbid conditions.

B. CLINICAL EXAMINATION AND INVESTIGATIONS

Medical practitioners do not require special skills to diagnose occupational and environmental health problems. A practical approach to examination and tests is useful in day-to-day practice. As most metals and chemicals affect multiple organs and systems, it is recommended to conduct a complete systemic examination with a special focus on blood, cardiac, gastrointestinal, lung, liver, central nervous system and kidney.
Laboratory testing should include the following:

1. Full Blood count,
2. Urine analysis,
3. Kidney function and
4. Liver function tests.

Chest X-ray and pulmonary function, ECG, and allergy testing may be performed where relevant. The determination of metals and chemicals in blood, urine, and tissues are used to confirm the diagnosis. It should be noted that generally each metal/chemical produces a constellation of symptoms and a clinical picture unique to them.

C. EFFECTS OF OCCUPATIONAL EXPOSURE ON REPRODUCTION AND FERTILITY, CANCER CAUSING AND IMMUNOLOGY (ALLERGIES)

REPRODUCTION AND FERTILITY

Other effects of occupational exposure specifically by metals and chemicals on the human body must be included in the investigation of possible occupational disease. In the case of reproductive system, this may manifest in altered sex hormone levels (endocrine disruptors), diminished libido and potency, menstrual disorders, premature menopause, delayed menarche, ovarian dysfunction, impairment of semen quality and reduced fertility. Toxic exposures can cause direct cell damage in the developing sperm and eggs. Cell damage may also be in the form of chromosomal abnormalities and gene mutations. The exposure dose is important, a low dose resulting in birth defects and a high exposure dose cab resulting in miscarriage or infertility. Maternal exposure during pregnancy may disturb foetal development by either directly or indirectly interfering with maternal, placental or foetal membrane functions.

Toxic exposures can induce many wide-ranging effects, such as foetal death, miscarriages (exposures in first trimester) intrauterine growth retardation, preterm birth, birth defect, postnatal death, disturbances in cognitive development and changes in immunological sensitivity, or childhood cancer. The mother's exposure at work to chemicals may also cause contamination of her breast milk.

The table details the occupational exposures that have been associated with reduced fertility and/or on biological indicators of reproductive functions as semen quality or endocrine disruption.

OCCUPATIONAL EXPOSURE	MEN	WOMEN
Arsenic	+	+
Benzene		+
Cadmium		+
Carbon disulphide	+	+
Chlorinated hydrocarbons such as DDT and trichloroethylene	+	+
Chromium compounds		+
Dinitrotoluene and toluene diamine	+	
Ethylene Glycol ethers	+	+
Halogenated hydrocarbons such as Tetrachloroethylene	+	+
Lead	+	+
Manganese	+	

OCCUPATIONAL EXPOSURE	MEN	WOMEN
Mercury	+	+
Polychlorinated biphenyls (PCB)	+	+
Pesticides	+	+
Styrene	+	

D. EFFECTS OF OCCUPATIONAL EXPOSURE: CARCINOGENS

Occupational carcinogens have an important role in the identification and prevention of cancer. They were the first human carcinogens identified and a large proportion of the carcinogens currently identified originate in the workplace. The IARC monograph series is the reference source for carcinogens. Agents are grouped into the following five categories.

GROUP 1	Carcinogenic to humans
GROUP 2A	Probably carcinogenic to humans
GROUP 2B	Possibly carcinogenic to humans
GROUP 3	Not classifiable as to its carcinogenicity to humans
GROUP 4	Probably not carcinogenic to humans

Target sites associated with occupational carcinogen exposure:

OCCUPATIONAL EXPOSURE	TARGET SITES
Arsenic	Lung, skin, bladder, liver, kidney, prostate
Benzene	Leukaemia
Beryllium	Lung
Cadmium	Lung, prostate, kidney
Chromium [hexavalent]	Lung, nasal cavity ,paranasal sinuses
Coal Tar Pitch volatiles	Lung, kidney, skin, bladder
Formaldehyde	Paranasal sinuses, nasal cavity & nasopharynx
Isopropyl alcohol	Paranasal sinuses
Nickel compounds	Lung, nasal cavity, paranasal sinuses
Trichloroethylene	Kidney
Vinyl chloride	Liver, brain, lung, lymphoma and leukaemia

Substances and mixtures that have been evaluated by IARC as probable (GROUP 2A) human carcinogens and that are occupational exposures:

SUBSTANCE OR MIXTURE	AFFECTED ORGAN
Creosotes	Skin

Polychlorinated biphenyls	Liver and biliary tract
Tetrachloroethylene	Cervix, oesophagus, non-Hodgkin lymphoma
Trichloroethylene	Liver and biliary tract, non-Hodgkin lymphoma, renal cell

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ANNEXURE E
EFFECTS OF INORGANIC LEAD ON ADULTS

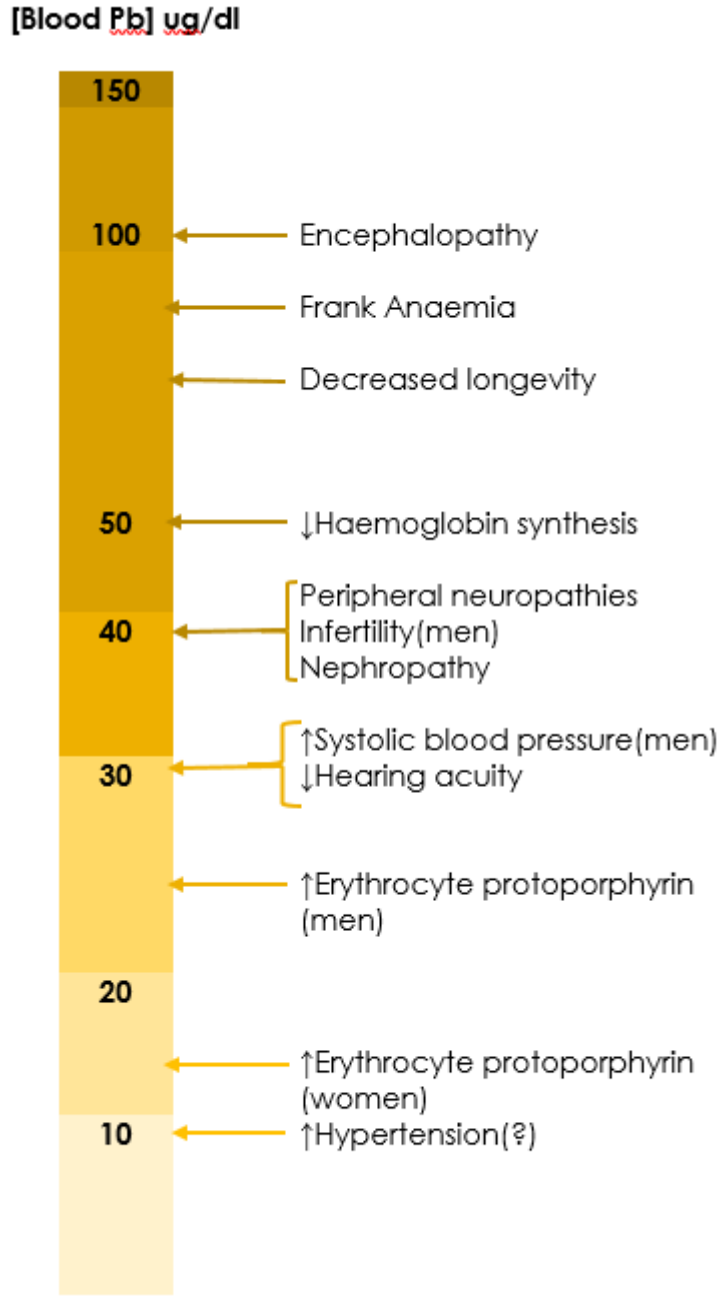


Image sourced from: Nader Rifai and Tietz, N.W. (2019). *Tietz fundamentals of clinical chemistry and molecular diagnostics.* Philadelphia: Saunders.

ANNEXURE F
ALCOHOL AND DRUGS IN THE WORKPLACE

The abuse of alcohol or drugs in the workplace while on duty and the consumption of alcohol or drugs before coming on duty is problem employers are faced with on a regular basis. The employer is faced with balancing the considerations with respect to the individual and the legal framework, with the obligation to safeguard the safety of the other workers and productivity. Alcoholism and drug use in the workplace results in poor job performance, lack of focus, absenteeism, increased health-related problems, use of medical aid funds and fatal accidents. In South Africa, the scale of the problem is not defined. However, there are published cases from the department of Labour and private law firms that provide some insight into the legal handling of individual cases that provide some terms of reference into the appropriate management of alcoholism in the workplace. Interestingly, a US study found that while alcoholism can affect any industry and any organisation, big or small, workplace alcoholism is especially prevalent in the following industries: (1) food service, (2) construction, (3) mining and drilling, (4) excavation, and (5) installation, maintenance and repair. It is important to understand the legal framework that informs the policy an employer will implement in his/her workplace. There are two important acts to consider. They include (A) The Employment Equity Act No. 55 of 1998, and (B) The OHSA No. 85 of 1993 (CO General safety regulations GNR.1031 of 30 May 1986).

A. THE EMPLOYMENT EQUITY ACT

The Employment Equity Act seeks to promote equal opportunity in the workplace and fair treatment in employment through the elimination of unfair discrimination. In particular, Section 7 of the Act relates to medical testing and states that " Medical testing of an employee is prohibited, unless:

- Legislation permits or requires the testing; or

- It is justifiable in the light of medical facts, employment conditions, social policy, the fair distribution of employee benefits or the inherent requirements of a job".

B. OHSAct No. 85 of 1993

The relevant provisions of the OHSA are contained in Sections 8 and 14.
Section 8 deals with the general duties of employers to their employees to ensure a safe and risk-free working environment. Section 14 (1) deals with the general duties of employees at work, and in general requires that an employee "take reasonable care for the health and safety of himself and of other persons who may be affected by his acts or omissions;"

C. GENERAL SAFETY REGULATIONS

Section 2A relates to intoxication as follows:

(1) Subject to the provisions of sub regulation (3), an employer or a user, as the case may be, shall not permit any person who is or who appears to be under the influence of intoxicating liquor or drugs, to enter or remain at a workplace.

(2) Subject to the provisions of sub regulation (3), no person at a workplace shall be under the influence of or have in his or her possession or partake of or offer any other person intoxicating liquor or drugs.

(3) An employer or a user, as the case may be, shall, in the case where a person is taking medicines, only allow such person to perform duties at the workplace if the side effects of such medicine do not constitute a threat to the health or safety of the person concerned or other persons at such workplace…."

Section 2a has several implications for both employers and employees. They include:

(1) It is being recognised that alcohol abuse is a growing problem impacting health and safety in the workplace;

(2) The provision for compulsory drug and alcohol testing to provide a safe working environment;

(3) The frequency of testing depends on the nature of work e.g., drivers of heavy vehicles compared with highly specialised activities such as the medical profession. The issues surrounding the relationship between the legal limit and the consequences of being "under the influence" are contentious and have surfaced in case law and,

(4) The OHSA could therefore offer employers an opportunity and a justification for implementing random testing to ensure that employees are not intoxicated while performing their duties.

D. SUBSTANCE ABUSE PROGRAM

In order to comply with the OHSAct, General safety regulations and Labour laws, it is recommended that employers consider the following when implementing a program in their workplace:
1. Substance abuse policy,
2. Testing,
3. Awareness by training and education and
4. An employee assistance program/EAP.

1. Substance abuse Policy

The labour law guidelines recommend that an employer institute very clear workplace policies. The recommendations to be considered suggest that " The policy should be clear – (1) zero tolerance, allowance for limits, and whether to relate limits with level of functioning to decide on fitness for duty; (2) the policy must stipulate your test procedure e.g. breathalyser test for alcohol; (3) The policy must state that note will be taken of circumstantial evidence, such as bloodshot eyes, slurred speech, the smell of alcohol on the breath, unsteadiness on feet, dishevelled appearance, aggressive or abusive or arrogant or out of character behaviour, and the inability to walk a 10 metre straight line with the arms held out horizontally. In addition to these recommendations, specific questions are required to develop unambiguous policy and procedural statements which impact on labour and employment equity issues such unfair dismissal due to unclear policy or non-compliance with procedures. The following questions require careful thought and include:

- What is the purpose/goal of your policy?
- Who is covered by your policy?
- When does your policy apply?
- What behaviour is prohibited?

- Will employees be required to notify you of alcohol/ drug-related convictions? Does the policy include pre-employment checks or potential incumbents?
- Does your policy include searches?
- Does your program include drug testing?
- Who is covered by the program?
- How to deal with impaired workers.
- How chronic users will be assisted e.g. rehabilitation programs.
- What method of testing is to be used?
- Conditions under which drug testing will be conducted: Pre-employment/Random/Cause testing
- Who is responsible for the testing? What are the limitations of using the result generated?
- What will the consequences be if your policy is violated? Should discipline, counselling, treatment and or rehabilitation be first-line responses?
- Are there Return-to-Work Agreements?
- During treatment should there be paid leave granted, or must the work schedule be adjusted e.g. part-time employment or different duties?
- What type of assistance is available?
- How is employee confidentiality protected? If the employer is made aware, there is a strict ethical and legal obligation to keep the information confidential and in addition, it cannot be disclosed to law enforcement or any other persons without express consent for the involved person. Often, these issues are noted by a medical professional. This person is bound both by ethical and medical confidentiality and can therefore report only on fitness for duty to the employer without revealing the reason in the case of an employee being unfit due to drug or alcohol abuse.
- Who is responsible for enforcing your policy?
- How will your policy be communicated to employees?
- Further considerations as made by the employer.

2. Testing

There are various ways to test for drug or alcohol use, ranging from blood and hair analysis to simple oral fluid, urine and breathalyser tests. Each type of test has its own virtues, mostly relating to the window of detection and ease of use.

Marker tests for alcohol abuse:

Analyte	Cut-off	Sensitivity	Specificity	Use	Amount and time of alcohol use to cause abnormal marker	Time to normalise with abstinence
Ethanol	0.05 g/dl (SA legal limit when driving)			Detecting acute alcohol use. Detect tolerance (>0.15 g/dl without intoxication or >0.3 g/dl at any time)	For blood alcohol >0.05 g/dl after 1h: >2 beers in 70 kg person.	Hours, depending on dose
	Comment: Short detection time limits use					
GGT-CDT index	Male 4.18 Female 3.81	89 96	98 97	Detect heavy drinking	>40 g/d (3 or more beers/d) for more than 1m	2-3weeks
	Comment: GGT-CDT is calculated by a mathematical formula that weighs GGT and % CDT increased sensitivity without affecting specificity. Detects greater alcohol abuse than CDT and GGT alone. Performance is similar whether or not heavy drinkers are contrasted with abstainers or moderate drinkers which is useful for screening. Correlates with the amount of alcohol used. Use of MCV, ALT or AST as a third component did not add value. Liver disease in heavy drinkers did not influence GGT-CDT performance.					
% CDT	2.47	93	97	Most useful to monitor abstinence in alcoholics. Also detects heavy drinking for at least 1 week in alcoholics.	50 - 80 g/d (4-6 beers/d) for at least 1 week in alcoholics.	2-4 weeks
	Comment: % CDT (Carbohydrate deficient transferrin). Normal transferrin has 4 carbohydrate chains. With excessive alcohol use, forms of transferrin that contain no, one or two carbohydrate chains, collectively known as CDT, increase. In alcoholics that relapse, lower use can lead to rapid re-elevation. Most accurate single serum marker for chronic alcohol use and recent heavy drinking readily available. Main strength is specificity. Single episodes of acute alcohol intoxication do not elevate CDT. Decreased sensitivity to detect alcohol abuse in females. False positive results may occur due to non-alcoholic liver disease (primary biliary cirrhosis, chronic active hepatitis, chronic Hepatitis C, hepatocellular carcinoma), carbohydrate deficient glycoprotein syndrome (rare), cystic fibrosis, pregnancy, untreated galactosaemia, rectal carcinoma, senile dementia, depression and solvent abuse. False positive results do not occur with genetic transferrin variants or high transferrin concentrations with the N-Latex INA (immuno-nephelometric assay) currently in use. %CDT methods include immunoassays, capillary electrophoresis and HPLC. Results and cutt-off values from different methods cannot be used interchangeably. Cut-offs are for the N-Latex INA.					

Analyte	Cut-off	Sensitivity	Specificity	Use	Amount and time of alcohol use to cause abnormal marker	Time to normalise with abstinence
GGT (U/L) **(indirect)**	Male 85 Female 65	30 23	94 92	Detect heavy drinking in the general population (1022 males,583 females) (USA)	>70g/day (>5 beers/day) >55 g/day (>4 beers/day) >40g/day (>3 beers/day) in chronic alcoholics	2-5 weeks
	Comment: GGT is a liver enzyme. Most commonly used marker. Increase in absence of other causes should raise suspicion of excessive drinking. Rapid fall with abstinence is highly suggestive that suspicion is correct. Does not increase with binge drinking in non-alcohol abusers. False negative: no longer increased in some chronic drinkers. Rarely increases in individuals <30 years old. False positive results may occur due to a wide range of medication (hormones, anticonvulsants), generalised liver damage, non- alcoholic fatty liver disease, any cause of biliary damage or stasis, hepatic congestion (CCF), pancreatitis, Diabetes Mellitus, obesity, smoking, hyperlipidaemia, hyperthyroidism or severe trauma.					
MCV (Fl) **(indirect)**	96	45	94	Marker of chronic alcoholics with sustained heavy drinking Detect heavy drinking among: heavy drinkers (n=165) moderate drinkers (n=51) abstainers (n=35) (Finland)	>60 g/day (>4 beers/day) For at least a month	2-4 months
	Comment: Mean corpuscular volume (MCV) is the size of the red blood cells. Good specificity (very few tee-totallers and social drinkers will have increased MCV). Easily obtained. Use encouraged when considering chronic alcohol abuse and dependence. Poor screening marker of acute ethanol intake. Takes several months to reflect changes in drinking. May continue to rise after use stopped in alcohol dependence. Cannot monitor abstinence or relapse. False positive results may occur due to Vitamin B12 or foliate deficiency, hypothyroidism, haemolytic disease, non-alcoholic liver disease, age, smoking or medication (anticonvulsant					
AST/ALT **(indirect)**	>2	Low	90	Detects alcohol-induced liver damage	-	-

Analyte	Cut-off	Sensitivity	Specificity	Use	Amount and time of alcohol use to cause abnormal marker	Time to normalise with abstinence
				Distinguish alcohol induced from non-alcohol induced liver disease		
	Comment: Indicates advanced alcoholic liver disease rather than heavy alcohol consumption					

Drugs of abuse tests

Urine drug screening is usually the first step of drug testing and a positive outcome should be confirmed. Confirmatory tests should be conducted with the *Chain of Custody* procedure in order to ensure that results can be used in a court of law. Ampath Pathologists will testify in a court of law if the following conditions were adhered to:

- Consent given by worker,
- Worker fully informed as to the meaning and impact of drugs of abuse testing,
- Worker informed he/she cannot be compelled to submit him or herself to a drug of abuse test,
- No negative inference may be drawn from an employee's failure to submit him or herself to a drug of abuse test,
- Worker should give consent for disclosure of results to employer,
- Instruction for drug testing by Medical Practitioner acting on behalf of employer and
- Chain of Custody was followed.

Ampath offers the following Drugs of abuse screening (Cassette or Immunoassay) & Confirmatory tests on urine:

Drug of abuse	Screening (Cassette)	Screening (Immunoassay)	Confirmatory	Street name(s)	Detection time
Drugs of Abuse screen	POCDOA *(10 panel)*	DOA *(# below Immunoassay + Confirmatory incl., 10 panel)*	***NB: arrange chain of custody if needed***		
Amphetamine	POCDOA	AMPH#	ATSC	Speed, Crystal, Ice, Uppers	1-4 days
Barbiturates		BARBU	MISCC	Blue heavens, Velvet, Devil, Red devils, Pink lady, Purple hearts	1-14 days
Benzodiazepines		BENZU#	BENZOUC	Benzos, Mellow, Downers, Ativan,	1-9 days, up to 30 days

Drug of abuse	Screening (Cassette)	Screening (Immunoassay)	Confirmatory	Street name(s)	Detection time
				Rohypnol, Valium, Serepax	
Cannabis		CANN#	CANC	Dagga; Marijuana; Pot; Weed, Whoonga (Nyaope)	2-5 days (infrequent use), 3-4 weeks (chronic use), 6-11 weeks (heavy use)
Cocaine (Benzoylecgonine)		COCA#	COCC	Crack, Coke, Rock, Snow, Flake, Blow	2-3 days, up to 9 days
Methadone		METH#	MISCC	Meth, Methadone	1-3 days
Methamphetamine (Tik)		-	ATSC	Tik-Tik	1-4 days
Opiates		OPIA#	OPIAC 6-Acetyl morphine (Heroin),Codeine Morphine	Morphine: Junk, White Stuff, "M", Heroin: Horse, White Lady, "H"	7-54 hours (infrequent use) Up to 12 days (chronic use)
PCP (Phencyclidine)		-	PCPC	PCP, Angel dust	1 day to 2 weeks, up to 1 month
Tricyclic antidepressants		-			
Ecstasy (MDMA, MDA, MDEA)			ATSC#	"X"	At least 24 hours
LSD (Lysergic Acid Diethylamide)	-	-	LSDC	Acid	0-2 days
Methcathinone (CAT)	-	-	ATSC#	CAT	1-4 days
Mandrax (Methaqualone)	-	MAND#	MANDC	Mandrax; Soaps, Love Pill	90-225 hours
PPX (Propoxyphene)	POCPPXS	-	PPXC	PPX, Doloxene	1-2 days

Chain of Custody

Definition:
A record of the sequences of individuals who had custody of a sample, to ensure the integrity of the sample. It refers to a trail showing the collection, transport, analysis and result of a sample.

Personnel requirements:
A competent staff member, hereafter referred to as "the collection officer", must have received training on the chain of custody procedure and must supervise the site and perform the collection.

Identification and documentation:

Identify the donor with photo identification (ID book/ID card or driver's licence). The Collection officer and the donor must complete the section: **DONOR IDENTIFICATION by photo** on the Chain of custody request form.

Explain the following to the donor:

- Specimen/s collection procedure,
- Chain of custody process,
- Test/s are confirmatory, using different methodology than screening tests,
- Confirmatory test results can stand in a court of law,
- To whom the results will be reported, e.g., clinician or designated representative of the employer (Occupational Health practitioner, human resources officer etc.) and
- Results will be acted upon according to the employer's substance abuse policy.

The donor must provide voluntary informed consent. Make use of the **Donor's Statement of Voluntary Informed Consent** form provided. Document use of any illicit, prescription or over-the-counter substances by the donor during the previous two weeks on the Ampath generic request form.

Collection of blood specimen for Chain of Custody Blood Ethanol (COCEBC):

- Label two grey top (Sodium Fluoride Potassium Oxalate) tubes with donor name, surname, date and time of collection.
- Clean the skin with sterile water (do not use alcohol containing swabs e.g. Webcol).
- Collect blood into the two labelled tubes as per standard procedure.
- The donor must sign on the tubes as confirmation of identification.
- Proceed to section 5: Documentation and Specimen Preparation for Transport.

Collection of Urine Specimen:

Secure Site:

- Urine collection must take place in a standard toilet cubicle to minimise opportunity for tampering/adulteration with specimen (e.g. no taps as in toilet for disabled individuals).
- Inspect the cubicle to ensure it is free of any potential interfering substances (remove dustbin, cleaning products and all other containers, furniture etc.).
- If possible, use a cubicle without a window; if not possible, the window must be shut to prevent that a substituted urine specimen be handed to the individual.

Preparation of urine specimen donor:

- The donor may not take any belongings into the cubicle (e.g. handbag).
- Remove all unnecessary outer garments (e.g. jackets).
- The donor must empty all his/her pockets (a physical body search is not required).
- The donor must wash and dry his/her hands before the sample is donated.
- Keep the donor under direct supervision up until collection, to prevent access to adulterants.
- Instruct the donor to immediately void the urine specimen and immediately hand the container to the collection officer, without taking any other actions like flushing the toilet, opening the window, etc.

Urine specimen collection:

- Label the container with donor name, surname, date and time of collection.
- If available, place barcode labels onto the urine specimen and all supporting forms as well as chain of custody envelope.

- The donor must enter the secured cubicle and close the door, to give the donor visual privacy.
- The collection officer must remain directly outside the closed door to listen for any suspicious sounds (e.g. opening of window or toilet tank, flushing, etc.).
- The donor must hand the freshly collected urine specimen to the collection officer as soon as possible and within a reasonable time period for voiding of urine.
- If the donor claims that he/she is unable to pass urine, the collection officer must request the donor to try to void a urine specimen.
- If the donor still cannot void urine, the donor must drink 250ml (a standard glass) of water, and again after 30 minutes (2 glasses over 1 hour), the donor should be able to void 20 ml urine within 3 hours.
- If the donor still cannot void urine, the collection procedure is terminated.
- On receipt of the urine specimen, the collection officer must immediately ensure that the urine specimen is still warm (at body temperature) and document any obvious unusual findings (e.g. colour, soapy appearance etc) on the generic request form.
- If the container has a tamper proof seal, the **donor** must sign/initial on the seal. If the container does not have a tamper proof seal, the donor must sign/initial on the urine container.
- The collection officer must seal the urine container in an envelope in the presence of the donor. Refer to section 5: Documentation and Specimen preparation for Transport.

Termination of urine collection procedure:
If attempted tampering/adulteration is detected, or if the donor fails/refuses to cooperate with any requirement above, the collection procedure is terminated and the collection officer must:

- Complete the Chain of Custody request form in full and note the reason for termination of collection on the form.
- Inform the referring client immediately and document the name of the person spoken to, date and time on Chain of Custody request form.

Documentation and Specimen preparation for Transport:
Complete the following forms:

1. **Ampath generic request form**
   - The donor must sign this form
   - Write the requested test(s) to be performed, on the form
   - Note if the specimen was warm to the touch upon receipt and document any obvious unusual findings (e.g., colour, soapy appearance etc.). Refer to section 4.3 – Urine specimen collection.
   - Document use of any illicit, prescription or over-the-counter substances by the donor during the previous two weeks on the Ampath generic request form. Refer to section 2.

2. **Chain of Custody – Informed consent form**

   The donor and collection officer must complete and sign this form. If this form is not completed and sent in, testing will not commence.

3. **Chain of Custody request form**

- The donor and collection officer must sign this form in all the designated fields.
- Tick the requested mnemonic:
  COCD for Chain of Custody Drugs of abuse on Urine.
  COCEBC for Chain of Custody Blood Ethanol
- **DO NOT add any other mnemonics/tests as this will influence the Chain of Custody trail.**
- Label envelope as follows:

  Employee/patient Surname, Name
  Ampath
  CHAIN OF CUSTODY specimen
  Esoteric Science
  166 Witch Hazel Avenue
  Techno Park
  Centurion

- Ensure that the specimen container lid is closed properly.
- Place the specimen in an Ampath specimen plastic bag.
- Place Ampath Generic request form and Informed Consent Form in the document sleeve of the Ampath specimen bag.
- Place the specimen bag in the envelope in front of the patient/employee.
- Seal envelope.
- Donor and collection officer must sign across the envelope's seal **(Should the envelope have more than one sealed flap, sign across all)**.
- Donor and collection officer must complete section "Specimen sealed in Envelope" on Chain of Custody form.
- Attach Chain of Custody form to envelope.
- Place the envelope in a plastic bag to prevent water damage to envelope.
- Messenger collecting the specimen must complete section: " Courier information - Courier 1"

ANNEXURE G
METAL ALLERGIES IN THE WORKPLACE

Metals have been implicated in causing sensitisation and allergic diseases. These patients may present with skin reactions/dermatitis, eye infections, respiratory symptoms, nasal symptoms, loosening of orthopaedic and dental prosthesis. Other symptoms may also occur. This hypersensitivity reaction caused by metals is classified using the Gel and Coombs classification system for allergic reactions; as a Type IV hypersensitivity reaction. Type IV hypersensitivity is often called delayed type hypersensitivity as the reaction takes two to three days to develop and is not antibody mediated but is a cell-mediated response.

It is important to differentiate between metal allergy and toxicity. Metal toxicity is the toxic effect of certain metals in the body. Different metals will impact on different bodily functions or organs, refer to Target organs for metal exposure. The level of metal exposure and possible toxicity is obtained when the specific metal's level is determined in blood or urine samples and compared to a normal reference range and the biological exposure index (BEI).

The lymphocyte proliferation test (LPT) test however does not, however, measure the levels of metals in the body, but measures whether the patient is allergic to metals by measuring the Type IV delayed hypersensitivity reaction. Blood samples may

show levels of metals below the official biological exposure index or with-in the normal reference range – but the patient may still be allergic. For allergic individuals, there is no such thing as a "safe" limit. Even trace amounts of a metal may cause or worsen health problems if the metal triggers an immune reaction.

A Type-IV allergic reaction is mediated by T-lymphocytes (or memory lymphocytes) that have had prior contact with the given allergen. The LPT test procedure involves the isolation of white blood cells (lymphocytes) from whole blood and then tests against allergens chosen according to the patient's occupational history. The blood is then incubated for five days and the lymphocyte reaction is measured thereafter. The level of reactivity is measured as a Stimulation Index (SI). A value over 3 indicates a positive reaction to a given allergen.

Metal Allergy testing

Patient preparation:
The patient must not have taken cortisone two weeks prior to testing.
Patients on long term cortisone must first receive approval from their doctor to stop their medication before testing can be done.
Sample type: 4-6 Citrate tubes

Collection instructions:
This test is not performed over weekends and must be performed within 24 hours after collection; therefore blood samples can only be drawn Sundays to Thursdays. Blood must reach the lab before 24hours after collection. Do not centrifuge the tubes. Send at room temperature.

The Metal and Metal Alloy Test List:

Metal	Mnemonic
Aluminium	ALW
Arsenic	ARW
Barium	BAW
Beryllium	BEW
Cadmium	CDW
Chromium	CRW
Cobalt	COWS
Copper	CUW
Ethyl Mercury	HGEW
Galium	GAW
Gold	AUW
Indium	INW
Inorg Mercury	HGIW
Iridium	IRW
Iron	FEW
Lanthanum	LW
Lead	PBW
Manganese	MNW
Methyl Mercury	HGMW
Molybdenum	MOW

Nickel	NIW
Palladium	PDW
Phenyl Mercury	HGPW
Platinum	PTW
Ruthenium	RUTW
Silver	AGW
Thimerosal	THIMW
Tin	SNW
Titanium	TIW
Vanadium	VW
Zinc	ZNW
Zirconium	ZRW

ANNEXURE H

AMPATH QUALITY ASSURANCE

Quality assurance is a program for the systematic monitoring and evaluation of the various aspects of a laboratory to ensure that standards of quality are being met. Ampath's quality assurance system is based on the ISO 15189 guidelines for medical laboratories. This guideline specifies requirements for quality and competence in medical laboratories. Ampath is accredited at the South African National Accreditation System (SANAS). A list of Ampath accredited laboratories is available on the SANAS website (https://www.sanas.co.za/). SANAS is recognised by the South African Government as the single National Accreditation Body to confirm that Laboratories, Certification Bodies, Inspection Bodies, Proficiency Testing Scheme Providers and Good Laboratory Practice (GLP) test facilities are competent to carry out specific tasks in terms of the Accreditation for Conformity Assessment, Calibration and Good Laboratory Practice Act (Act 19 of 2006). All requirements as stipulated by the ISO 15189 standard are followed for all testing performed within Ampath. Laboratory testing is subject to many influences that can affect the integrity of the result reported. These are:

- **Pre-Analytical:** Activities that happen prior to analysis.
- **Analytical:** Actual specimen analysis.
- **Post-Analytical**: Activities after analysis.

Pre-Analytical

From the instant a sample is collected, a chain of events is set into motion. All of these must be done in a correct manner to ensure reliable results:

- Collection: The correct specimen must be collected from the specific patient.
- Test request: The correct test must be requested and marked on the Request form.
- Patient & Test ordering: Patients with corresponding tests required must be ordered on the Laboratory Information System.
- Sample transportation: Samples must be transported in a way to ensure sample integrity (e.g. cold storage and transportation).

Analytical

Once a specimen is received in the laboratory, quality assurance procedures guide and monitor all related activities. This ensures precision and accuracy of results.

Staff

All staff are deemed competent prior to performing any analysis. The training comprises theoretical as well as practical training. Theoretical training is achieved through lectures that the trainer facilitates either in a classroom situation as well as explanations during the on-the-job practical training. Practical training is facilitated through one-on-one training with the trainer. The method of training is facilitated through the following process:

- Presentation and demonstration by the trainer,
- Intern performing under supervision ,
- Feedback to intern and
- Follow up by facilitator and assessment of intern's competence.

Method validation
All tests used are validated prior to use to ensure that the test is fit for the intended use.

Instrumentation
Instrument operations: Relevant instrumentation is serviced and calibrated regularly to ensure quality results.

Quality control (QC)
A commercial Quality control (QC) sample, with known target value and range, is run to verify that the test is working properly. Quality control is a measure of precision or how well the measurement system reproduced the same result over time and under varying operation conditions. Precision is the indication of the repeatability and reproducibility of the results. For a batch to be valid, the QC result must be within a 2 SD from the mean target value of the control. If a QC is outside the 2SD range, the batch is re-run after investigation as to the reason for the QC failure. The West Guard rules are used to interpret QC performance. Patient samples are tested in batches. The QC is run at the beginning and end of each batch.

Proficiency testing
External Quality control samples are tested monthly to measure the laboratory's accuracy. Blood samples with unknown values are received and tested, known as "blind testing". Results are submitted to the proficiency scheme and our results are compared to our method peers.

Post Analytical

Once results are generated, post analytical procedures ensure the timeous resulting of these results:

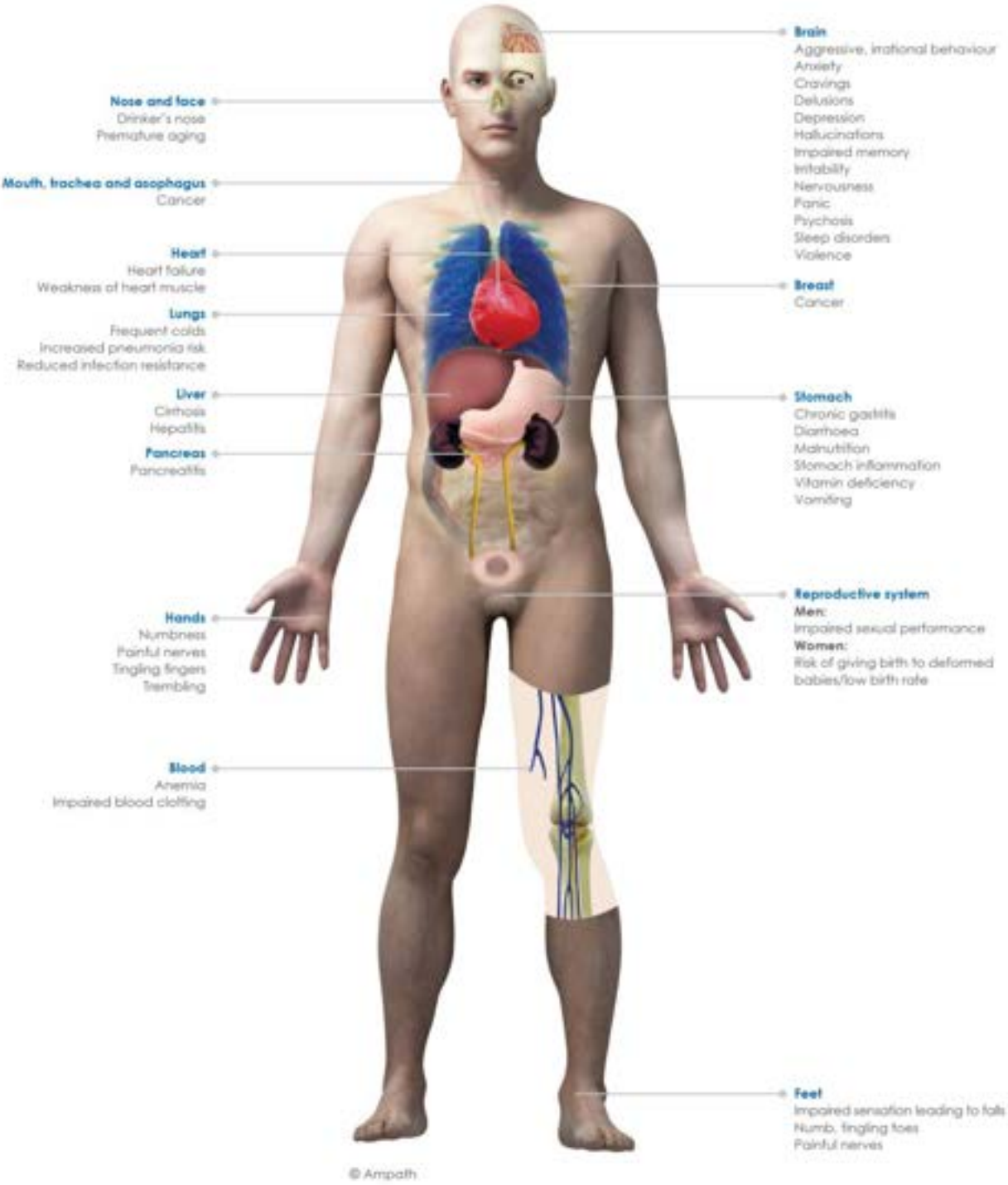
- Reports: The laboratory has to ensure that the confidential results are sent to the provider.
- Turnaround time (TAT): Each test has an expected TAT for test results. If these cannot be met-the client has to be informed.
- Reference intervals: Are included, where available, in laboratory results.
- Interpretation and commenting by Pathologists prior to release of reports: All results are verified by a Pathologist and comments added, where applicable.

ANNEXURE I
WORK-RELATED HAZARDOUS CHEMICAL AND METAL EXPOSURE

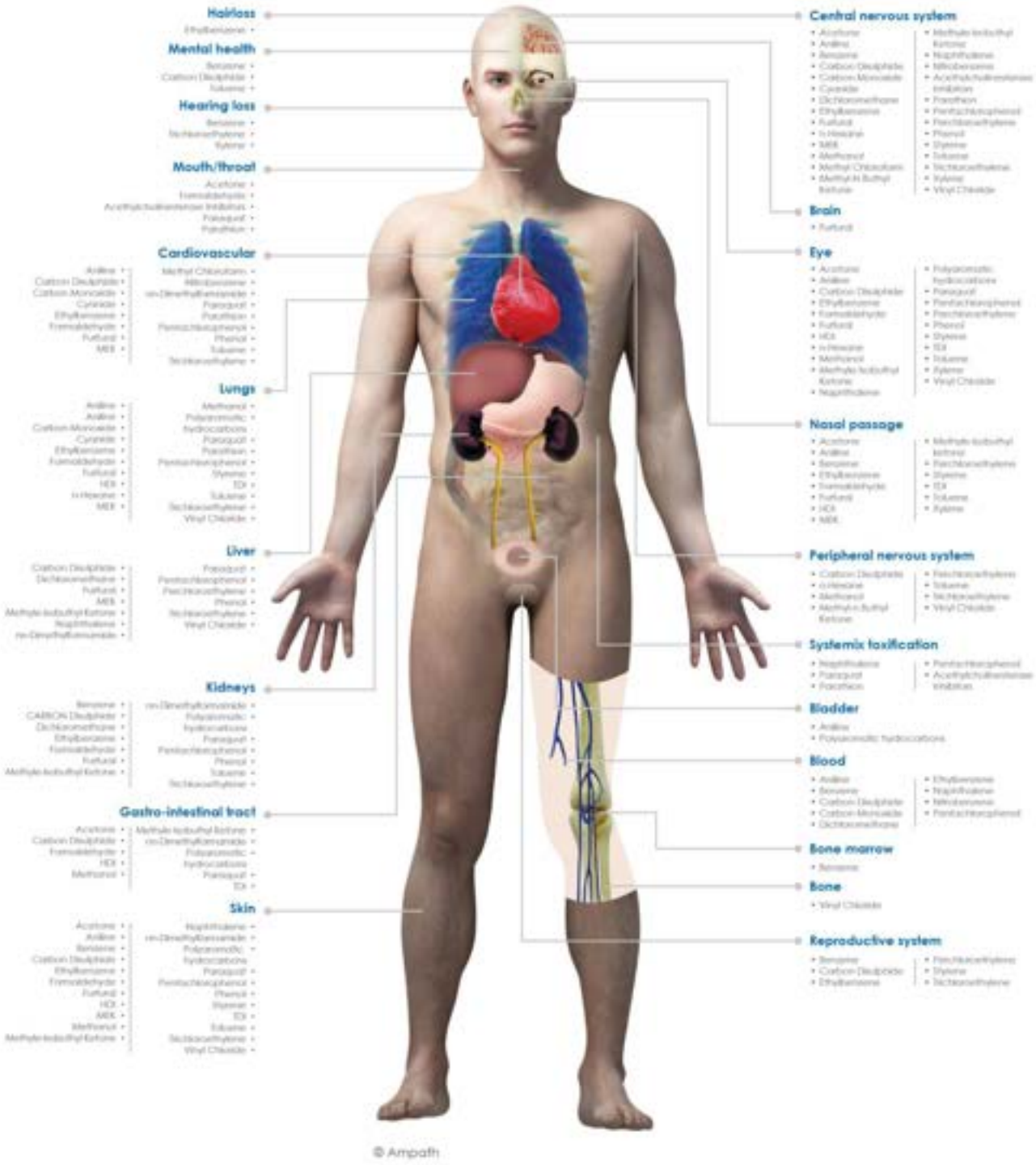
Occupations and occupational groups	Hazardous chemical and metal exposure
Aircraft and aerospace industries	Beryllium (alloys); polycyclic aromatic hydrocarbon (PAH), aluminium, cadmium (welding and spray painting), chromium (welding and spray painting), manganese, n-hexane, resins (amines, phenol, styrene), isocyanates; cobalt; nickel (welding); mercury (laboratories and engineering); phosgene (welding); methyl ethyl ketone
Cement industry, including laying	Asbestos, hexavalent chromium, thallium
Founding	Silica, asbestos (furnace), lead, zinc, chromium, nickel, manganese, beryllium; copper; cobalt (brass); cadmium; vanadium, cyanide, tin sulphur compounds, PAHs (benzo-a-pyrene, cresol, naphthalene in coke oven workers), coal pitch tar, benzene, toluene, xylene, ammonia, aldehydes (formaldehyde, furfural); fluoride
Rubber and tyre manufacturing industry	Acrylonitrile, benzene, creosote, acetaldehyde; styrene; solvents - toluene, xylene
Paint industry - manufacture and painting/paint stripping/spray painting	Lead, mercury, thallium; chromium, cadmium, solvents (petroleum, toluene, xylene, ketones); chlorinated hydrocarbons; aromatic hydrocarbons
Oil and natural gas production	Volatile organic compounds; benzene; xylene; toluene; ethylbenzene; n-hexane
Automotive industry/drivers	Asbestos; cadmium; hexavalent chromium; solvents; isocyanates; aliphatic amines; n-hexane; PAH; metals - welding
Carpentry and woodworks, furniture manufacture, timber preservation	Arsenic (wood preservatives), chromium, creosote, isocyanates, pentachlorophenol (PCP); toluene; xylene; MEK; trichloroethylene; coal pitch tar (roofers)
Glass/pottery/ceramic/related production	Arsenic, beryllium (high-tech ceramics); Thallium, arsenic (art glass workers); lead, cadmium, chromium, arsenic, copper, nickel, cobalt, manganese or tin, styrene; formaldehyde, solvents (includes chlorinated and hydrocarbon)
Dry cleaning	Organic solvents – perchloroethylene
Electroplating/plating/ polishing/anodising/ colouring	Chromium; Cadmium, nickel; diisocyanates; epoxy or polyurethane paint
Leather/fur/footwear industry	Arsenic, chromium (tanning, fur dyeing); organic solvents (benzene, formaldehyde); pentachlorophenol; toluene
Electrical appliances and equipment	Lead, antimony, arsenic in lead-acid battery manufacture; nickel; cadmium; electric cable manufacture- aluminium, cadmium; beryllium; mercury (electrical meters)
Petroleum refining/petrochemical manufacturing	PAH; aliphatic hydrocarbons - ethylene; aromatic hydrocarbons - benzene, toluene, xylene, styrene, ethyl benzene
Plastic industry	Acrylonitrile, benzene, cadmium (pigment); diisocyanates; phenol; styrene; formaldehyde; phthalates; toluene; xylene
Welding	Cadmium (radiator welding, use of cadmium-based solders) chromium (stainless steel, mild steel), isocyanides, lead, aluminium (electric welding), manganese, fluorides; beryllium; cobalt (stellite welding)
Printing processes including inks and toners	Chromium, n-hexane, epoxy resins, formaldehyde, isocyanate, cadmium pigments, acrylic resins

Occupations and occupational groups	Hazardous chemical and metal exposure
Agricultural (includes plantations; seasonal and migrant workers; greenhouse and nursery; horticulture, floriculture; mushrooms; crops for beverage industry; tobacco; vegetables; grape vineyards)	Pesticides i.e. organophosphate, carbamates, and organochlorine; pentachlorophenol; herbicides, fumigants i.e. carbon disulphide; fungicides; insecticides, benzene, solvents; sulphides
Textile industry	Cadmium, chromium; epoxy resins; isocyanate; formaldehyde; carbon disulphide; amines
Manufacture of insecticides, fungicides, weed killer, animal dips, fertiliser/pest control/ veterinary work	Arsenic, creosote, mercury (fungicides), organophosphates, acetaldehyde, carbon disulphide,nitrogen compounds - ammonia; pyrethroids; urea; captofil
Smelting (includes non-ferrous)	Arsenic; copper, zinc, lead;, cadmium; antimony; chromium; cobalt; manganese; mercury (gold); beryllium; fluoride (aluminium smelting); creosote aluminium - pot room workers, thallium
Production of pigments/dyes	Arsenic, cadmium, chromium, mercury, thallium; lead; toluene
Electricians, electrical component, and electronics manufacture	Asbestos (computer cabling), mercury {electrical meters}, beryllium and arsenic (electronics), lead,
Repair of home appliances	Asbestos, chromium; cadmium; beryllium; aluminium; polyurethane; isocyanates; epoxy resins; formaldehyde; ethanolamine
Mining	Asbestos, silica, lead, mercury, vanadium, chromium, cobalt, manganese, thallium; arsenic; benzene
Fire-fighters	PVC; polychlorinated biphenyls
Battery construction and disposal, semiconductors, solar batteries, diodes	Cadmium (alkali and nickel-cadmium) , nickel; lead, arsenic (lead storage battery factory); antimony (starter battery production); plastics - polyethylene, PVC; polyamides
Construction industry/ demolition of buildings/site excavations/road building	Silica, asbestos; chromium; nickel; lead; epoxy and acrylic resins; isocyanates; PAHs (asphalt)
Emergency and security services	Benzene and other petroleum related products; carbon monoxide; hydrogen cyanide; formaldehyde; PVC; polyurethane
Health facilities and services	Mercury (dental work, laboratory), disinfectants; pharmaceuticals; sterilisers; pesticides; anaesthetic gases; laboratory reagents
Transport and warehousing	PAHs; formaldehyde; asbestos; lead; isocyanates; cadmium; chromium; manganese; cobalt; beryllium; refrigerants; benzene-containing petrochemicals; fumigants; pesticides; carbon tetrachloride
Hotel and restaurant	Disinfectants, pesticides
Education and training	Organic solvents, pigments, dyes, metals, plastics, minerals - arts & crafts; formaldehyde - chemistry & biology; asbestos, lead, pesticides

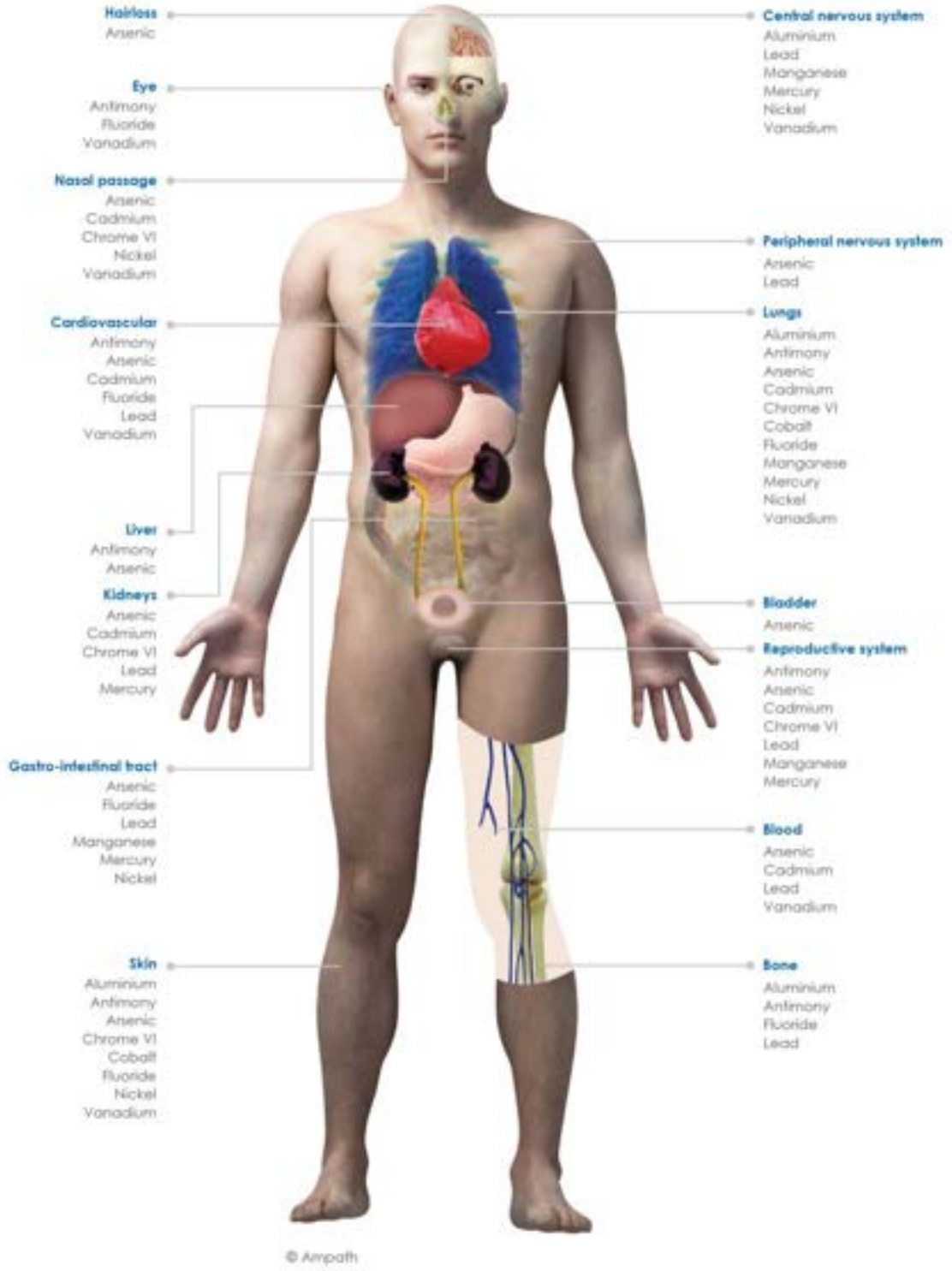
ANNEXURE J
TARGET ORGANS: ALCOHOL ABUSE



TARGET ORGANS: CHEMICAL EXPOSURE



TARGET ORGANS: METAL EXPOSURE



ANNEXURE K
ICOH Code of Ethics

The pages that follow reflect the official ICOH code of ethics.

INTERNATIONAL CODE OF ETHICS

FOR OCCUPATIONAL HEALTH PROFESSIONALS

THIRD EDITION

First edition: 1992
Second printing: 1994
Third printing: 1996
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Third edition: 2014

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PREFACE

1. There are several reasons why the International Commission on Occupational Health (ICOH) has committed itself in the development of an International Code of Ethics for Occupational Health Professionals, as distinct from codes of ethics for all medical practitioners. The first one is the increased recognition of the complex and sometimes competing responsibilities of occupational health and safety professionals towards the workers, the employers, the public, public health and labour authorities and other bodies such as social security and judicial authorities. The second one is the increasing number of occupational health and safety professionals as resulting from the compulsory or voluntary establishment of occupational health services. Yet another factor is the emerging development of a multidisciplinary approach in occupational health which is implying an involvement in occupational health services of specialists who belong to various professions.

2. The International Code of Ethics for Occupational Health Professionals is relevant to many professional groups carrying out tasks and having responsibilities in enterprises as well as in the private and public sectors concerning safety, hygiene, health and the environment in relation to work. The term occupational health professionals category is for the purpose of the Code defined as a broad target group whose common vocation is a professional commitment in pursuing an occupational health agenda. The scope of this Code covers activities of occupational health professionals both when they are acting in individual capacity and as part of organizations or undertakings providing services to clients and customers. The Code applies to occupational health professionals and occupational health services regardless of whether they operate in a free market context subject to competition or within the framework of public sector health services.

3. The 1992 International Code of Ethics first edition laid down general principles of ethics in occupational health. These are still valid but need to be updated and rephrased to reinforce their relevance in the changing environment where occupational health is practiced. Changes in working conditions and in social demand should be taken

into account including those brought about by political and social developments in societies; demands on utility value, continued quality improvements and transparency; globalization of the world economy and liberalization of international trade; technical development and introduction of information technology as an integral element of production and services. All these aspects have repercussions on the context surrounding the occupational health practice and thereby influence the professional norms of conduct and the ethics of occupational health professionals.

4. The preparation of an International Code of Ethics for Occupational Health Professionals dates back to 1987 when it was discussed by the Board of the ICOH in Sydney. The prepared draft was subject to a process of consultations. The 1992 Code of Ethics for Occupational Health Professionals was approved by the ICOH Board in November 1991 and published in English and French in 1992, with many reprints and translation into other languages in the following years. A Working Group was established in 1993 with the aim of updating the International Code of Ethics for Occupational Health Professionals and agreed with the ICOH Board in 1997 that an in-depth revision of the Code of Ethics was necessary aiming at supplementing the Code with new issues and themes needing to be addressed. The reconstituted Working Group on Ethics in Occupational Health (J.F. Caillard, G.H. Coppée and P. Westerholm) started the revision process of the Code in 1999 in consultation with selected ICOH members retaining its original structure and reorganizing the text in a more systematic manner.

5. The updated version of the Code of Ethics for Occupational Health Professionals was approved by the ICOH Board in March 2002. The 2002 Code was widely recognized and used for the elaboration of national codes of ethics and for educational purposes. It was adopted as terms of reference in Argentina and Italy in the Framework Act for occupational safety and health. It was also included into the Rosenstock and Cullen text edition. Apart from the ICOH official languages, the ICOH Code of Ethics was translated into Chinese, Greek, Italian, Japanese, Portuguese, Spanish and Turkish. In 2010, the United Nations Medical Directors Working Group agreed to advise that any

UN organizational statements of ethics in occupational health matters should be guided by, and consistent with the ICOH Code of Ethics. Furthermore there have been many adoptions on a voluntary basis as a standard for defining and evaluating professional conduct and it was widely referred to in occupational health and related fields.

6. The ICOH Board decided in 2008 to review the 2002 Code, and commissioned the Working Committee on Ethics and Transparency of the Board to perform the task at a Board meeting in Cape Town in March 2009. The committee consisted of Board Members P. Westerholm (chair), G. Costa, M. Guillemin, J. Harrison and J. Howard Jr. acting as the ICOH Board Code Review Group. Board member M. Fingerhut was affiliated to the Group to strengthen capacity for liaison in view of the global scope of the task. For strengthening field contacts, the Code Review Group was expanded by affiliating J.F. Caillard (ICOH Past President) and S. Iavicoli (ICOH Secretary-General). For expanding contacts with world regions and professional networks in Latin and South Americas, Africa and Asia, J. Rodriguez-Guzman, L. London and S. Horie were commissioned and affiliated to the Code Review Group. In addition, a task group was constituted as a subgroup of the Code Review Group to address Ethical Code issues related to the cultural context on the African continent, with G. B. Tangwa, R. B. Matchaba-Hove, A. Nyika, N. MKhize and R. N. Nwabueze. The review work was carried out by drafting a series of text reviews. The Code Review Group members used opportunities at ICOH conferences in Europe, Africa, South and Latin Americas and Asia to discuss Code review matters with the members they met and with other professional networks.

7. A progress report of the Code Review Group was presented and discussed at the ICOH Board Midterm Meeting in Milan in February 2011. The ICOH Membership became involved through communication of the preliminary review results to ICOH scientific Committees Chairs and Secretaries and ICOH National Secretaries. The Code Review Group´s report submitted by P. Westerholm, Chair of the Ethics and Transparency Committee, in September 2011 was discussed at a Code review meeting organized by ICOH President K. Kogi and attended by ICOH Officers and the members of the Ethics and Trans-

parency Committee. It was agreed to minimize the changes in the Code by examining the issues requiring revisions. A special session at the ICOH Congress held in Cancun in March 2012 further discussed these issues based on the review results. The changes required for the Code were discussed at a workshop on the ICOH Code of Ethics organized at the University of Occupational and Environmental Health in Kitakyushu in August 2012. To finalize the Code revision work in view of the suggested amendments generated throughout the review process, ICOH President K. Kogi organized a Code Editing Group comprising ICOH Vice-Presidents S. Lehtinen and B. Rogers, Secretary-General S. Iavicoli, Past President J. Rantanen and Board members G. Costa, N. Kawakami, C. Nogueira, E. Santino and P. Westerholm. In the meeting of the Code Editing Group held in June 2013, the draft revisions of the Code were finalized. In addition, we greatly acknowledge the editorial support for this third edition of Mr. Carlo Petyx (Coordinator), Ms. Valeria Boccuni, Ms. Erika Cannone, Mr. Pierluca Dionisi and Ms. Antonella Oliverio.

8. In the review process, the fundamental point of departure and aim have been to retain the already existing structure of the 2002 Code throughout the review, for the purpose of serving continuity and recognition of its contents by the successive generations of ICOH members and all readers within the occupational health professional community of the world. The review process has resulted in working materials and documentation envisaged to be made available as supplementary entries on the ICOH website following the adoption of the revised Code by the ICOH Board. The proposed Code revisions were presented to the ICOH Board Midterm Meeting held in Helsinki in February 2014. By further revising the proposed changes, the ICOH Board adopted the new International Code of Ethics for Occupational health Professionals on 10 February 2014.

9. This Code of Ethics represents an attempt to translate in terms of professional conduct the values and ethical principles in occupational health. It is intended to guide all those who carry out occupational health activities and to set a reference level on the basis of which their performance can be assessed. Its purpose is also to contribute to the development of a common set of principles for cooperation between

all those concerned as well as to promote teamwork and a multidisciplinary approach in occupational health. It provides a framework against which to document and justify departures from accepted practice and places a burden of responsibility on those who do not make their reasons explicit. It should also be noted that more detailed guidance on a number of particular aspects can be found in national codes of ethics or guidelines for specific professions. Furthermore, the Code of Ethics does not aim to cover all areas of implementation or all aspects of the conduct of occupational health professionals or their relationships with social partners, other professionals and the public. It is acknowledged that some aspects of professional ethics may be specific to certain professions and need additional ethical guidance.

10. It should be stressed that ethics should be considered as a subject that has no clear end boundaries and requires interactions, multidisciplinary co-operation, consultations and participation. The process may turn out to be more important than its ultimate outcome. A code of ethics for occupational health professionals should never be considered as «final» but as a milestone of a dynamic process involving the occupational health community as a whole, the ICOH and other organizations concerned with safety, health and the environment, including employers' and workers' organizations.

11. It cannot be overemphasized that ethics in occupational health is by essence a field of interactions between many partners. Good occupational health is inclusive, not exclusive. The elaboration and the implementation of professional conduct standards do not involve only the occupational health professionals themselves but also those who will benefit from or may feel threatened by their practice as well as those who will support its sound implementation or denounce its shortcomings. This document should therefore be kept under review and its revision should be undertaken when deemed necessary. Comments to improve its content should be addressed to the Secretary-General of the International Commission on Occupational Health.

12. The Code of Ethics of the International Commission on Occupational Health may be freely reproduced. Proposals of translation into other languages, other than English and French, must be addressed

jointly to ICOH President and Secretary-General. Translation into other languages, other than the official ones, must be done by an ad hoc Working Group appointed by ICOH President. ICOH President could nominate a Peer Reviewing Group to revise the translated version, if necessary. The Chair of the Working Group will submit the final revised translated text for approval by ICOH President. Translated versions of ICOH Code of Ethics must include a copy of the Code either in English or French. Printing of the ICOH Code of Ethics is subject to prior authorization by ICOH President. Any financial support for printing by any kind of organizations has to be preliminarily communicated and approved by ICOH President.

Kazutaka Kogi, MD, DMSc
ICOH President

Sergio Iavicoli, MD, PhD
ICOH Secretary-General

INTRODUCTION

1. The aim of occupational health practice is to protect and promote workers' health, to sustain and improve their working capacity and ability, to contribute to the establishment and maintenance of a safe and healthy working environment for all, as well as to promote the adaptation of work to the capabilities of workers, taking into account their state of health.

2. The field of occupational health is broad and covers the prevention of all impairments arising out of employment, work injuries and work-related disorders, including occupational diseases, the protection and promotion of workers' health and all aspects relating to the interactions between work and health. Occupational health professionals should be involved, whenever possible, in the design and choice of health and safety equipment, appropriate work methods and procedures and safe work practices relevant to health, safety and work ability of workers. They should encourage workers' participation in this field as well as feedback from experience.

3. On the basis of the principle of equity, occupational health professionals should assist workers in obtaining and maintaining employment notwithstanding their health deficiencies or their handicap. It should be duly recognized that there are particular occupational health needs of workers as determined by factors such as gender, age, ethnicity, physiological condition, social aspects, communication barriers or other factors. Such needs should be met on an individual basis with due concern to protection of health in relation to work and without leaving any possibility for discrimination.

4. For the purpose of this Code, the expression «occupational health professionals» is meant to include all those who, in a professional capacity, carry out occupational safety and health tasks, provide occupational health services or are involved in an occupational health practice. A wide range of disciplines are concerned with occupational health since it is at an interface between technology and health involving technical, medical, social and legal aspects. Occupational health professionals include occupational health physicians and nurses, la-

bour inspectors, occupational hygienists and occupational psychologists, specialists involved in ergonomics, in rehabilitation therapy, in accident prevention and in the improvement of the working environment as well as in occupational health and safety research. The competence of these occupational health professionals should be mobilized within the framework of a multidisciplinary team approach.

5. Many other professionals from a variety of disciplines such as chemistry, toxicology, engineering, radiation health, epidemiology, environmental health, environmental protection, applied sociology, health and social insurance and health education may also be involved, to some extent, in occupational health practice. Furthermore, public health and labour authorities, employers, workers and their representatives and first aid workers have an essential role and even a direct responsibility in the implementation of occupational health policies and programmes, although they are not occupational health specialists by profession. Finally, many other professions such as lawyers, architects, manufacturers, designers, work analysts, work organization specialists, teachers in technical schools, universities and other institutions as well as the media personnel have an important role to play in relation to the improvement of the working environment and of working conditions.

6. The term «employers» means persons with recognized responsibility, commitment and duties towards workers in their employment by virtue of a mutually agreed relationship. The term «workers» applies to any persons who work, whether full time, part time or temporarily for an employer; this term is used here in a broad sense covering all employees, including management staff, the self-employed and informal sector workers (a self-employed person is regarded as having the duties of both an employer and a worker). The expression «competent authority» means a minister, government department or other public authority having the power to issue regulations, orders or other instruction having the force of law, and who is in charge of supervising and enforcing their implementation.

7. There is a wide range of duties, obligations and responsibilities as well as complex relationships among those concerned and involved

in occupational safety and health matters. In general, these duties, obligations and responsibilities are defined by statutory regulations. Each employer has the responsibility for the health and safety of the workers in his or her employment. Each profession has its responsibilities which are related to the nature of its duties. It is important to define the role of occupational health professionals and their relationships with other professionals, with the competent authority and with social partners in the purview of economic, social, environmental and health policies. This calls for a clear view about the ethics of occupational health professionals and standards in their professional conduct. When specialists of several professions are working together within a multidisciplinary approach, they should endeavour to base their action on shared sets of values and have an understanding of each other's duties, obligations, responsibilities and professional standards.

8. Some of the conditions of execution of the functions of occupational health professionals and the conditions of operation of occupational health services are often defined in statutory regulations, such as regular planning and reviewing of activities and continuous consultation with workers and management. Basic requirements for a sound occupational practice include a full professional independence, i.e. that occupational health professionals must enjoy independence in the exercise of their functions which should enable them to make judgments and give advice for the protection of the workers' health and for their safety within the undertaking in accordance with their knowledge and conscience. Occupational health professionals should make sure that the necessary conditions are met to enable them to carry out their activities according to good practice and to the highest professional standards. This should include adequate staffing, training and competence development, which includes the continuous updating of knowledge and skills, and support from and access to an appropriate level of senior management.

9. Further basic requirements for acceptable occupational health practice, often specified by national regulations, include free access to the workplace, and to relevant information needed for occupational health objectives. Other basic requirements are the possibility of taking samples and assessing the working environment, making job analyses and

participating in enquiries and consulting the competent authority on the implementation of occupational safety and health standards in the undertaking. Special attention should be given to ethical dilemmas which may arise from pursuing simultaneously objectives which may be competing such as the protection of employment and the protection of health, the right to information and confidentiality, and the conflicts between individual and collective interests.

10. The occupational health practice should meet the aims of occupational health which have been defined by the ILO and WHO in 1950 and updated as follows by the ILO/WHO Joint Committee on Occupational Health in 1995:

> *Occupational health should aim at: the promotion and maintenance of the highest degree of physical, mental and social well-being of workers in all occupations; the prevention amongst workers of departures from health caused by their working conditions; the protection of workers in their employment from risks resulting from factors adverse to health; the placing and maintenance of the workers in an occupational environment adapted to his physiological and psychological capabilities; and, to summarise, the adaptation of work to man and of each man to his job. The main focus in occupational health is on three different objectives: (i) the maintenance and promotion of workers' health and working capacity; (ii) the improvement of working environment and work to become conducive to safety and health; and (iii) development of work organisations and working cultures in a direction which supports health and safety at work and in doing so also promotes a positive social climate and smooth operation and may enhance productivity of the undertakings. The concept of working culture is intended in this context to mean a reflection of the essential value systems adopted by the undertaking concerned. Such a culture is reflected in practice in the managerial systems, personnel policy, principles for participation, training policies and quality management of the undertaking.*

11. It cannot be overemphasized that the central purpose of any occupational health practice is the primary prevention of occupational and work-related diseases and injuries. Such practice should take place under controlled conditions and within an organized framework involv-

ing competent occupational health services universally accessible for all workers. This practice must be relevant, knowledge-based, sound from scientific, ethical and technical points of view, and appropriate to the occupational risks in the enterprise and to the occupational health needs of the working population concerned.

12. It is increasingly understood that the purpose of a sound occupational health practice is not merely to perform assessments and to provide services but implies caring for workers' health and their working capacity with a view to protect, maintain and promote them and taking into account the family situation and the life circumstances outside work. This approach of occupational health practice and occupational health promotion addresses workers' health and their human and social needs in a comprehensive and coherent manner which includes preventive health care, health promotion, curative health care, first-aid rehabilitation and compensation where appropriate, as well as strategies for recovery and reintegration into the working environment. Similarly, the importance of considering the links between occupational health, environmental health, quality management, product safety and stewardship, public and community health and security is increasingly understood. This strategy is conducive to the development of occupational safety and health management systems, an emphasis on the choice of clean technologies and alliances with those who produce and those who protect in order to make development sustainable, equitable, socially useful and responsive to human needs.

BASIC PRINCIPLES

The following three paragraphs summarize the principles of ethics and values on which is based the International Code of Ethics for Occupational Health Professionals.

The purpose of occupational health is to serve the protection and promotion of the physical and mental health and social well-being of the workers individually and collectively. Occupational health practice must be performed according to the highest professional standards and ethical principles. Occupational health professionals must contribute to environmental and community health.

The duties of occupational health professionals include protecting the life and the health of the worker, respecting human dignity and promoting the highest ethical principles in occupational health policies and programmes. Integrity in professional conduct, impartiality and the protection of the confidentiality of health data and of the privacy of workers are part of these duties.

Occupational health professionals are experts who must enjoy full professional independence in the execution of their functions. They must acquire and maintain the competence necessary for their duties and require conditions which allow them to carry out their tasks according to good practice and professional ethics.

DUTIES AND OBLIGATIONS OF OCCUPATIONAL HEALTH PROFESSIONALS

Aims and advisory role

1. The primary aim of occupational health practice is to safeguard and promote the health of workers, to promote a safe and healthy working environment, to protect the working capacity of workers and their access to employment. In pursuing this aim, occupational health professionals must use validated methods of risk assessment and health promotion, propose effective preventive measures and follow up their implementation. While responding to the health and safety needs expressed by employers, workers or authorities, the occupational health professionals should be proactive in terms of improving health and safety at work on the basis of their professional competence and ethical judgment. The occupational health professionals must provide competent and honest advice to the employers on fulfilling their responsibility in the field of occupational safety and health as well as to the workers on the protection and promotion of their health in relation to work. The occupational health professionals should maintain direct contact with safety and health committees, where they exist.

Knowledge and expertise

2. Occupational health professionals must continuously strive to be familiar with the work and the working environment as well as to develop their competence and to remain well informed in scientific and technical knowledge, occupational hazards and the most efficient means to eliminate or to minimize the relevant risks. As the emphasis must be on primary prevention defined in terms of policies, design, choice of clean technologies, engineering control measures and adapting work organization and workplaces to workers, occupational health professionals must regularly and routinely, whenever possible, visit the workplaces and consult the workers and the management on the work that is performed.

Development of a policy and a programme

3. The occupational health professionals must advise the management

and the workers on factors at work which may affect workers' health. The risk assessment of occupational hazards must lead to the establishment of an occupational safety and health policy and of a programme of prevention adapted to the needs of undertakings and workplaces. The occupational health professionals must propose such a policy and programme on the basis of scientific and technical knowledge currently available as well as of their knowledge of the work organization and environment. Occupational health professionals must ensure that they possess the required skill or secure the necessary expertise in order to provide advice on programmes of prevention which should include, as appropriate, measures for monitoring and management of occupational safety and health hazards an understanding of national regulatory requirements, and, in case of failure, for minimizing consequences. The quality and effectiveness of occupational health programmes should be regularly audited in the objective of continual improvement.

Emphasis on prevention and on a prompt action

4. Special consideration should be given to the rapid application of simple preventive measures which are technically sound and easily implemented. Further evaluation must check whether these measures are effective or if a more complete solution must be sought. When doubts exist about the severity of an occupational hazard, prudent precautionary action must be considered immediately and taken as appropriate. When there are uncertainties or differing opinions concerning nature of the hazards or the risks involved, occupational health professionals must be transparent in their assessment with respect to all concerned, avoid ambiguity in communicating their opinion and consult other professionals as necessary.

Follow-up of remedial actions

5. In the case of refusal or of unwillingness to take adequate steps to remove an undue risk or to remedy a situation which presents evidence of danger to health or safety, the occupational health professionals must make, as rapidly as possible, their concern clear, in writing, to the appropriate senior management executive, stressing the need for

taking into account scientific knowledge and for applying relevant health protection standards, including exposure limits, and recalling the obligation of the employer to apply laws and regulations and to protect the health of workers in their employment. The workers concerned and their representatives in the enterprise should be informed and the competent authority should be contacted, whenever necessary.

Information, communication and training

6. Occupational health professionals must contribute to the information for workers on occupational hazards to which they may be exposed in an objective and understandable manner which does not conceal any fact and emphasizes the preventive measures. The occupational health professionals must co-operate with the employer, the workers and their representatives to ensure adequate information and training on health and safety to the management personnel and workers. In communicating about risks at work and their management, occupational health professionals are required to address language barriers, cross-cultural differences and other diversities among the management personnel and workers that may affect the effectiveness of communication. Occupational health professionals must provide appropriate information to the employers, workers and their representatives about the level of scientific certainty or uncertainty of known and suspected occupational hazards at the workplace.

Commercial secrets

7. Occupational health professionals are obliged not to reveal industrial or commercial secrets of which they may become aware in the exercise of their activities. However, they must not withhold information which is necessary to protect the safety and health of workers or of the community. When needed, the occupational health professionals must consult the competent authority in charge of supervising the implementation of the relevant legislation.

Health surveillance

8. The occupational health objectives, methods and procedures of health

surveillance must be clearly defined with priority given to adaptation of workplaces to workers who must receive information in this respect. The relevance and validity of these methods and procedures should be consistent with available scientific evidence and relevant good practice. The surveillance must be carried out with the non-coerced informed consent of the workers. The potentially positive and negative consequences of participation in screening and health surveillance programmes should be discussed as part of the consent process. The health surveillance must be performed by an occupational health professional approved by the competent authority.

Information to the worker

9. The results of examinations, carried out within the framework of health surveillance must be explained to the worker concerned. The determination of fitness for a given job, when required, must be based on a good knowledge of the job demands and of the work-site and on the assessment of the health of the worker. The workers must be informed of the opportunity to challenge the conclusions concerning their fitness in relation to work that they feel contrary to their interest. An appeals procedure must be established in this respect.

Information to the employer

10. The results of the examinations prescribed by national laws or regulations must only be conveyed to management in terms of fitness for the envisaged work or of limitations necessary from a medical point of view in the assignment of tasks or in the exposure to occupational hazards. In providing such information, the emphasis should be placed on proposals to adapt the tasks and working conditions to the abilities of the worker. General information on work fitness or in relation to health or the potential or probable health effects of work hazards, may be provided with the informed consent of the worker concerned, in so far as this is necessary to guarantee the protection of the worker's health.

Danger to a third party

11. Where the health condition of the worker and the nature of the tasks performed are such as to be likely to endanger the safety of others, the

worker must be clearly informed of the situation. In the case of a particularly hazardous situation, the management and, if so required by national regulations, the competent authority must also be informed of the measures necessary to safeguard other persons. In his advice, the occupational health professional must try to reconcile employment of the worker concerned with the safety or health of others that may be endangered.

Biological monitoring and investigations

12. Biological tests and other investigations must be chosen for their validity and relevance for protection of the health of the worker concerned, with due regard to their sensitivity, their specificity and their predictive value. Occupational health professionals must not use screening tests or investigations which are not reliable or which do not have a sufficient predictive value in relation to the requirements of the work assignment. Where a choice is possible and appropriate, preference must always be given to non-invasive methods and to examinations, which do not involve any danger to the health of the worker concerned. An invasive investigation or an examination which involves a risk to the health of the worker concerned may only be advised after an evaluation of the benefits to the worker and the risks involved. Such an investigation is subject to the worker's informed consent and must be performed according to the highest professional standards. It cannot be justified for insurance purposes or in relation to insurance claims.

Health promotion

13. When engaging in health education, health promotion, health screening and public health programmes, occupational health professionals must seek the participation of both employers and workers in their design and in their implementation. They must also protect the confidentiality of personal health data of the workers, and prevent their misuse.

Protection of community and environment

14. Occupational health professionals must be aware of their role in relation to the protection of the community and of the environment. With a view to contributing to environmental health and public

health, occupational health professionals must initiate and participate, as appropriate, in identifying, assessing, advertising and advising for the purpose of prevention on occupational and environmental hazards arising or which may result from operations or processes in the enterprise.

Contribution to scientific knowledge

15. Occupational health professionals must report objectively to the scientific community as well as to the public health and labour authorities on new or suspected occupational hazards. They must also report on new and relevant preventive methods. Occupational health professionals involved in research must design and carry out their activities on a sound scientific basis with full professional independence and follow the ethical principles relevant to health and medical research work. These include social and scientific value, scientific validity, fair subject selection, favourable risk benefit ratio, informed consent, respect for potential and enrolled subjects, review of protocols and potential conflicts of interest by an independent and competent ethics committee and protection of confidential data. The occupational health professionals have a duty to make their research results publicly available. They are accountable for the accuracy of their reports.

CONDITIONS OF EXECUTION OF THE FUNCTIONS OF OCCUPATIONAL HEALTH PROFESSIONALS

Competence, integrity and impartiality

16. Occupational health professionals must always act, as a matter of prime concern, in the interest of the health and safety of the workers. Occupational health professionals must base their judgments on scientific knowledge and technical competence and call upon specialized expert advice as necessary. Occupational health professionals must refrain from any judgment, advice or activity which may endanger the trust in their integrity and impartiality.

Professional independence

17. Occupational health professionals must seek and maintain full professional independence and observe the rules of confidentiality in the execution of their functions. Occupational health professionals must under no circumstances allow their judgment and statements to be influenced by any conflict of interest, in particular when advising the employer, the workers or their representatives in the undertaking on occupational hazards and situations which present evidence of danger to health or safety. Such conflicts may distort the integrity of the occupational health professionals who must ensure that the harm does not accrue with respect to workers' health and public health as a result of conflicts.

Equity, non-discrimination and communication

18. The occupational health professionals must build a relationship of trust, confidence and equity with the people to whom they provide occupational health services. All workers should be treated in an equitable manner, without any form of discrimination as regards their condition, gender, social aspects, convictions or the reason which led to the consultation of the occupational health professionals. Occupational health professionals must establish and maintain clear channels of communication among themselves, the senior management responsible for decisions at the highest level about the conditions and

the organization of work and the working environment in the undertaking, and with the workers' representatives.

Organizational ethics and contracts of employment

19. The public or private institutions and organizations employing occupational health professionals should adopt a programme of organizational ethics that is aligned with the ethical principles of this Code. These institutions and organizations should enable and support the conduct of occupational health professionals according to the principles of the Code. Occupational health professionals must request that a clause on ethics be incorporated in their contract of employment. This clause on ethics should include, in particular, their right to apply professional standards, guidelines and codes of ethics. Occupational health professionals must not accept conditions of occupational health practice which do not allow for performance of their functions according to the desired professional standards and principles of ethics. Contracts of employment should describe advisory roles and responsibilities, state professional independence of occupational health professionals and contain the guidance on the legal, contractual and ethical aspects. Approaches for the management of conflict, access to medical records and the protection of confidential information should also be addressed. Occupational health professionals must ensure that their contract of employment or service does not contain provisions which could limit their professional independence. In case of doubt about the terms of the contract legal advice must be sought and the competent authority must be consulted as appropriate.

Records

20. Occupational health professionals must keep good records with the appropriate degree of confidentiality for the purpose of identifying occupational health problems in the enterprise. Such records include data relating to the surveillance of the working environment, personal data such as the employment history and occupational health data such as the history of occupational exposure, results of personal monitoring of exposure to occupational hazards and fitness certificates. Workers must be given access to the data relating to the surveillance of the working environment and to their own occupational health records.

Medical confidentiality

21. Individual medical data and the results of medical investigations must be recorded in confidential medical files which must be kept secured under the responsibility of the occupational health physician or the occupational health nurse. Access to medical files, their transmission and their release are governed by national laws or regulations on medical data where they exist and relevant national codes of ethics for health professionals and medical practitioners. The information contained in these files must only be used for occupational health purposes.

Collective health data

22. When there is no possibility of individual identification, information on aggregate health data on groups of workers may be disclosed to management and workers' representatives in the undertaking or to safety and health committees, where they exist, in order to help them in their duties to protect the health and safety of exposed groups of workers. Occupational injuries and work-related diseases must be reported to the competent authority according to national laws and regulations.

Relationships with health professionals

23. Occupational health professionals must not seek personal information which is not relevant to the protection, maintenance or promotion of workers' health in relation to work or to the overall health of the workforce. Occupational health physicians may seek further medical information or data from the worker's personal physician or hospital medical staff, with the worker's informed consent, but only for the purpose of protecting, maintaining or promoting the health of the worker concerned. In so doing, the occupational health physician must inform the worker's personal physician or hospital medical staff of his or her role and of the purpose for which the medical information or data is required. With the agreement of the worker, the occupational health physician or the occupational health nurse may, if necessary, inform the worker's personal physician of relevant health data as well as of hazards, occupational exposures and constraints at work which represent a particular risk in view of the worker's state of

health.

Combating abuses

24. Occupational health professionals must co-operate with other health professionals in the protection of the confidentiality of the health and medical data concerning workers. Occupational health professionals must identify, assess and point out to those concerned procedures or practices which are, in their opinion, contrary to the principles of ethics embodied in this Code and inform the competent authority when necessary. This concerns in particular instances of misuse or abuse of occupational health data, concealing or withholding findings, violating medical confidentiality or of inadequate protection of records in particular as regards information placed on computers.

Relationships with social partners

25. Occupational health professionals must increase the awareness of employers, workers and their representatives of the need for full professional independence and commitment to protect medical confidentiality in order to respect human dignity and to enhance the acceptability and effectiveness of occupational health practice.

Promoting ethics and professional conduct

26. Occupational health professionals must seek the support and co-operation of employers, workers and their organizations, as well as of the competent authorities, professional and scientific associations and other relevant national and international organizations, for implementing the highest standards of ethics in occupational health practice. Occupational health professionals must institute a programme of professional audit of their activities to ensure that appropriate standards have been set, that they are being met, that deficiencies, if any, are detected and corrected and that steps are taken to ensure continuous improvement of professional performance.

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