AMPATH MEDICAL SURVEILLANCE GUIDELINES

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INTRODUCTION

Occupational health is a multi-dimensional field, encompassing science, social progress, economics, law, employment studies, and issues common to every family. The health and safety of people at work is a critical concern for all societies and all countries.

The field of occupational health touches on fundamental aspects of working life:

- making a living
- providing for a family
- staying healthy
- avoiding unnecessary risk of injury
- protecting oneself and others from harm
- creating useful products and services for societal benefit
- anticipating and preventing future problems
- sharing information
- the right to knowledge of potential health hazards
- fairness and justice in the treatment of workers
- achieving responsibility and accountability in the workplace
- minimizing the risk of necessary but hazardous work

Occupational health (medical surveillance and biological monitoring) excellence requires a multi-disciplinary approach to prevent health impairment that might result from excessive, acute or chronic exposure to occupational hazards and risks in particular for chemical agents. Early detection of such excessive and hazardous exposures may significantly decrease the occurrence of adverse health effects, by reducing the level of exposure, in areas and people found to be at risk. Monitoring of exposure is a procedure which consists of the routine assessment and the interpretation of biological (human) and/or ambient (environmental) parameters in order to detect the earliest physiological, abnormal and pathological changes

This approach requires a systematic Program of anticipation, recognition and identification of:

- the definition of permissible levels of exposure, that is, levels that according to the present status of knowledge are estimated to cause no adverse effects during the lifetime of the workers,
- the regular assessment of the health risks associated with exposure by comparing the employee populations health outcomes with their exposures limits and
- implementing and reviewing preventive actions and health assessments.

The World Health Organization defines Occupational Health as follows:

- Promotion and maintenance of the highest degree of physical, mental and social well-being of workers in all occupations
- Prevention among workers of departures from health caused by their working conditions
- Protection of workers in their employment form risks resulting from factors which are inimical to health
- The placing and maintenance of a worker in an occupational environment adapted to his physiological and psychological conditions.

DEFINITIONS

MEDICAL SURVEILLANCE (OCCUPATIONAL HEALTH AND SAFETY (OHS) ACT)

Medical Surveillance means 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.

HEALTH SURVEILLANCE

Health surveillance is the periodic medico-physiological examination of exposed workers (prior, during and on leaving the workplace) with the objective of protecting health and preventing occupationally-related disease. The detection and diagnosis of established disease is outside the scope of this definition even though both exposure monitoring and diagnosis of occupational disease programs are interrelated with health surveillance – health surveillance data should trigger a critical review of exposure information and ensure appropriate referral for the diagnosis of occupational disease. Similarly, as for health surveillance, exposure and occupational disease data should trigger a review of health surveillance program with necessary improvements.

BIOLOGICAL MONITORING (OCCUPATIONAL HEALTH AND SAFETY (OHS) ACT)

Biological monitoring means 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 are undertaken.

BIOLOGICAL EFFECT MONITORING

Biological effect monitoring is the measurement of a reversible biochemical change caused by the absorption of the substance,the degree of change being below that associated with toxic injury (poisoning) and not associated with a known irreversible pathological effect. Exposure can be assessed either by measuring the concentration of the agent in the workplace air by stationary or personal sampler **(ambient monitoring)**, or by measuring some biological parameters **(biological monitoring)**. Strictly speaking, biological monitoring of exposure to chemical agents means measurement of a substance or its metabolite in various biological media (text from the ACGIH in the USA, BAT in Germany and HCS regulation in SA defines the most accepted standards (BEIs – Biological Exposure Indices) and regularly updates the set of acceptable levels, however the relationship between increased levels and early reversible effects or even early adverse effects remain problematic for some substances (as we lack comprehensive studies or scientific agreement) e.g. zinc/copper/manganese in urine for "metal" exposure, acetone in urine or blood for acetone exposure, toluene-diamine in urine for TDI exposure etc.). Sometimes, the concept of biological monitoring is extended to include also the detection of early reversible non-adverse effect **(biological effect monitoring)** (e.g. δ-aminolaevulinic acid in urine or zinc protoporphyrin in blood for assessment of exposure to lead, red-cell and serum cholinesterase for organophosphate pesticide exposure etc.). The detection of an adverse effect (e.g. increased protein in urine) indicates that exposure is or has been excessive and therefore such a measurement is more logically included in a program of **early detection of health impairment** due to chemicals rather than in a biological monitoring program for evaluating exposure.

Monitoring: systematic continuous or repetitive health-related activity designed to lead, if necessary, to corrective actions.

Ambient monitoring is the measurement and assessment of agents at the workplace and it evaluates ambient exposure and health risk compared to an appropriate reference.

OCCUPATIONAL EXPOSURE LIMIT (HAZARDOUS CHEMICAL SUBSTANCES (HCS) REGULATIONS ISSUED UNDER OHS ACT)

This is the time-weighted average of an airborne hazardous chemical substance (OEL–TWA): the average concentration of a substance in the air over an eight hour work day and a 40-hour work week. Workers exposed to these levels repeatedly are not expected to develop adverse reactions. These levels are considered to be a guide line in the practice of occupational hygiene and not hard and fast rule on hazardous and non-hazardous levels. OEL-CL is the ceiling limit, which should not be exceeded and OEL-RL is the recommended limit.

BIOLOGICAL EXPOSURE INDICES (BEI) (HCS REGULATIONS ISSUED UNDER OHS ACT)

Biological Exposure Index of a hazardous substance: this level is a guideline in the monitoring of potentially toxic substances in biological samples. This index serves to alert the occupational health practitioner to the potential exposure of an individual employee to the hazard, thus ensuring correct investigation of the employees' environment and personal habits, and ensuring

appropriate action in reducing further exposure of the employee. The BEI values have been determined over an eight hour workday and a 5-day work week, and are considered to be a guideline in the practice of occupational hygiene and not a hard and fast rule on hazardous and non-hazardous levels.

TOXICOLOGY

Toxicology is the study of the adverse effects of chemicals on living organisms and the assessment of the probability of their occurrence.

HAZARDOUS CHEMICAL SUBSTANCE (HCS)

Hazardous chemical substance refers to any chemical substance (solid, liquid, gas, vapor, dust, powder, etc.) that has the potential to cause harm. This is usually a chemical that is listed in the tables published in the Regulations for Hazardous Chemical Substances of the Occupational Health and Safety Act.

BIOLOGICAL EXPOSURE INDEX (BEI)

Biological exposure index (BEI) is a reference value intended as a guideline for the evaluation of potential health hazards as listed in Table 3 of the Hazardous Chemical Substance Regulation of the Occupational Health and Safety Act 85 of 1993.

TIME WEIGHTED AVERAGE (TWA)

Eight hours per day, 40 hours per week

DOSE

This is the amount of a chemical /agent absorbed or retained in an organism during a specific time interval.

BODY BURDEN

In general this refers to the total amount of a specific chemical/agent in the body of an individual employee 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 and may contribute to the body burden so often observed in the industry.

EPIDEMIOLOGY

This is 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.

EXPERIMENTAL TOXICOLOGY (TOXICITY TESTING)

Experimental toxicology is the evaluation of the inherent toxicity of a hazardous substance. It is usually performed on laboratory animals and set standard protocols are available for the testing of toxicity. This is a laboratory-based discipline that also evaluates the potential harm to human being (employees).

There are several ways to classify toxicity. It can be classified chemically or physiologically, through the route of administration (which can be dermally, or by inhalation or by ingestion). Toxicity can be classified according to acute or chronic effects in terms of signs or symptoms, or according to site of action or effect on an organ system i.e. carcinogenic, allergenic, teratogenic, mutagenic, etc. Testing would look for long-term and specific effects of exposure to chemicals.

The most common tabulation for toxicity classes is according to the LD50 dose (LD = lethal dose) as is seen in the table on the next page. This, in practical terms, means that when 100 test animals are given the same dose, 50 will die.

Toxicity rating	Oral LD single dose (mg/kg) 50
Extremely	1 or less
Highly	1 – 50
Moderately	50 – 500
Slightly	> 0.5 – 5 g
Practically non-toxic	5 – 15
Relatively harmless	15 g and more

LABORATORY TOXICOLOGY (ANALYTICAL CHEMISTRY)

Analytical chemistry is the field of expertise consisting of laboratory-based test methods to evaluate toxicity and adverse effects in test subjects through measurement of specific substances or their metabolites. The main methods of analysis are as follows:

- Atomic Absorption Spectrophotometry – metals
- Inductively Coupled Plasma Mass Spectrometry – metals
- Electrochemical Methods – metals
- Emission Spectroscopy – metals
- Colorimetric – acids, alcohols etc.
- Mass Spectrometry – phenols (organics), phosphates etc.
- Gas Chromatography – acids, amines, alcohols, nitrates (organics) etc.
- High Performance Liquid Chromatography – organics, complex mixtures
- Liquid Chromatography – organic compounds, alcohol, ketones etc.
- Gas Chromatography – organic compounds, alcohol, ketones etc.

INTERNAL DOSE

Biological monitoring of exposure attempts to estimate the internal dose on the basis of our knowledge of the fate of the chemical in the body. But depending on the chemical and the analysed biological parameter, the term internal dose may cover different concepts.

Firstly, internal dose may mean the **amount of chemical recently absorbed**. Hence, a biological parameter may reflect the amount of chemical recently absorbed either shortly before sampling (for example, the concentration of a solvent in the alveolar air or in blood during the work shift) or during the preceding day (for example, the concentration of a solvent in alveolar air or in blood collected 16 hours after the end of exposure) or during past months when the chemical has a long biological half-time (for example, the concentration of some metals in blood). 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. For example, the concentration of polychlorinated biphenyls in blood is a reflection of the amount accumulated in the main sites of deposition (i.e. fatty tissues). Finally, with ideal biological monitoring tests, the internal dose means the amount of chemical bound to the critical sites of action (target dose or biological effective dose). Such tests can be developed, when the critical sites are easily accessible (e.g. haemoglobin (carboxyhaemoglobin) in case of exposure to carbon monoxide or to methaemoglobin forming agents) or when the chemical interacts with a blood constituent in a similar way as with the critical target molecule (e.g. haemoglobin alkylation reflecting binding to DNA in the target tissue). In the latter situation the amount bound to the blood constituent is used as a surrogate of the biologically effective dose

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BIOLOGICAL MONITORING

The objective of medical surveillance is to cover the spectrum of potential effects of a hazardous chemical substance on an employee from absorption of the substance through to clinical disease. Medical surveillance will include a history taking and physical assessment component as well as the following separate entities: biological monitoring and biological effect monitoring, which are considered below.

Biological monitoring measures the biochemical concentrations of HCS 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 body by measuring indicators in representative biological samples, typically urine or blood (usually not related to the target organ).

CLASSIFICATION OF BIOLOGICAL MONITORING APPROACHES

The biological tests currently used for monitoring of exposure to chemicals can be classified in three categories.

DETERMINATION OF THE CHEMICAL OR ITS METABOLITES IN BIOLOGICAL MEDIA OR EXHALED AIR

The great majority of the tests currently available for biological monitoring of exposure to chemical rely on the determination of the chemical or its metabolites in biological media. The biological media most commonly used are urine, blood and 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. The **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 substances) or when sensitive methods for detecting the metabolites are not available.

Techniques are also being developed (neutron activation analysis/x-ray fluorescence) for directly monitoring in vivo the concentration of some metals in tissues such as cadmium in liver and kidney and lead in bones. So far, these techniques are not applicable routinely.

Non-selective tests are used as non-specific indicators of exposure to a group of chemicals. As an example of non-selective exposure tests, one can cite the determination of diazo-positive metabolites in urine for monitoring exposure to aromatic amines, the analysis of thio-ethers in urine to assess exposure to mutagenic and carcinogenic substances and the determination of the mutagenic activity of urine. 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.

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 pseudo-cholinesterase activity in serum to assess exposure to organophosphorus compounds. Others include the inhibition of the erythrocyte enzyme δ-aminolaevulinic dehydratase and the increase of zinc protoporphyrin which are generally considered as indicators of lead exposure, although they are not agent specific as such. Also the urinary excretion of beta-hydroxycortisol or D-glucaric acid may be used as an indicator of exposure to chemical inducing mono-oxygenase enzymes.

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. Progress in this monitoring approach is to be expected namely with the development of a new generation of tests based on immunological or GC-MS techniques. The latter have the potential of detecting with a great specificity (agent specific parameter) and sensitivity subtle alterations

induced in the target and non-target molecules (e.g. DNA, haemoglobin, albumin) by reactive chemicals (e.g. mutagens and carcinogens). The DNA adducts can be measured either in hydro lysates of DNA molecules (e.g. in white blood cells) or in degradation products of DNA released in body fluids (e.g. urine). Much research is still needed before these tests can be introduced in the routine biological monitoring of workers.

COMPARISON OF AMBIENT AND BIOLOGICAL MONITORING OF EXPOSURE

Biological monitoring of exposure may offer several advantages over environmental monitoring to evaluate the internal dose and hence to estimate the health risk. Biological monitoring takes into consideration absorption through the skin or the gastrointestinal tract. Personal hygiene habits vary from one person to another. The lack of care in personal hygiene can lead to significant ingestion of the substance (hand 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.

Moreover, it is well known that great inter-individual variation exists in the absorption rate of a chemical through the lungs, the skin or the gastrointestinal tract as well as in the capacity for metabolising and excreting the substance. In some cases, even if strict personal hygiene measures can be 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 work load 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.

Biological monitoring also reflects non-occupational background exposure (leisure activity, residency, dietary habits, smoking, etc.) which may also be expressed in the biological level. The organism integrates the total external (environmental and occupational) exposure into one internal load.

For all of the above reasons, it is clear that for many industrial pollutants, the measurement of the concentration in air (low level) may not necessarily prevent an excessive intake by the exposed workers. Ambient monitoring is usually done to identify and quantify specific contaminants present in the environment, to determine compliance status with respect to various occupational health standards or to evaluate the effectiveness of engineering controls installed to minimize workers' exposure. Depending on the type of air sampling system selected – stationary (area) or personal – the estimate of the risk may be carried out on a group or on an individual basis. Ambient monitoring is more suited 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. Individual measurements, such as biological monitoring may entail higher costs than air monitoring.

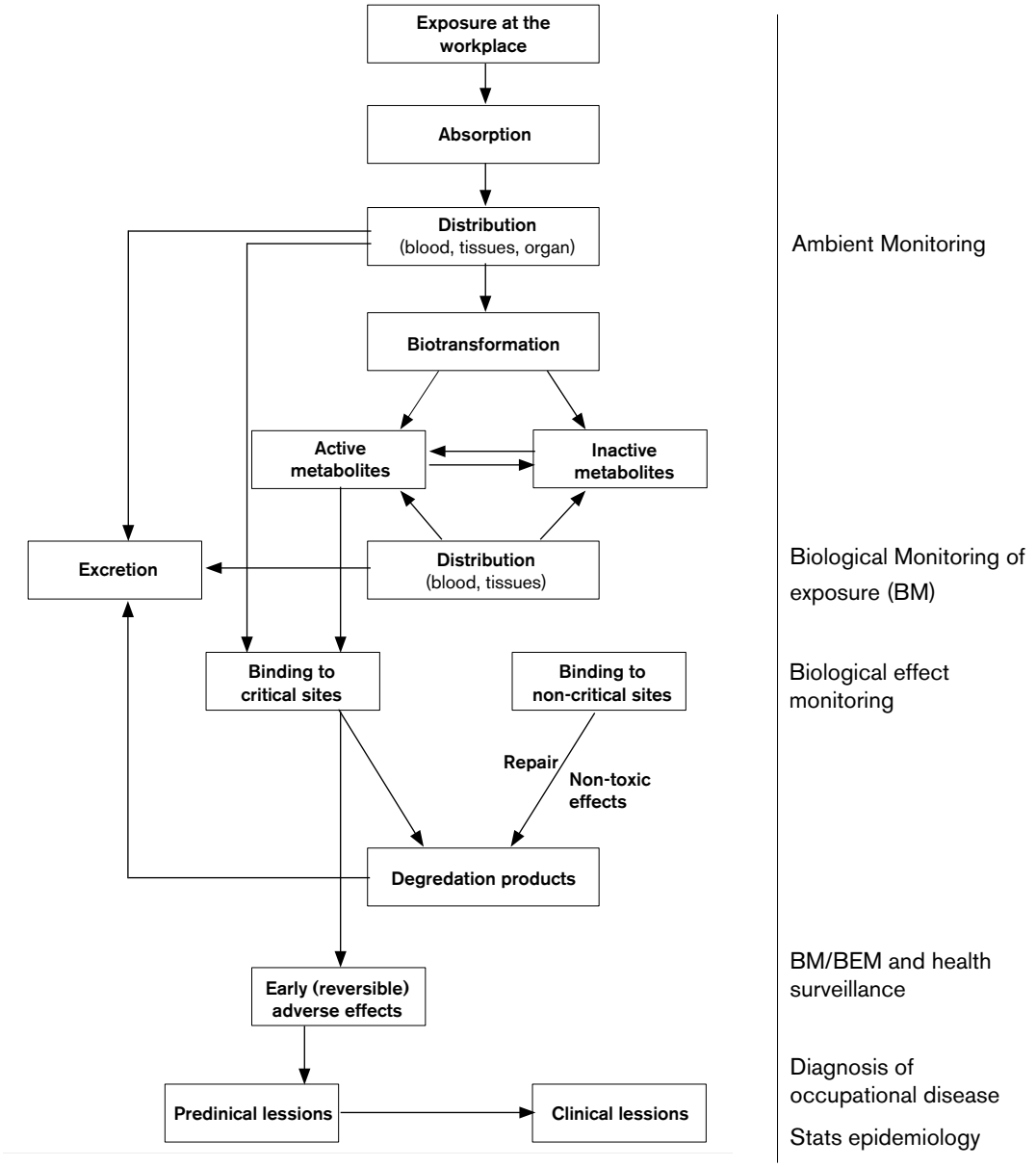
Moreover, in the case of exposure to chemical substances that exhibit their toxic action at the site of contact (e.g. eye mucosa or lung irritants, respiratory tract carcinogens) and are poorly absorbed, a biological parameter reflecting the internal dose is not necessarily related to the health risk. Only a few biological tests have been proposed for the identification or the monitoring of chemicals present at the interface between the environment and the body (skin, gastrointestinal mucosa, respiratory tract mucosa). The analysis of nickel in the nasal mucosa which is at present only an experimental technique, and the counting of asbestos bodies in sputum could be considered as examples of such tests. Except for these very few tests, direct toxic effects at this interface between the environment and the body, can only be prevented by keeping the airborne and surface concentrations of the substance below a certain level.

There is sometimes greater difficulty in obtaining biological samples (e.g. blood collection may be considered as invasive by workers) than air samples. 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.

IMPLEMENTATION OF SAMPLING FOR BIOLOGICAL MONITORING

BIOLOGICAL MEDIA

The majority of the 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.



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 since depending on the substance; the analysis should be performed on whole blood, plasma, serum or erythrocytes. The appropriate anticoagulant (if necessary) must be selected. The biological parameter to be assessed can be either equally distributed between the different blood constituents or can 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. Some chemicals or their metabolites can be transported in blood free or bound to proteins. The analytical method must take this property into account.

The blood concentration of many volatile solvents has frequently 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.

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 effectively more 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, end of shift samples are more appropriate. It should, however, be realized 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.

The determination of urinary creatinine and/or density 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. 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 re-absorption. 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 organ abnormalities– e.g. lung, liver and blood cells may need to be excluded or noted).

COLLECTION OF SPECIMENS

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 the table below for the BEI sampling time.)

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

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ACCEPTABILITY OF URINE SPECIMENS

Urine specimens that are highly dilute 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

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

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 as 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.

GUIDELINES ON BLOOD AND URINE SAMPLE COLLECTIONS

BLOOD COLLECTION

- **Responsibilities of the company**
  - Provide clean environment
  - Clean working surfaces
  - Chair(s) for employee(s)
  - Clean water to wash arms, Savlon or soap or, if at all possible, a shower and clean overall
  - Preferably paper towels to dry arms.
- **Responsibilities of the Practitioner taking blood**
  The person from whom the blood is to be taken must have either:
  - Showered and put on a clean overall; or
  - Come directly from home and therefore not exposed to the work environment; or
  - Removed the contaminated overall 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.
  - Informed consent must be collected from the person being tested
- **Procedure for taking blood**
  - Spirits should be available for use
  - Clean venipuncture area with spirits
  - Apply tourniquet
  - Insert needle, place the tube into the needle, release the tourniquet
  - Complete blood-taking procedure
  - Remove filled blood-tube and tilt gently several times
  - Remove needle from arm and cover venipuncture site with clean swab, pressing firmly for about two minutes or until bleeding has stopped
  - Do not bend arm
  - Apply plaster

URINE COLLECTION

- **Responsibility of the company**
  - Provide clean environment
  - Provide showers and clean toilet facilities
- **Responsibility of the occupational health nurse**
  - Ensure the employee showers prior to urine collection; or the worker produces a urine sample on entering the factory site before changing out of personal clothing into working overalls.
  - The sample should be collected either as a pre-shift sample (collected on the morning of the first work-day) 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.
- The sample should be collected into a clean urine container.

- **Procedure for collecting a urine sample**
  - If possible ensure that the employee is well-hydrated by giving 200mls 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 20 – 30 ml 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.

QUALITY ASSURANCE

Each aspect of biological monitoring 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 a representative sample of 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 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 to lab requests.

In addition 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). 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 accurately measure the worker's BEI®. When blind challenges are used, the spiked determinant should be in the same chemical form and matrix as that being analysed by the laboratory.

NOTATIONS

- **B** denotes background, and refers to a determinant that may be present in biological specimens collected from non-occupationally exposed subjects, at a concentration which could affect interpretation of the result. The BEI® value incorporates these background concentrations.
- **Nq** meaning non quantitative, indicates compounds for which biological monitoring should be considered. This is based on the review, although there was insufficient data to determine a specific BEI®.
- **Ns** refer to non-specific determinants, as they are also observed after exposure to other chemicals.
- **Sq,** i.e. semi quantitative, refers to a biological determinant that is an indicator of exposure to the chemical, although the quantitative interpretation of the measurement is ambiguous. These should only be used as screening tests if quantitative tests are impractical or as confirmatory tests if the quantitative test is non-specific and the determinant's origin is questionable.

APPLICATION OF BEIS®

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, dependence 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 eight 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, regard- less 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.

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 (BEIs®) 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 terms, 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 as 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 some 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 confounding 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 enzymes 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 thioethers concentration in urine and the mutagenic activity of urine. 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 has to consider the possible physico-chemical 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:

- independent, where the substances exert their own toxicity independent of each other,
- additive, where the combined effect of the two chemicals is equal to the sum of the effects produced by the individual agents,

- synergistic, where the combined effect of the two chemicals is much greater than the sum of the effects of each agent given separately,
- antagonistic, where two chemicals administered together interfere with each other, or
- potentiating, where a substance of low or no toxicity enhances the toxicity of another chemical.

Results are generally interpreted by 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 by 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 or non-occupational exposure, or genetic polymorphism.

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BIOLOGICAL EFFECT MONITORING

Biological effect monitoring determines the intensity of biochemical or physiological change 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 factors that should be considered and how they may impact the testing.

- 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 environment: pollution of drinking water and food; air pollution; working with toxic substances (hobbies/sport etc.). The work exposure is then added to by the environmental exposure increasing the BM results.
- Behavioural patterns: personal hygiene; smoking; substance abuse; failure to change overalls; 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 in the workplace and ingests chemicals through this route.
- Timing of specimen collection.
- Inappropriate specimen collection: i.e. contamination of the sample and its container by the environment: the inappropriate storage and preservation of the sample; utilization of inappropriate sample collection equipment, e.g. stainless steel needles may contaminate a blood sample for chrome or nickel analysis.
- Laboratory analysis error: the laboratory should practice disciplined quality control procedure, utilizing the appropriate equipment for the analysis of the substance.

HEALTH PROGRAM

The basic principle of Occupational Health is to protect and prevent occupational disease. Good occupational health practice provides for individual employee health protection and prevention through anticipation, recognition, identification and monitoring of workplace stresses and employees health. More and more the risk assessment, occupational risk and exposure profiling is becoming individualized as opposed to generalized (homogeneous exposure group) health programs. However in many instances it is worthwhile to consider an “occupation or risk based” occupational health program, in particular for specialized occupations, food handlers and 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.

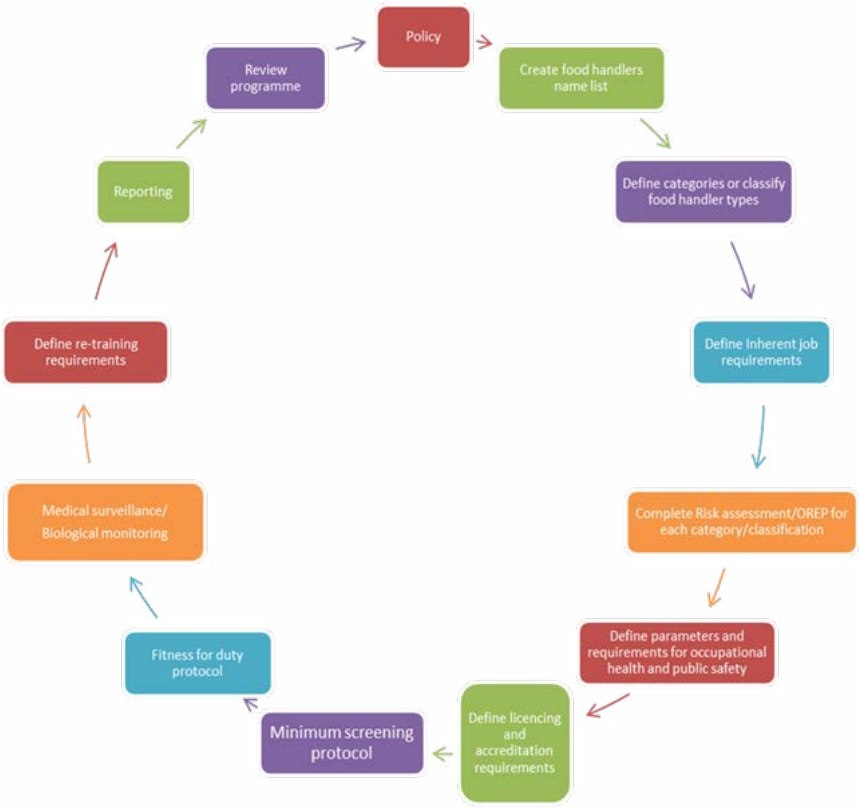
The principle of occupational health practice does not differ from the occupational or risk based approach, in fact it strengthens it. The purpose of occupational health is to serve the health and social well-being of the workers individually and collectively. Occupational health practice must be performed 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 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.

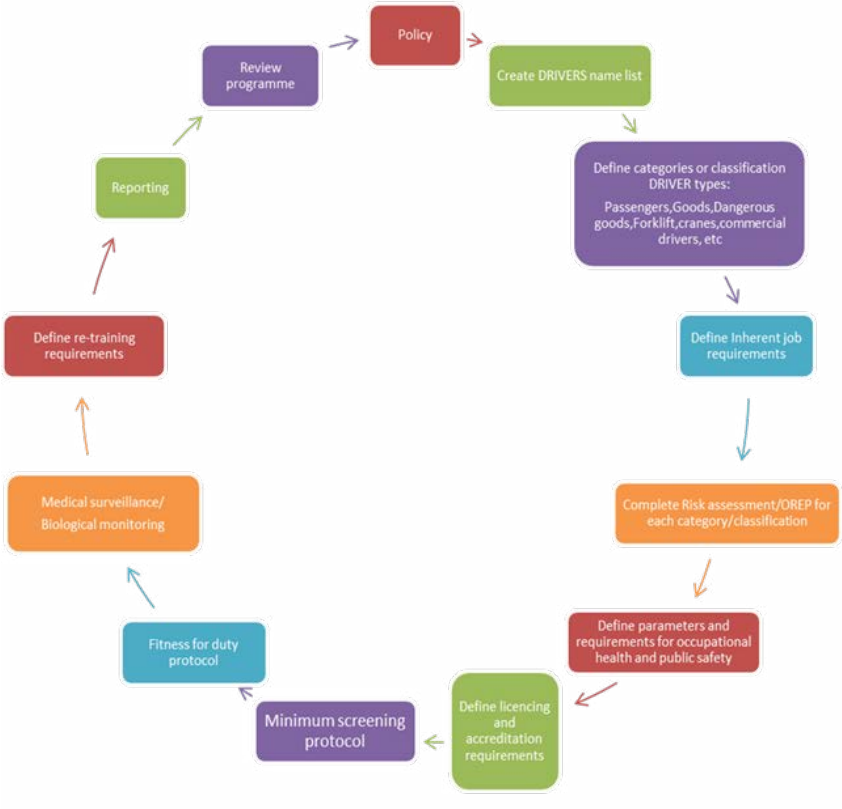
Occupational health professionals are experts who must enjoy full professional independence in the execution of their functions. They must acquire and maintain the competence necessary for their duties and require conditions which allow them to carry out their tasks according to good practice and professional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

Below find a basic framework for food handlers’ and driver’s health program, as such all occupational health programs is evolutionary in nature, and requires regular review, consultation and updates. This framework can be adapted for any occupation, risk or workplace.

FOOD HANDLER'S HEALTH PROGRAMME



DRIVER'S HEALTH PROGRAMME



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SUBSTANCE ABUSE IN THE WORKPLACE

ALCOHOL IN THE WORKPLACE

The abuse of alcohol or drugs in the workplace while on duty, the consumption of alcohol or drugs before coming on duty is a 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 in the workplace results in poor job performance, lack of focus, absenteeism, increased health-related problems and use of medical aid funds.

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 provides some term 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 organization, big or small, workplace alcoholism is especially prevalent in the following industries:

- food service,
- construction,
- mining and drilling,
- excavation, and
- 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.

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.

Testing of an employee to determine that employee's HIV status is prohibited unless the Labour Court determines such testing justifiable". This section of the Employment Equity Act as it relates to medical testing, applies to all employees and employers except, members of the National Defence Force, National Intelligence Agency, South African Secret Service, South African National Academy of Intelligence, and to the directors and staff of Comsec.

THE OHSA NO. 85 OF 1993

The relevant provisions of the OHSA are contained in Sections 8 and 14, and Section 2a of the general safety regulations. 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;"

Section 2A has been inserted by the Minister of Labour into the regulations in 2003 and relates with intoxication as follows:

- 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.
- 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.
- 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 the both employers and employees. They include:

- It is being recognized that alcohol abuse is a growing problem impacting on health and safety in the workplace;
- The provision for compulsory drug and alcohol testing to provide a safe working environment;
- The frequency of testing depends on the nature of work e.g. drivers of heavy vehicles compared with highly specialized 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,
- The OHSA could therefore offer employers with an opportunity and a justification for implementing random testing to ensure that employees are not intoxicated while performing their duties.

In order to comply with the OHSA Act and Labour laws, it is recommended that employers consider the following when implementing a program in their workplace. It usually requires:

- a substance abuse policy,
- creating awareness by training and education,
- testing, and
- an employee assistance program/ EAP.

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:"

- zero tolerance, allowance for limits, and whether to relate limits with level of functioning to decide on fitness for duty;
- the policy must stipulate your test procedure e.g. breathalyser test for alcohol;
- 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 his feet, dishevelled appearance, aggressive or abusive or arrogant or out of character behaviour, and the inability to walk a 10 meter 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? What method of testing is to be used? Who is responsible for the testing? What are 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 response?
- 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 to be made by the employer:

Workers consider privacy to be a fundamental right. The use of alcohol is normally done in the worker's free time and off-premises. The choice of testing that involves taking samples of bodily fluids is often considered by workers as intrusive, humiliating and degrading as it is done under watchful eyes. The timing of testing is also a contentious issue. Should testing be done randomly, pre-employment, when there is reasonable suspicion, post-accident, periodically, or in safety-sensitive jobs? The use of questionnaires in the workplace to elicit the prior or current use of alcohol is often regarded as an invasion of privacy by employees. The recommended WHO AUDIT questionnaire is designed to be used as a screening tool. The employer

perspective is that it is found to be unreliable and largely dependent on honesty and mental status. Observation and monitoring by supervisors for signs of intoxication is favoured by employees relative to the other methods. This again becomes unethical and speculative unless supervisors are confined to instances where an employee is clearly under the influence and hence cannot function in the job at an acceptable level of performance.

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. The laboratory offers the following tests as markers of alcohol use shown in Table below.

ALCOHOL USE

Analyte	Cut-off	Sensitivity	Specificity	Use	Amount and time of alcohol use to cause abnormal marker	Time to normalize with abstinence	Comment
Ethanol	0.05 g/dL (SA legal limit when driving)			Detect 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 1hr: > 2 beers in 70 kg person.	Hours, depend on dose	Short detection time limits use.
GGT-CDT index	Male 4.18 Female 3.81	89 96	98 97	Detect heavy drinking.	> 40 g/day (3 or more beers/day) for more than 1min	2–3 weeks	GGT-CDT is calculated by a mathematical formula that weighs GGT and %CDT. Increased sensitivity without affecting specificity. Detects more alcohol abusers than CDT and GGT alone. Performance is similar whether or not heavy drinkers are contrasted with abstainers or moderate drinkers which are useful for screening. Correlates with the amount of alcohol used. Use of MCV, ALT or AST as a 3rd 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. Detect heavy drinking for at least 1w in alcoholics.	50–80 g/d (4–6 beers/d) for at least 1w in alcoholics.	2–4 weeks	%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. Cutt-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 normalize with abstinence	Comment
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	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	Mean corpuscular volume (MCV) is the size of the red blood cells. Good specificity (very few teetotallers 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 (anticonvulsants, azathioprine or zidovudine).
AST/ALT (indirect)	> 2	Low	90	Detect alcohol-induced liver damage.			Indicates advanced alcoholic liver disease rather than heavy alcohol consumption

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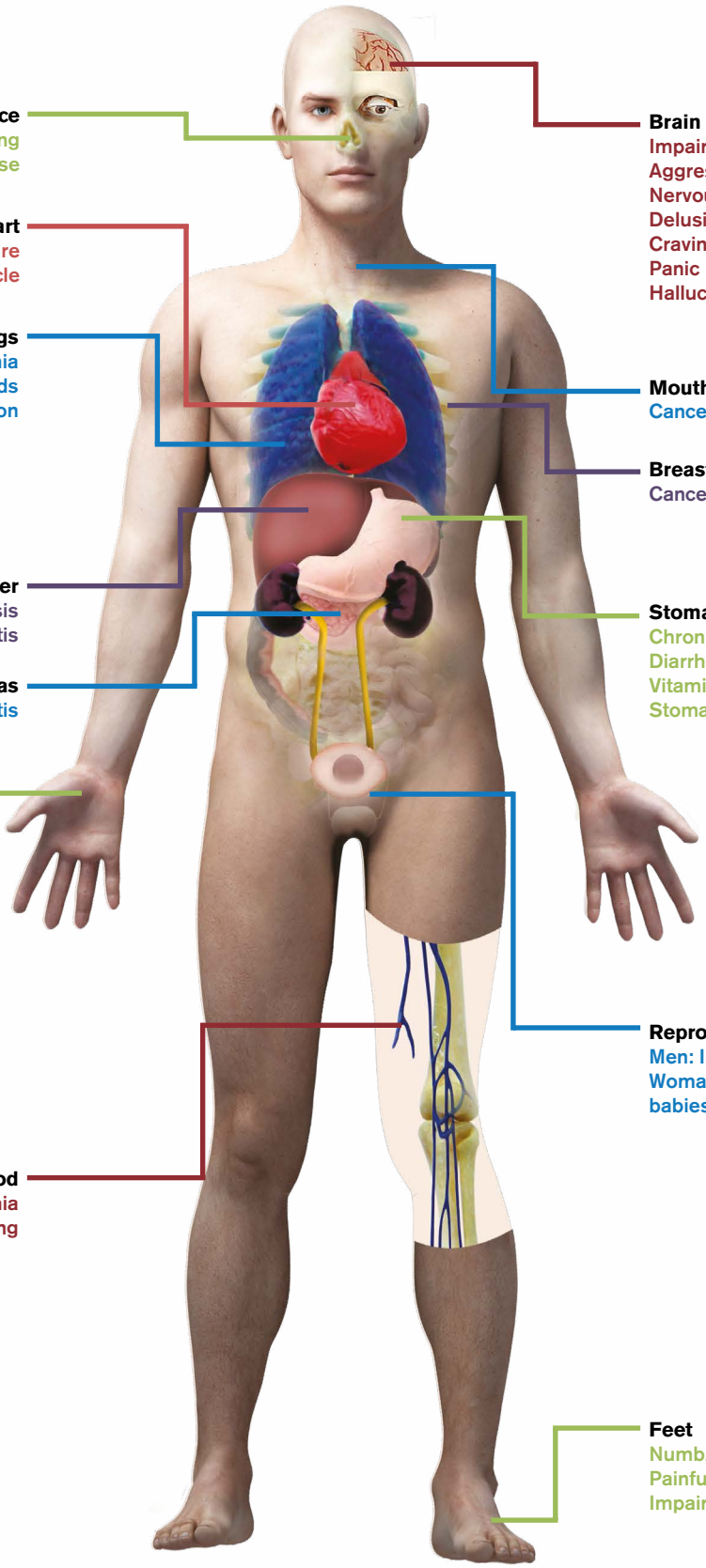
TARGET ORGANS FOR ALCOHOL ABUSE



Nose and face
Pre-mature aging
Drinker's nose

Heart
Heart failure
Weakness of heart muscle

Lungs
Increased risk of pneumonia
Frequent colds
Reduced resistance to infection

Liver
Cirrhosis
Hepatitis

Pancreas
Pancreatitis

Hands
Trembling
Tingling finger
Numbness
Painful nerves

Blood
Anaemia
Impaired blood clotting

Brain
Impaired memory
Depression
Aggresive, irrational behaviour
Nervousness
Violence
Delusions
Irritability
Cravings
Anxiety
Panic
Psychosis
Hallucinations
Sleep disorders

Mouth, trachea and esophagus
Cancer

Breast
Cancer

Stomach
Chronic gastritis
Vomiting
Diarrhoea
Malnutition
Vitamin deficiency
Stomach inflammation

Reproductive system
Men: Impaired sexual performance
Woman: Risk of giving birth to deformed babies/low birth-rate babies

Feet
Numb, tingling toes
Painful nerves
Impaired sensation leading to falls

DRUGS IN THE WORKPLACE

Even though drug abuse may be viewed to be an individual's problem, the abuse directly impacts on the workplace as well as on co-workers. The impact of drug abuse focuses of four major issues in the workplace:

- Loss of production
- Extra sick leave/absenteeism
- Increased injuries/accidents
- Fatal accidents

The aim of drug abuse testing is to provide a safe and productive working environment. The South African courts have found that the prevention of alcohol abuse at work is a management responsibility and that it is the employer who should ensure that employees may not commence with work if drunk .This would also apply to employees under the influence of drugs. As with alcohol abuse in the workplace, it is important to understand the legal framework that informs the policy an employer will implement in his/her workplace. The two important acts to consider is the Employment Equity Act No. 55 of 1998, and the OHSA No. 85 of 1993. 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 the job.

In many cases substance abuse is a dismissible offence and the drug policy should therefore be jointly created by both employee representatives and employer.

ELEMENTS OF A DRUG POLICY WOULD TYPICALLY INCLUDE:

- Purpose and objective of the program
- Definition of substance abuse
- Statement of who is covered by the program
  - Job categories/High risk areas
- Statement on employee's right to confidentiality
- Training to be provided to prevent abuse
- Restriction/control of illegal drugs in the workplace
- How to deal with impaired workers
- How chronic users will be assisted e.g. rehabilitation programs
- Conditions under which drug testing will be conducted
  - Pre-employment/random testing/cause testing
- Test methods to be used for screening and confirmatory tests
- Disciplinary actions

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 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	DOAQL				
(* below incl. 10 panel)	DOA (# below Immunoassay+ Confirmatory incl. 10 panel)	NB: arrange chain of custody if needed			
Amphetamine	AMPHS*	AMPH#	AMPHC	Speed, Crystal, Ice, Uppers	1–4 d
Barbiturates	BARBUS*	BARBU	MISCC	Blue heavens, Velvet, Devil, Red devils, Pink lady, Purple hearts	1–14 d
Benzodiazepines	BENZUS*	BENZU#	MISCC	Benzos, Mellow, Downers, Ativan, Rohypnol, Valium, Serepax	1–9 days, up to 30 days
Cannabis	CANNS*	CANN#	CANC	Dagga; Marijuana; Pot; Weed; ,Whoonga (Nyaope)	2–5 d infrequent use); 3–4 w (chronic use); 6–11 w (heavy use)
Cocaine (Benzoyl ecgonine)	COCAS*	COCA#	COCC	Crack; Coke; Rock; Snow; Flake; Blow	2–3 d up to 9 d
Ecstasy (MDMA, MDA, MDEA)	-	-	ECST#	"X"	At least 24 h
LSD (Lysergic Acid Diethylamide)	-	-	LSDC	Acid	0–2 d
Mandrax (Methaqualone)	-	MAND#	MANDC	Mandrax; Soaps; Love Pill	90–225 hours
Methadone	METHS*	METH#	MISCC	Meth; Methadone	1–3 d
Methamphetamine (Tik)	METAMQ*	-	METAM#	Tik-Tik	1–4 d
Methcathinone (CAT)		-	METHCAT#	CAT	1–4 d
Opiates 6-Acetyl morphine (Heroin),Codeine Morphine	OPIAS*	OPIA#	OPIAC	Morphine: Junk, White Stuff, "M", Heroin: Horse, White Lady, "H"	7–54 h (infrequent use) Up to 12 d (chronic use)
PCP (Phencyclidine)	PCPS*	-	PCPC	PCP, Angel dust	1d – 2w, up to 1m
PPX (Propoxyphene)	-	-	PPXC	PPX, Doloxene	1–2 d
Tricyclic antidepressants	TADU*	-	-		

CONDITIONS UNDER WHICH A PATHOLOGIST WILL TESTIFY IN COURT

- Consent given by worker
- Worker fully informed on 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
- If Chain of Custody was followed

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 resulting of a sample.

HOW SHOULD SAMPLES BE COLLECTED?

- Give the worker the labelled tamper proof specimen container.
- Specimen should be collected under supervision to exclude tampering.
- On receipt of the sample- after collection-ensure that the sample was freshly passed (should still be at body temperature).
- An adulteration test will be performed on all confirmatory samples received.

WHAT DOCUMENTATION MUST BE COMPLETED?

- Ampath generic requisition form.
  - Ensure that the patient signs this form.
  - Note on this form what specific drug needs to be confirmed.
- Chain of custody requisition form.
  - Ensure that the patient signs this form.
  - Mnemonic COCD is preprinted on this form – DO NOT add any other mnemonics as this will influence the Chain of Custody trail.

HOW MUST THE SAMPLE BE SENT TO AMPATH?

- Label envelope as follows:
  Ampath
  CHAIN OF CUSTODY SAMPLE
  Esoteric Science
  166 Witch Hazel Avenue
  Techno Park
  Centurion
- Place urine container in an Ampath specimen plastic bag.
- Place Generic Ampath request form as well as urine sample (in plastic bag) in envelope in front of the worker.
- Seal envelope. Worker and sister to sign across the envelope's seal.
- Complete section "Envelope sealed by" on Chain of Custody (COCD) form.
- Attach COCD request form to envelope and place in plastic envelope
- Messenger collecting the sample to sign at section: "Collecting drivers name:"
- Processing, data capture and forwarding to Esoteric Science as per Ampath internal policy

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HAZARDOUS CHEMICAL SUBSTANCE REGULATIONS

HAZARDOUS CHEMICAL SUBSTANCES REGULATIONS (OHS ACT NO 85 OF 1993)

DESIGNING AND IMPLEMENTING A PROGRAM OF MEDICAL SURVEILLANCE

The following steps should be included in any program:

- Risk assessment to determine the potential exposure to and routes of absorption of any HCS, as required by regulation 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 HCSs in the form of BEIs in Table 3 of the Regulations. Criteria for interpreting spirometry have also been published. However, in many cases, the OHP will have to develop pragmatic criteria in the context of the specific workplace.
- Standardisation of test 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 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 by 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 the employer needs to be notified of such details of the medical findings as are necessary to evaluate the workplace problem and take remedial action.
- Record-keeping. Includes both medical records and exposure information for every employee. While the employer is responsible for record-keeping 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.

ASSESSMENT OF POTENTIAL EXPOSURE

1. An employer or self-employed person shall, after consultation with the relevant health and safety representative or relevant health and safety committee, cause an immediate assessment to be made and thereafter at intervals not exceeding two years, to determine if any employee may be exposed by any route of intake.
2. An employer shall inform the relevant health and safety representative or relevant health and safety committee in writing of the arrangements made for the assessment contemplated in sub regulation (1), give then reasonable time to comment thereon and ensure that the results of the assessment are made available to the relevant representative or committee who may comment thereon.
3. When making the assessment, the employer or self-employed person shall keep a record of the assessment and take into account such matters as:
   a. The HCS to which an employee may be exposed
   b. What effects the HCS can have on an employee
   c. Where the HCS may be present and in what physical form it is likely to be
   d. The route of intake by which and the extent to which an employee can be exposed; and
   e. The nature of the work, process and any reasonable deterioration or failure of any control measures
4. If the assessment made in accordance with sub regulation (3) indicates that any employee may be exposed, the employer shall ensure that monitoring is carried out in accordance with the provisions of regulations 6 and 7 and that the exposure shall be controlled as contemplated in regulation 10.

5. An employer shall review the assessment required to sub regulation (1) forthwith if:
   a. There is reason to suspect that the previous assessment is no longer valid; or
   b. There has been a change in a process involving an HCS or in the methods, equipment or procedures in the use, handling, control of processing of the HCS, and the provisions of sub regulations (2) and (3) shall apply.

TABLE 3 SUBSTANCES

CHEMICAL	METABOLITE/SUBSTANCE MEASURED	SAMPLING TIME
ANILINE	Total p-Aminophenol in urine Methaemoglobin in blood	End of shift During or end of shift
ARSENIC AND SOLUBLE COMPOUNDS INCLUDING ARSINE	Inorganic arsenic metabolites in urine	End of work-week
BENZENE	Total Phenol in urine Benzene in exhaled air: Mixed-exhaled End-exhaled	End of shift Prior to next shift
CADMIUM	Cadmium in urine Cadmium in blood	Not critical Not critical
CARBON DISULFIDE	2-Thiothiazolidine-4-carboxylic acid in urine	End of shift
CARBON MONOXIDE	Carboxyhemoglobin in blood Carbon monoxide in end-exhaled air	End of shift End of shift
CHLOROBENZENE	Total 4-Chlorocatechol in urine Total p-Clorophenol in urine	End of shift End of shift
CHROMIUM (VI),	Water soluble fume Total chromium in urine	Increase during shift End of shift at end of work-week
N,N-DIMETHYLFORMAMIDE (DMF)	N-Methylformamide in urine	End of shift
ETHYL BENZENE	Mandelic acid in urine Ethyl benzene in end-exhaled air	End of shift at end of work-week
FLUORIDES	Fluorides in urine	Prior to shift End of shift
FURFURAL	Total Furoic acid in urine	End of shift
n-HEXANE	2,5-Hexanedione in urine n-Hexane in end-exhaled air	End of shift
MERCURY	Total inorganic mercury in urine Total inorganic mercur y in blood	Prior to shift End of shift at end of work-week
METHAEMOGLOBIN INDUCERS	Methaemoglobin in blood	During or end of shift
METHANOL	Methanol in urine Formic acid in urine	End of shift Before shift at end of work-week
METHYL CHLOROFORM	Methyl chloroform in end-exhaled air Trichloroacetic acid in urine Total trichloro-ethanol in urine Total trichloroethanol in blood	Prior to the last shift of work-week End of work-week End of shift at end of work-week End of shift at end of work-week
METHYL ETHYLKETONE	MEK in urine	End of shift
METHYL ISOBUTYL KETONE	MIBK in urine	End of shift
NITROBENZENE	Total p-Nitrophenol in urine Methaemoglobin in urine	End of shift at end of work-week End of shift
ORGANOPHOSPHORUS CHOLINESTERASE INHIBITORS	Cholinesterase activity in red cells	Discretionary
PARATHION	Total p-Nitrophenol in urine Cholinesterase activity in red cells	End of shift Discretionary
PENTACHLOROPHENOL	Total PCP in urine Free PCP in plasma	Prior to the last shift of work-week End of shift
PERCHLOROETHYLENE	Perchloroethylene in end-exhaled air Perchloroethylene in blood Trichlorocetic acid in urine	Prior to the last shift of work-week Prior to the last shift of work-week End of work-week
PHENOL	Total Phenol in urine	End of shift
STYRENE	Mandelic acid in urine Phenylglyoxylic acid in urine Styrene in venous blood	End of shift Prior to next shift End of shift Prior to next shift End of shift Prior to next shift

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CHEMICAL	METABOLITE/SUBSTANCE MEASURED	SAMPLING TIME
TOLUENE	Hippuric acid in urine Toluene in venous blood o-Cresol in urine	End of shift End of shift End of shift
TRICHLOROETHYLENE	Trichloroacetic acid in urine Trichloroacetic acid and trichloroethanol in urine Free trichloroethanol in blood Trichloroethylene in end-exhaled air	End of work-week End of shift at end of work-week End of shift at end of work-week
XYLENE	Methylhippuric acid in urine	End of shift Last four hours of shift

AVAILABLE TESTS

CHEMICAL EXPOSURE PROFILES

CHEMICAL-METABOLITE	SPECIMEN	MNEMONIC	CONTAINERS
Acetone exposure:			
Acetone	urine	ACET	UR 25ml
Aniline exposure:			
Methaemoglobin	blood	MHB	Heparin
Benzene exposure:			
Phenol	urine	BEN	UR 25ml
tt Muconic acid	urine	BENT	UR 25ml
Phenylmercapturic acid	urine	BENP	UR 25ml
Benzene	blood	BENB	EDTA 1
Carbaryl exposure:			
1-Napthol	urine	NAPHT	UR 25ml
Carbon Disulphide exposure (2-thiothiazolidine):			
TTCA	urine	CDE	UR 25ml
Coal tar exposure (Polycyclic aromatic hydrocarbon exposure):			
1-Hydroxypyrene	urine	PAH	UR 25ml
Cyanide exposure:			
Cyanide(acute exposure)	blood	CYAN	EDTA 1
Thiocyanate	urine	THIOU	UR 25ml
Dimethylformamide exposure:			
Formamide + N-Methylformamide	urine	DMF	UR 25 ml
Ethyl benzene exposure:			
Mandelic acid + Phenylglyoxylic acid	urine	EB	UR 25 ml
Ethyl benzene	blood	EBB	EDTA 1
Ethylene Glycol Exposure:			
Oxalic acid	urine	EGE	UR 25ml
Formaldehyde exposure:			
Formic acid	urine	FOR	UR 25ml
Furfural exposure:			
Furoic acid	urine	FUR	UR 25ml

CHEMICAL-METABOLITE	SPECIMEN	MNEMONIC	CONTAINERS
Glue and thinners sniffing:			
Toluene	urine	TOLCH	UR 25ml
Phenol	urine	BEN	UR 25ml
tt Muconic acid	urine	BENT	UR 25ml
Phenylmercapturic acid	urine	BENP	UR 25ml
Xylene	urine	XYL	UR 25ml
Ethylbenzene	urine	EB	UR 25ml
Herbicide exposure:			
Paraquat	urine	PARAQ	UR 25ml
Hexane exposure:			
2,5 Hexanedione	urine	HEXAN	UR 25ml
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	UR 25ml
Isopropanol exposure:			
Acetone	urine	ISOP	UR 25ml
Methanol exposure:			
Formic acid	urine	METHE	UR 25ml
Methanol	urine	METHU	UR 25ml
Methanol	blood	METHB	FLUORIDE 2
Methylene chloride exposure:			
Dichloromethane	blood	DCMB	EDTA 2
Carboxyhaemoglobin	blood	CHB	HEP1
Methylethyl ketone (MEK) exposure:			
Methylethyl ketone (MEK)	urine	MEK	UR 25ml
Methyl Isobutylketone (MIBK) exposure:			
Methylisobutylketone (MIBK)	urine	MIBK	UR 25ml
Methyl-n-butylketone(MBK) exposure:			
2,5 Hexanedione	urine	MBK	UR 25ml
Monobromomethane (Methylbromide) exposure:			
Bromide	serum	BR	Plastic tube 1
Parathion	blood	CHEWB	EDTA 1
Pesticide/Insecticide exposure:			
Organophosphates, Carbamates			
ChE Whole blood	blood	CHEWB	EDTA 1
WB ChE/Hb Ratio	blood	CHEWB + HB	EDTA2
Pseudocholinesterase – serum (ChE)	serum	CHS	SST 1

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CHEMICAL-METABOLITE	SPECIMEN	MNEMONIC	CONTAINERS
Pentachlorophenol exposure:			
Pentachlorophenol	urine	PCPE	UR 25ml
Phenol exposure:			
Phenol	urine	PHENOL	UR 25ml
Polycyclic aromatic hydrocarbon exposure (Coal tar exposure):			
1-Hydroxypyrene	urine	PAH	UR 25ml
Styrene exposure:			
Styrene	Blood	STYRB	EDTA 1
Mandelic acid & Phenylglyoxylic acid	urine	STYRU	UR 25ml
Tetrachlorethylene(perchloroethylene) exposure:			
Trichloracetic acid	urine	TETRA	UR 25ml
Perchlorethylene	blood	TETRAB	EDTA 1
Thiocyanate exposure:			
Thiocyanate	urine	THIOU	UR 50ml
Trichloroethylene exposure:			
Total trichloro compounds	urine	TRIC	UR 25ml
Trichloroacetic acid	urine	TRIC	UR 25ml
Trichloroethanol	urine	TRIC	UR 25ml
Trichlorethane exposure (Methyl chloroform):			
Trichloroacetic acid	urine	TRIMCU	UR 25ml
Trichlorethane & Trichloroethanol	blood	TRIMCB	EDTA 2
Trinitrotoluene Exposure			
Total Amino-dinitrotoluene	urine	TNTE	UR 25ml
Toluenediisocyanate exposure:			
Isocyanates-Toluenediamine	urine	TDI	UR 25ml
TDI(toluene diisocyanate)IgE	serum	ISOT	SST1
Toluene exposure(Hippuric acid & O-Cresol):	**urine**	**TOLCH**	**UR 25ml**
Hippuric acid	urine	TOLH	UR 25ml
Ortho cresol	urine	TOLC	UR 25ml
Toluene	blood	TOLB	EDTA 1
Xylene exposure:			
Methylhippuric acid	urine	XYL	UR 25ml
Xylene	blood	XYLB	EDTA 1

METAL EXPOSURE PROFILES

METAL	SPECIMEN	MNEMONIC	CONTAINER
Aluminum	urine	ALU	UR 25ml
	serum	AL	SST 1
Antimony	urine	SBU	UR 25ml
Arsenic	urine	ASU	UR 25ml
	blood	ASB	EDTA 1
Bromide	blood	BR	SST 1
Cadmium	urine	CDU	UR 25ml
	blood	CD	Heparin 1
Chromium	urine	CHU	UR 25ml
	blood	CRB	EDTA 1
Cobalt	urine	COU	UR 25ml
	blood	CO	EDTA 1
Copper	urine	CUEX	UR 25ml
	blood	CU	SST 1
Fluoride random	urine	FLU	UR 25ml
Fluoride pre shift	urine	FLPREU	UR 25ml
Fluoride post shift	urine	FLPOSTU	UR 25ml
Lead	urine	PBU	UR 25ml
	blood	PBB	EDTA 1
Manganese	urine	MNU	UR 25ml
	blood	MNB	EDTA 1
Mercury	blood	HGB	EDTA 1
	urine	HGU	UR 25ml
Molybdenum	urine	MOU	UR 25ml
Nickel	urine	NIU	UR 25ml
	blood	NI	EDTA 1
Platinum	urine	PTU	UR 25ml
	blood	PTB	EDTA 1
Selenium	urine	SEU	UR 25ml
	serum	SE	SST 1
Thallium	urine	TLU	UR 25ml
Titanium	urine	TIU	UR 25ml
	blood	TI	K02
Vanadium	urine	VU	UR 25ml
Zinc	urine	ZNU	UR 25ml
	plasma	ZN	Heparin 1
Heavy Metal profile Arsenic, Mercury, Cadmium, Cobalt, Lead ,Chromium	urine	HMPIND	UR 25ml

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BIOLOGICAL EFFECT MONITORING

CHEMICAL-METABOLITE	SPECIMEN	MNEMONIC	CONTAINERS
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
Blood count & PLT	blood	FBC	EDTA 1
Hemoglobin	blood	HB	EDTA 1
Urea & Elctrolytes	serum	UE	SST 1
Dipstix	urine	URCHEM	UR 25ml

FOOD HANDLERS SCREENING

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

TEST INTERPRETATION

GENERAL NOTES

1. The reference ranges provided are applicable to adults only.
2. The reference ranges provided may vary according to instrument and methodology.
3. 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 (TCO2)	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	1.7 - 8.3	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, immobilization
Urate	M 0.21 - 0.43 F 0.16 - 0.36	mmol/l	↑ Gout, renal failure, insulin resistance syndrome, alcoholism, malignancies (e.g. leukemia, lymphoma, multiple myeloma), psoriasis, drugs (e.g. diuretics, salicylates, etc) ↓ Syndrome of inappropriate ADH secretion, pregnancy
eGFR	>60	mL/min/ 1.73m^2	↓ eGFR = estimated (calculated) Glomerular Filtration Rate: Renal impairment

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	5 - 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.
Alanine amino Transferase (ALT)	M < 50 F < 35	U/l	↑ Acute hepatitis, chronic hepatitis, liver cirrhosis, liver cell necrosis (e.g. hypoxic shock, paracetamol overdosage), 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 anemia, megaloblastic anemia, rhabdomyolysis, vigorous exercise, muscular dystrophy.
Lactate dehydrogenase (LD)	100 - 250	U/l	↑ Megaloblastic anemia, haemolytic anemia, leukemia, 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, thalassamia ↓ Anaemia
	Inland: F 4.13 - 5.67 Sea level: F 3.80 - 4.80	10^{12}/L	
Haematocrit	Inland: M 43.0 - 55.0Sea 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 defiency). Round macrocytes are associated with liver disease, hypothyroidism, antiretroviral therapy, alcohol, chemotherapy, reticulocytosis and myelodysplasia. ↓ **Microcytic** red cells Iron deficiency, thalassaemia, other haemoglobin defects, anaemia of chronic disease, lead poisoning, sideroblastic anemia.
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, Bilirubinaemia, auto-agglutination, lipaemic Sample.

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Red cell distribution width (RDW)	12.0 - 14.5	%	If raised it means there are red cells of different sizes. Often the earliest sign of iron deficiency.
Platelet count	150 - 450	10^9/L	↑ **Thrombocytosis:** Reactive causes should firstly be excluded e.g. iron deficiency, trauma, infection, and malignancy. If no reason is found and platelets remain increased, a chronic myeloproliferative disorder should be excluded. ↓ **Thrombocytopenia:** Production defect - bone marrow infiltration / failure or peripheral loss mechanism e.g. immune (ITP), hypersplenism, DIC, TTP, etc.
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 e.g. viral infections, bordetella pertussis, stress lympocytosis (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 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.

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) .SANAS is recognized by the South African Government as the single National Accreditation Body that gives formal recognition 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 are followed for all testing performed with-in 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: Patient 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

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

- **Staff**
  All staff is deemed competent prior to performing any analysis. The training comprise of theoretical as well as practical training. Theoretical training is achieved through lectures that the trainer facilitates either in a classroom situation or 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
  - 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-also called "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.
- Turn Around 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.

EXPOSURE PROFILES

DESIGNING EXPOSURE PROFILES

Chemical and metal 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 effect monitoring of each exposure. Below general references and information used to generate these profiles found in this medical surveillance guideline which can assist in compiling your own exposure profiles.

ABBREVIATIONS

MSDS	Material Safety Data Sheet
IUPAC	International Union of Pure and Applied Chemistry
CAS	Chemical Abstracts Service
NIOSH	National Institute of Occupational Safety and Health
ACGIH	Association Advancing Occupational and Environmental Health

CDC	Centre for Disease Control
OSHA	Occupational Safety and Health Administration
ILO	International Labour Organization
BEI	Biological Exposure Index
OHSAct	Occupational Health and Safety act (85 of 1993)
STEL	Short term exposure limit

Topic	Reference & Information
Chemical Formula	MSDS/NIOSH
Synonyms	MSDS/NIOSH/ACGIH
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
Description	MSDS/NIOSH/ACGIH
Uses	NIOSH/ ACGIH/published literature
Routes of Exposure	NIOSH/ ACGIH/MSDS/ CDC /published literature
Metabolism (Includes absorption & elimination)	ILO/NIOSH/CDC/OSHA/ published literature
Effects of Occupational Exposure	ILO/NIOSH/CDC/OSHA published literature
Confounders/Non Occupational Exposure	Genetic, lifestyle and non-occupational reasons for exposure must be fully explored prior to surveillance program starting ILO/NIOSH/CDC/OSHA /published literature

OCCUPATIONAL EXPOSURE

Medical Surveillance	**Physical**	As per Occupational Medical Program-indicate: • Baseline and periodic testing • Frequency of testing • Medical questionnaire and exam based on exposure
	Sentinel health events	Effect monitoring based on health events/effects
Biological Monitoring	Exposure specific metabolites to be tested as well as BEI per exposure used as per OH Act. If not specified in act, company should decide on BEI to be used. Ampath then provide a BEI from the following references from other countries: • USA • Lauwerys R.R. et al.Industrial Chemical Exposure. Guidelines for Biological monitoring • NIOSH/ACGIH • 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 workplace	
Reproductively	ILO/CDC	
Toxicity	Linked to short term effects (STEL) and post “toxic exposure” ACGIH STEL limits.	

WORK RELATED HAZARDOUS CHEMICAL EXPOSURE

OCCUPATIONS AND OCCUPATIONAL GROUPS	HAZARDOUS CHEMICAL 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]; methylethylketone
Cement industry including laying	Asbestos, hexavalent chromium, thallium
Founding	Silica, asbestos [furnace], lead, zinc, chromium, nickel, manganese, beryllium; copper; cobalt (brass); cadmium; vanadium, cyanide, tinsulphur 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/solders	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,
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; etholamine
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]

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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 resturant	Disinfectants, pesticides
Education and training	Organic solvents, pigments, dyes, metals, plastics, minerals - arts & crafts; formaldehyde - chemistry & biology; asbestos, lead, pesticides

CHEMICAL EXPOSURES: DNA ADDUCTS

The evidence that chemicals can induce cancer in humans has been accumulating for more than two centuries (eg. Skin cancer found in polyaromatic hydrocarbons [PAH] exposure, the case of chimney sweeps and Dr Pott is an n notable example). Cancer can be caused in several ways such as by physical (e.g. radiation), chemical (eg. carcinogenic chemicals), and biological agents like viruses.

Chemical carcinogens are chemicals that cause tumour formation. In general, these genotoxic, meaning that they are chemical agents with an intrinsic chemical characteristic allowing its interaction with cellular deoxyribonucleic acid (DNA) causing alterations of the DNA structure. The genotoxic process includes both direct and indirect effects in DNA. These are:

- the induction of mutations (gene, chromosomal, genomial, recombinational) that at the molecular level are similar to events known to be involved in carcinogenesis,
- indirect surrogate events associated with mutagenesis (e.g., unscheduled DNA synthesis (UDS) and sister chromatid exchange (SCE), or
- DNA damage (e.g., the formation of adducts), which may eventually lead to mutations. Figure 1 below schematically demonstrates the possible outcomes after DNA damage post exposure to chemical carcinogens.

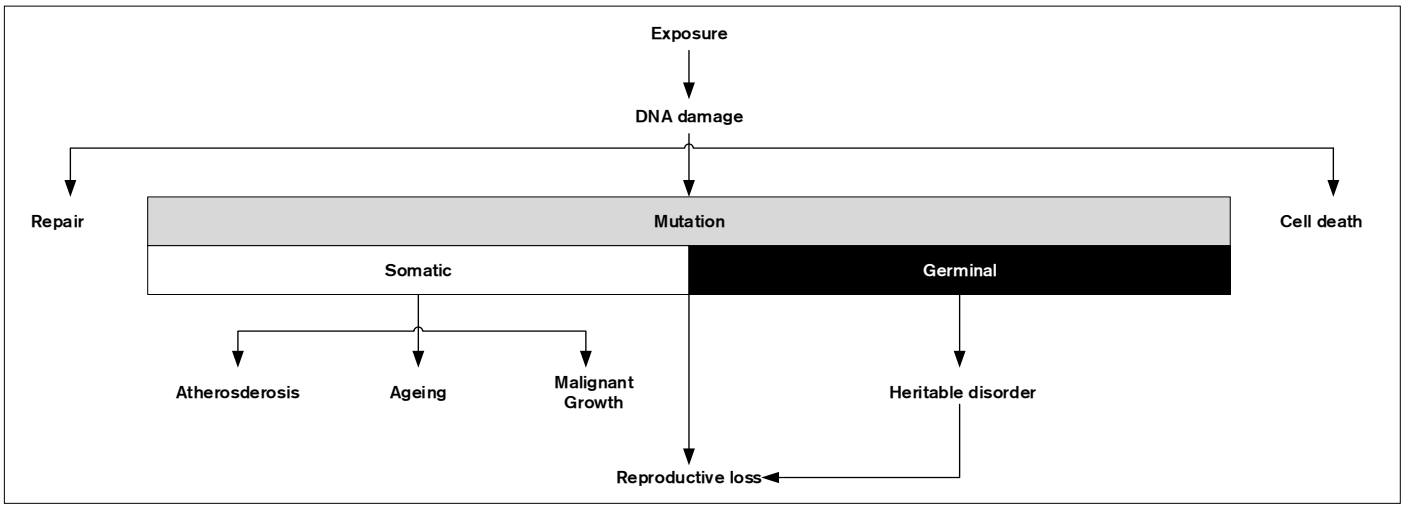

FIGURE 1: Possible outcomes after DNA damage post exposure to chemical carcinogens.

When a chemical binds to DNA, the DNA becomes damaged, and proper and complete replication cannot occur to make the normal intended cell. This could be the start of a mutation, or mutagenesis. Without effective DNA repair, which happens naturally under normal circumstances, this can lead to carcinogenesis and tumour formation. A central property of chemical carcinogenesis is that the covalent binding of carcinogens to DNA is causally related to tumorigenesis. This has been demonstrated by the following observations i.e.

- The majority of carcinogens are also mutagens;
- The mutagenic and carcinogenic properties of many carcinogens depend upon their conversion to electrophilic derivatives that react with nucleophilic sites within DNA;
- The extent of DNA adduct formation can often be correlated with the magnitude of mutagenic and carcinogenic responses; and
- The activation of certain proto-oncogenes can be accomplished through the interaction of carcinogens with DNA.

The role of DNA adducts in chemical carcinogenesis will be discussed here as it is one of the most widely researched and often used as a biomarker of exposure, but, more importantly, a marker of genotoxicity. Procarcinogens such as benzo[a]pyrene are chemically inert precursors that are metabolically converted into highly reactive carcinogens which react with DNA and form DNA adducts. It undergoes two sequential oxidation reactions mediated by cytochrome P450 enzymes, which results in benzo[a]pyrenediol epoxide (BPDE), the carcinogenic metabolite (Refer to figure 2 below) that is able to form a covalent DNA adduct. Note that benzo[a]pyrene and other potential chemical carcinogens also form non-reactive metabolites that are excreted in the urine.

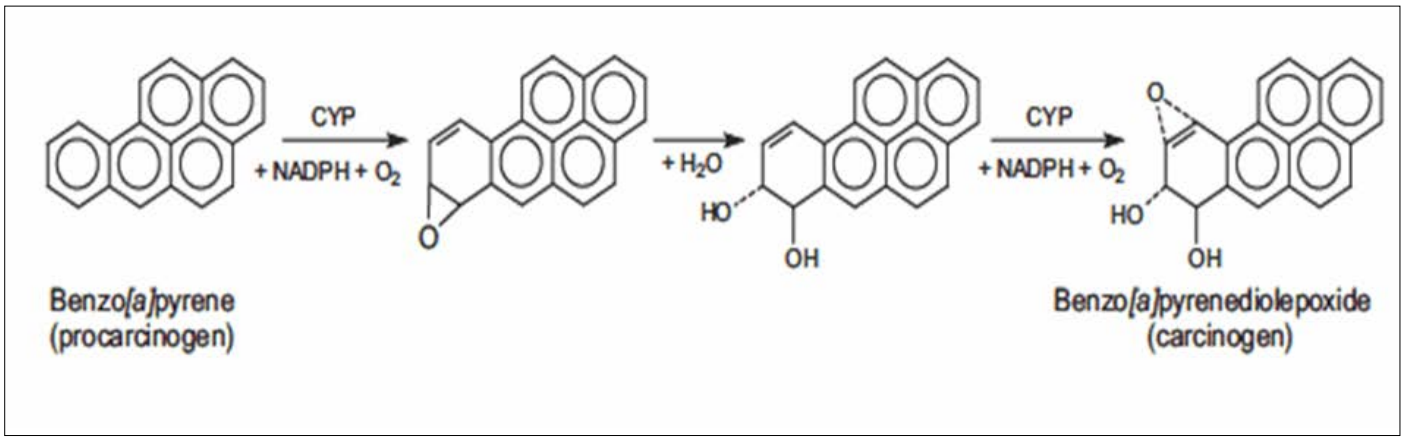

FIGURE 2: Benzo[a]pyrene is oxidized by P450 enzymes to create the highly carcinogenic BPDE.

Several techniques (e.g. immunoassay, fluorescence assay, gas chromatography-mass spectroscopy (GC-MS), 32P-postlabelling and mass spectrometry (MS) analysis) have been developed for the analysis of PAH derived DNA adducts. Blood samples are collected for direct measurement of the compound, for measurement of metabolites, or for determination of protein or DNA adducts (e.g., albumin or hemoglobin adducts, and DNA adducts in circulating lymphocytes). Accessible tissue cells, such as epithelial cells from the buccal area of the mouth, are also sampled for identification of DNA adducts.

Several studies have demonstrated that not all carcinogens show a linear relationship between exposure dose and DNA adduct concentrations. It is therefore advised that caution be exercised when interpreting and extrapolating causal relationships between chemical exposure and carcinogenesis.

Ampath suggests at least an ethics clearance for any such testing in the workplace, preferable a formal research study designed according to international research principles.

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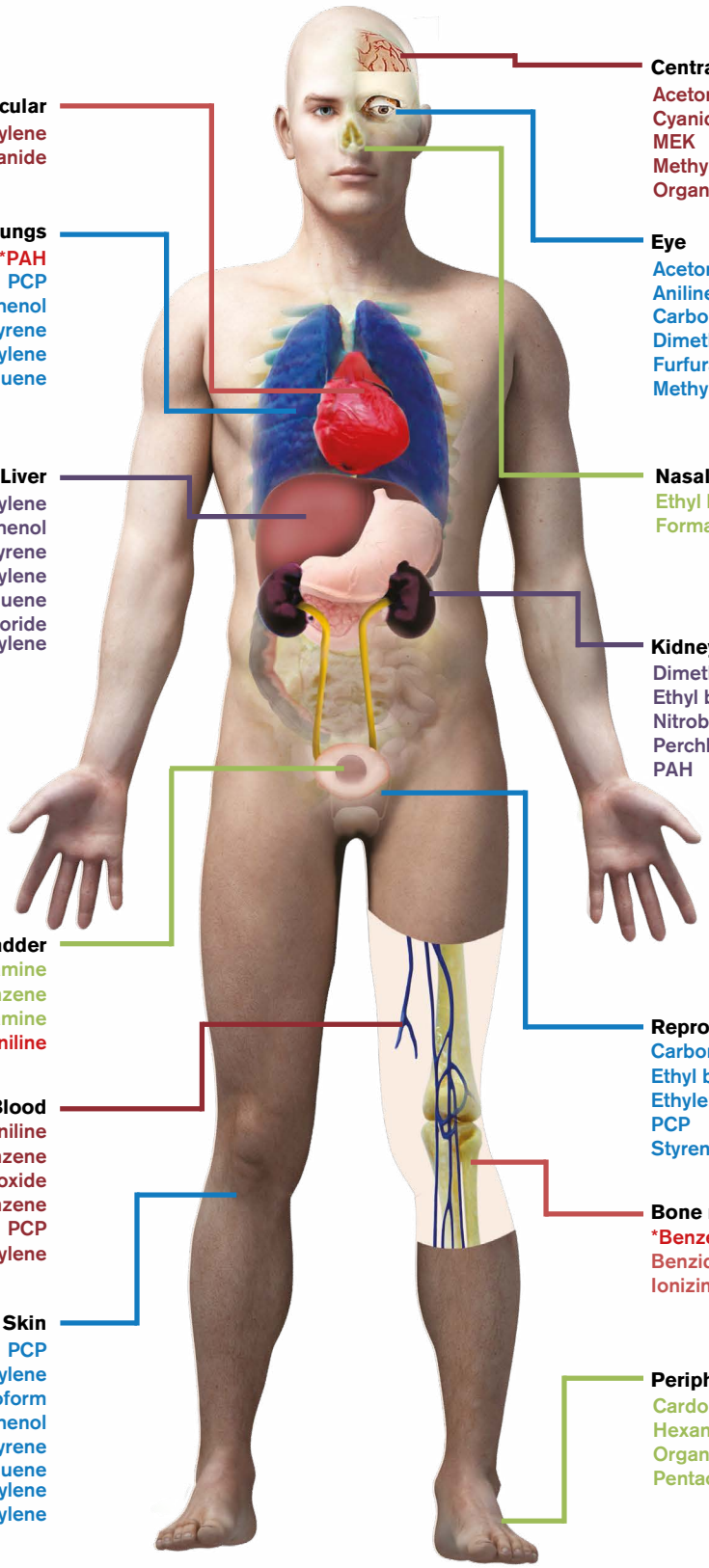
TARGET ORGANS FOR CHEMICAL EXPOSURE



Cardiovascular
Carbon monoxide
Carbon disulphide
Xylene
Cyanide

Lungs
Acetone
Aniline
Cyanide
Ethyl benzene
Furfural
MEK
Isocyanate
*PAH
PCP
Phenol
Styrene
Trichloroethylene
Toluene

Liver
Carbon tetrachlorine
Carbon disulphide
Dimethylformamide
MEK
MIBK
Nitrobenzene
Organophosphate
PCP
Perchloroethylene
Phenol
Styrene
Trichloroethylene
Toluene
Vinyl chloride
Xylene

Bladder
B Napthylamine
Dinitrobenzene
Acrylamine
*Aniline

Blood
Aniline
Benzene
Carbon monoxide
Nitrobenzene
PCP
Xylene

Skin
Acetone
Aniline
Benzene
PAH
Ethyl benzene
Furfural
MEK
MIBK
PAH
PCP
Perchloroethylene
Methyl chloroform
Phenol
Styrene
Toluene
Trichloroethylene
Xylene

Central nervous system
Acetone
Cyanide
MEK
Methyl chloroform
Organophosphate
Trichloroethylene
Styrene
Xylene
Nitrobenzene

Eye
Acetone
Aniline
Carbon disulphide
Dimethyl sulphate
Furfural
Methyl alcohol
Methanol
MEK
Toluene
Trichloroethylene
Xylene
Styrene

Nasal passages
Ethyl benzene
Formaldehyde

Kidneys
Dimethylformamide
Ethyl benzene
Nitrobenzene
Perchloroethylene
PAH
Trichloroethylene
Phenol
Xylene
Toluene

Reproductive system
Carbon disulphide
Ethyl benzene
Ethylene glycol
PCP
Styrene

Bone marrow
*Benzene
Benzidine
Ionizing radiation

Peripheral nervous system
Cardon disulphide
Hexane
Organophosphate
Pentachloroethane
Parathion
Toulene
Xylene

*** Carcinogens**

ANILINE

Chemical Formula	$C_6H_5NH_2$
Synonyms	Aminobenzene, aminophen, aniline oil, benzenamine, phenylamine, CI 76000, CI Oxidation Base, NCI-CO 3736, Kyanol, Anyvim, Blue Oil
CAS number	62–53–3
Description	Aniline is a yellow to brown, clear, oily liquid with a fishy odor.
Uses	Used as a chemical intermediate for the dye, agricultural, polymer and rubber industries. It is also used as a solvent and has been used as an antiknock compound for gasolines. It is also utilized in the production of: MDI – methylene diphenyl diisocyanate PMPPI – Polymeric MDI
Routes of Exposure	Exposure may be through inhalation, ingestion and skin contact
Metabolism (Includes absorption & elimination)	Aniline is rapidly absorbed through inhalation, ingestion and skin contact. Fifteen to sixty percent of inhaled aniline is oxidized to p-aminophenol, which is excreted by the kidney. Exposure to Aniline causes the formation of methaemoglobin resulting in a functional anemia. 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.
Effects 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 two to four hours post exposure depending on the exposure level Aniline is irritating to mucous membranes and affects the eyes,nose and respiratory tract. Data on long term or chronic exposure have shown insufficient evidence of effects on organs such as liver and CNS. Symptoms of bladder cancer such as blood in urine,lumps in groin area, painfull urination and lower back pain has been observed-suspected bladder carcinogen. Aniline as a potential carcinogen to humans of the bladder but there is no information on its teratogenicity or reproductive toxicity in humans.
Confounders/ Non Occupational Exposure	Burning of plastic and tobacco and smoking. Small amounts found in food-such as apples, beans, rhubarb, corn and grans

OCCUPATIONAL EXPOSURE

Medical Surveillance	**Medical and occupational history**	
	Physical	As per occupational medical program
	Sentinel health events	Irritation and inflammation of eye, skin, respiratory tract. Anemia and Cyanosis Symptoms of bladder cancer
Biological Monitoring	**Sample:** Total p-aminophenol in urine Methaemoglobin in blood	**Sampling time:** ES (end of shift) During or end of shift
	Reference limits: 1. Total p-aminophenol:creatinine Not industrial exposed BEI (Biological exposure index) 2. Methaemoglobin in blood Not industrial exposed BEI (Biological exposure index	 Not determined 50 mg/g creatinine > 1.5% of haemoglobin
Biological Effect Monitoring	Blood Urine Spirometry	Full blood count, urea, creatinine, methaemoglobin Dipstix Yes

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ACETONE

Chemical Formula	C_3H_6O
Synonyms	Dimethyl ketone, ketone propane, propanone, 2-propanone
CAS number	67–64–1
Description	Highly volatile, flammable solvent, colorless liquid with a pungent or fruity odor
Uses	It is primarily used as an industrial solvent and chemical intermediate. Acetone is also found in paints, varnishes and lacquers and is used as a solvent for cements in the leather and rubber industries. Also used in fabric coating and dyeing process
Routes of Exposure	Exposure may be through inhalation, ingestion, dermal, and eye contact (limited due to volatility of acetone). In the occupational setting, inhalation from vapor is the most common exposure route. Endogenously it is a by-product of fat metabolism
Metabolism (Includes absorption & elimination)	Acetone, because of its solubility in water, is readily absorbed into blood stream and thus is transported rapidly throughout 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 vapor results in an estimated retention of 45% to a level of up to 80% of the total. It is thought that these levels of retention may be lower in women. The half-life in alveolar air is about four hours, in venous blood six hours, and in arterial blood four hours. The highest concentration of acetone in urine is about the to three and a half hours after exposure and declines thereafter. Work load affects the mean levels of acetone in body fluids i.e. higher workloads leading to higher levels in body fluids. Acetone is rapidly cleared from the body by metabolism and excretion. Exhalation is the major route of elimination for acetone and its terminal metabolite (carbon dioxide), and the fraction of administered acetone that is exhaled as unchanged acetone is dose-related. Urinary excretion of acetone and its metabolites occurs but this route of elimination is minor
Effects of Occupational Exposure	Short-term exposure usually results in eye irritation, dryness of the mouth and throat, nausea and vomiting, headache, sleepiness, dizziness and light-headedness, and fainting. Repeated exposure causes dermal inflammation, inflammation of the gastrointestinal tract, and respiratory tract. 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 1000 ppm. No indications of toxicity were reported following exposures to 2100 ppm for eight hours/day
Confounders/ Non Occupational Exposure	Endogenously produced acetone levels in blood/urine do not exceed 2mg/L.

OCCUPATIONAL EXPOSURE

Medical Surveillance	**Medical and occupational history**	
	Physical	As per occupational medical program
	Sentinel health events	Irritation and inflammation of eye, skin, respiratory tract and intestine. Contact or allergic dermatitis, CNS
Biological Monitoring	**Sample:** Acetone in urine Acetone in blood	**Sampling time:** ES (end of shift)
	Reference limits: 1. Acetone: creatinine Not industrial exposed BEI (Biological exposure index) Acetone Exposure *Maximum permissible concentration Isopropanol Exposure *Maximum permissible concentration 2. Acetone in blood Not industrial exposed BEI (Biological exposure index)	 < 2.0 mg/g creatinine 30.0 mg/g creatinine 50.0 mg/g creatinine < 0.2 mg/100mL 5.0 mg/100mL
Biological Effect Monitoring	Spirometry	Yes

BENZENE

Chemical Formula	C_6H_6
Synonyms	Benzol, coal naphtha, phenyl hydride, pyrobenzol, mineral naphtha, pyrobenzole, carbon oil. Some of these synonyms represent impure forms of benzene. Note that benzine is often confused with benzene. Benzine is also a petroleum distillate but is a different chemical substance to benzene.
CAS number	71–43–2
Description	Colorless, highly flammable liquid that dissolves in water, and is highly soluble in oil and fat. It is a solvent and a ubiquitous environmental pollutant, extraction of oils from seeds and nuts, chemical intermediate, manufacturing of detergents, explosives, pharmaceuticals and dyestuffs.
Uses	As solvent and raw material for chemical synthesis, as impurity in chemical processes (petrochemicals, toluene, xylene, paints, varnishes, rubber cements and lacquers. Has in the past been used in the rubber and leather industries
Metabolism (Includes absorption & elimination)	Route of entry: Most commonly by inhalation (vapors), dermal, ingestion. In liquid form, absorption through the skin is also significant. About a third of benzene retained in the body is excreted in urine as conjugated phenol and dihydroxyphenols. The remainder is either absorbed in tissue(fat) or exhaled as CO_2. In the OH setting, dermal absorption contributes to 20–40% of total dose of benzene absorbed.
Effects of Occupational Exposure	High concentrations by inhalation or oral ingestion lead to central nervous system depression and death. Long term occupational exposures lead to bone marrow depression (thrombocytopenia, anemia, granulocytopenia and aplastic anemia). Leukemia of the myelogenous variety is most common. Benzene is a known carcinogen (IARC category 1) and suspected human reproductive agent. Chronic exposure can manifest with renal tubular dysfunction, hepatocellular damage, and neurotoxicity (personality and mood changes, memory loss)
Confounders/ Non Occupational Exposure	Benzene is used in lacquers and paint removers prior to 1976. Benzene may be present in gasoline in varying percentages in some countries. Benzene may be found in exhaled air of people who use household products containing benzene. Phenol is a metabolic product of proteins, so background levels are affected by diet. Phenol is not a specific indicator of exposure to benzene and increases can be caused by exposure to phenol (inhalation or skin), if contact is made with disinfectants, germicidal paints, slimicides, drugs, mouthwash, soap. Smoking and toluene exposure increases benzene values. Co-exposure to toluene will decrease tt Muconic acid values. Tobacco smoke contains benzene and can cause an elevated background value of PMA.

OCCUPATIONAL EXPOSURE

Medical Surveillance	**Medical and occupational history**	
	Physical	As per occupational medical program
	Sentinel health events	Acute myeloid leukemia, dermatitis, decrease or absence of white cells (peripheral or bone marrow) and red cell cancers
Biological Monitoring	**Sample:** Phenol in urine (total)(generally not recommended) (t,t) Muconic acid in urine Phenylmercapturic acid in urine(highly specific) Benzene in blood(since unmetabolised higher in after shift workers)	**Sampling time:** ES (end of shift)
	Reference limits: 1. Phenol:creatinine Not industrial exposed BEI (Biological exposure index) 2. tt Muconic acid:creatinine Not industrial exposed BEI (Biological exposure index 3. Phenylmercapturic acid:creatinine Not industrial exposed BEI (Biological exposure index) 4. Benzene in blood Not industrial exposed Biological monitoring for industrial exposure	 < 20 mg/g creatinine 50 mg/g creatinine < 0.50 mg/g creatinine 1.50 mg/g creatinine < 15 ug/g creatinine 45 ug/g creatinine < 0.045 ug/100 mL *3.8 ug/l00 mL at end of shift at TLV 4.0 ppm
Biological Effect Monitoring	Blood Urine Spirometry Chest X ray	Full blood count & differential counts, ALT, AST, ALP, GGT, Urea, creatinine Dipstix Yes Yes

CARBON DISULPHIDE

Chemical Formula	CS_2
Synonyms	Carbon Disulphide, Carbon bisulphide, dithiocarbonic, anhydride, carbon sulphide, sulfocarbonic anhydride, Weedtox
CAS number	75–15–0
Description	Pure carbon disulphide is a colorless liquid, evaporates rapidly at room temperature and is flammable.
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.
Metabolism (Includes absorption & elimination)	Absorbed chiefly through the lungs, entering the blood stream and being distributed through the body. 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 vapor during the first 15 minutes, but the proportion falls to about 40% after 45 minutes and remains that level for some time. In workers previously exposed, about 55% of inhaled vapor is absorbed during the first 15 minutes. Excretion through the lungs and urine is small, about 92% retained in the tissues and metabolized.
Effects 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, anemia, dermatitis, coronary artery disease (increases blood cholesterol levels). (High blood pressure), spasmatogenic effect, menstrual disorders and spontaneous abortions, depression and suicidal tendencies.
Confounders/ Non Occupational Exposure	Exposure to substance that are metabolized to carbon disulfide pesticide (Captan), disulfiram, some rubber accelerators and dithio carbonates (Thiram) may result in carbon disulfide and it metabolites in urine.

OCCUPATIONAL EXPOSURE

Medical Surveillance	**Medical and occupational history**	
	Physical	As per occupational medical program
	Sentinel health events	Cardiovascular, renal Neurological/psychiatric Liver and GI Reproductive Vision
Biological Monitoring	**Sample:** 2-thiothiazolidine – 4 carboxylic acid (TTCA) in urine	**Sampling time:** ES (end of shift)
	Reference limits: Not industrial exposed BEI (Biological exposure index)	 < 1 mg/g creatinine 5 mg/g creatinine
Biological Effect Monitoring	Blood Urine Spirometry Chest X-Rays ECG	Urea, creatinine, electrolytes, ALT, AST, ALP, GGT, lipogram Dipstix - Yes Yes
Reproductive/ Developmental Effects	May result in menstrual disorders, spontaneous abortion, and low sperm count	
Toxicity	Symptoms of moderate to severe intoxication have appeared in individuals chronically exposed to vapor concentrations averaging slightly in excess of 20 ppm. Concentration of 300 ppm or more can cause serious pathological changes after only a few days. Blood concentrations of 0,10-0.7 mg/L were observed during exposure to air concentrations of 80 ppm. The half-life (T1/2) for disappearance of the substance from blood is estimated at less than one hour. (Davidson md Freinleib, 1972)	

CARBON MONOXIDE

Chemical Formula	CO
Synonyms	Carbonic oxide, carbon oxide, exhaust gas, flue gas
CAS number	630–08–0
Description	Carbon monoxide is a colourless, odourless gas produced by partial oxidation of hydrocarbon gases from natural gas or by gasification of coal and coke
Uses	Liberation from emissions in enclosed places from exhaust fumes of internal combustion engines; from metallurgic industry and foundries; from chemical industry for synthesis and emission as result of incomplete combustion; liberation during acetylene welding; from enclosed areas as mines or tunnels; from fire-damp explosions; liberation from industrial heating
Metabolism (Includes absorption & elimination)	Route of entry: inhalation Absorbed and excreted unchanged via lungs. CO dissolved in blood is < 1%. Mode of action: forms carboxyhaemoglobin in blood and thus tissue anoxia. Excreted through the lung. It is a non-cumulative toxin.
Effects of Occupational Exposure	Headache, nausea, dizziness, weakness, rapid breathing, unconsciousness and death (if in excess of 3500 ppm). No cyanosis, usually a pink colour due to presence of carboxyhaemoglobin. Signs and symptoms: carboxhyhaemoglobin below 10%: none Electrocardiogram may show sinus tachycardia and ST segment and T wave abnormalities. Electroencephalogram may show focal and diffuse epiltiform discharges, which later disappear. Exposure to CO can aggravate heart disease and arterial disease and lead to chest pain in pre-existing cardiac disease
Confounders/ Non Occupational Exposure	CO formed endogenously from catabolism of haemoglobin and other proteins. Increase in pregnancy and haemolytic anaemia, both in blood and exhaled air. CO is ubiquitous in the environment (internal combustion, engines, heaters, furnaces). Commuting via congested road can increase COHb levels to 5% or more and CO in exhaled air to > 30 ppm. CO is a metabolite and methylene chloride, a solvent and paint stripper in common home use. Tobacco smoking can increase COHb to 10–15% and CO in exhaled air to > 50 ppm. The effects of CO are aggravated by heavy work, high altitude, and temperature.

OCCUPATIONAL EXPOSURE

Medical Surveillance	**Medical and occupational history**	
	Physical	As per occupational medical program
	Sentinel health events	Cardiovascular related disease, Pulmonary Disease, Blood cell changes
Biological Monitoring	**Sample:** Carboxy Hb in blood* * Non smokers	**Sampling time:** ES (end of shift)
	Reference range: < 8% of Hb	
Biological Effect Monitoring	Blood Urine Spirometry Chest X-Rays ECG	Full Blood count and differential, carboxyhaemoglobin levels (when over-exposure is suspected) - Yes Full Blood count and differential, carboxyhaemoglobin levels (when over-exposure is suspected) unless for pre-existing condition
Toxicity	It has been shown in number of studies that COHb concentrations of 10% or less adversely affect a person's ability to perform complex tasks as well as strenuous manual labour. (Cobum, et al, 1977) A biological action level of 5% has been proposed Exposure of CO concentrations greater than 50 000 ppm can cause cardiac arrhythmia or death prior to significant elevation of carboxyhaemoglobin (Beard, 1982)	

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CYANIDE

Chemical Formula	CN-(cyano functional group)
Synonyms	Hydrogen cyanide(HCN),hydrocyanic acid,Sodium cyanide(NaCN),Potassium cyanide(KCN)
CAS number	74–90–8
Description	Cyanides are fast acting poisons that can be lethal. Hydrogen cyanide is a pale blue or colorless liquid at room temperature and a colorless gas at higher temperature. It has a bitter almond-like odor. Sodium and potassium cyanide are white powders which may have a bitter almond-like odor.
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 waste water treatment can create cyanides.
Metabolism (Includes absorption & elimination)	Route of entry: 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 metabolized to thiocyanate, which is less harmful and excreted in urine. In large doses the body's ability to convert cyanide to thiocyanate is overwhelmed – cyanide combines with the ferric ion, preventing electron transport in the cytochrome system, bringing ATP production and oxidative metabolism to a halt, effecting the heart and CNS.
Effects of Occupational Exposure	Acute effect: Cyanide is extremely toxic and high exposure (100 mg/m^3) can cause death. Exposure to lower concentrations (6–49 mg/m^3) will cause weakness, headache, nausea, increase rate of respiration and eye and skin irritation. Long term exposure: Primarily effects the Central Nervous System. Cardiovascular and respiratory effects are also noted.
Confounders/ Non Occupational Exposure	Cyanide containing substances occur in numerous fruits, seeds, roots and pits such as almonds, peaches, plums, sorgum, spinach, soybeans, sweet potatoes, maize, millet and bamboo shoots. Sources of cyanide include exhaust fumes and cigarette smoking.

OCCUPATIONAL EXPOSURE

Medical Surveillance	**Medical and occupational history**	
	Physical	As per occupational medical program
	Sentinel health events	Central Nervous System, Respiratory, Cardiovascular
Biological Monitoring	**Sample:** Urine Thiocyanate Serum Thiocyanate Blood Cyanide	**Sampling time:** ES (end of shift) ES (end of shift) After acute exposure
	Reference range: 1. Not industrially exposed 2. Not industrially exposed 3. Not industrially exposed 4. Exposed to cyanide Toxic:	 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 Non-smokers < 16 ug/L Smokers < 41 ug/L Usually asymptomatic < 200 ug/L Conscious, flushed + tachycardia 500–1000 ug/L Stupor + agitation 1000–2500 ug/L Coma + potentially lethal > 2500 ug/L
	Blood cyanide concentrations are used primarily in the diagnosis of acute intoxication eg. after inhalation of cyanide or acute cyanide intake	
Biological Effect Monitoring	Blood Urine CNS ECG	U&E Urinalysis Parkinson-like syndromes Ischemic damage
Reproductive/ Developmental Effects	No carcinogenic effects or developmental effects noted	

ETHYL BENZENE

Chemical Formula	$C_6H_5C_2H_5/C_8H_{10}$
Synonyms	Ethylbenzol, EB, Phenylethene, Phenylethane
CAS number	100–41–4
Description	Colorless liquid with a distinctive aromatic odor detectable at 2–20 ppm. Natural constituent crude of petroleum. It is found in gasoline and diesel fumes.
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 also as a solvent and raw material for production of cellulose acetate, acetophenone, diethyl benzene and anthraquiones. Ethyl benzene is a minor component of gasoline and aviation fuels. Electroplating aluminium
Metabolism (Includes absorption & elimination)	Route of entry: inhalation, dermal and ingestion route. Most ethyl benzene is metabolized by mandelic acid within a day. Temporary storage may occur in fatty tissues.
Effects of Occupational Exposure	Acute: vapor or mist can irritate nose and throat. Inhales ethyl benzene may cause nausea, headache, vomiting and other symptoms of central nervous system depression. Human volunteers exposed at 85 ppm for eight hours reported no adverse health effects; at level of 100 ppm mild vertigo sleepiness and headache were reported. Exposure to 1000–2000 ppm for six 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. Ingestion: no human information available. Chronic exposure: Little evidence that of chronic low level toxicity to humans. Long term exposure may cause kidney, blood and testicular effects. Similarly to other hydrocarbons ethyl benzene vapor 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 defattening action.
Confounders/ Non Occupational Exposure	Non occupational exposure to ethyl benzene is unlikely. Occasionally ethyl benzene is a constituent of aromatic solvents used in household products such as paint thinners and motor cleaners. Combined exposure to ethylbenzene and xylene decrease the levels of metabolites and delays excretion of metabolites. As mandelic acid is also a metabolite for styrene it may be necessary to confirm exposure of ethylbenzene by determining concentrations in blood.

OCCUPATIONAL EXPOSURE

Medical Surveillance	**Medical and occupational history**	
	Physical	As per occupational medical program
	Sentinel health events	Skin, respiratoryeffects, kidney, blood and testicular
Biological Monitoring	**Sample:** Mandelic acid in urine Phenylglyoxylic acid in urine Ethyl benzene in blood	**Sampling time:** ES (end of shift) EW(end of work-week) ES (end of shift) EW(end of work-week) DS (during shift)
	Reference limits: 1. Mandelic acid Not industrial exposed: creatinine BEI (Biological Exposure Index) : 2. Phenylglyoxylic acid: creatinine + Mandelic acid: creatinine Not industrial exposed BEI (Biological Exposure Index) 3. Ethyl benzene blood Not industrial exposed BEI (Biological exposure index)	 <29 mg/g creatinine 1500 mg/g creatinine Not detected 1800 mg/g creatinine <0.2 ug/100mL 150 ug/100mL
Biological Effect Monitoring	Blood Urine Spirometry Chest X rays	Liver function tests Dipstix Yes Discretionary,based on spirometry
Toxicity	Ethylbenzene is classified as a Group 2B carcinogen by IARC (renal, lung, nasal and oral cavities)	

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NN-DIMETHYLFORMAMIDE

Chemical Formula	HCON $(CH_3)_2$
Synonyms	Dimethylformamide, N-formyldimethyl-amine, DMF, DMFA, N,N-dimethylmethanamide
CAS number	68–12–2
Description	Colorless to slight yellowish liquid with a fishy and unpleasant odor which is soluble in water. Odor easily detected below OEL level.
Uses	Solvents for liquids and gasses, including those used in artificial leather production. It is also used in the synthesis or organic compounds, manufacture of pol-yacrylic fibres, butadiene, pharmaceuticals, dyes petroleum products and other organic chemicals. Also used in adhesives, pesticides, epoxy formulations.
Metabolism (Includes absorption & elimination)	Route of entry: inhalation, skin absorption and ingestion. The following metabolites of DMF are excreted in urine: N-methyl-N-hydroxymethylformamide (DMF-OH or, alternatively, HMMF), N-monothylformamide, formamide, mercapturic acid. Elimination's through urine is biphasic with half-lives of three hours and seven hours respectively. Monophasic elimination with a four hour half-life has also been reported.
Effects of Occupational Exposure	Acute: inhalation of vapor 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 illness. (Porphyric symptomatology)
Confounders/ Non Occupational Exposure	Non-occupation exposure is uncommon. Some household products, such as paint remover, printing ink and shoe polish, may contain N,N-dimethylformamide. Exposure results in alcohol intolerance.

OCCUPATIONAL EXPOSURE

Medical Surveillance	**Medical and occupational history**	
	Physical	As per occupational medical program
	Sentinel health events	Liver, Kidney, CVS and porphyric disease
Biological Monitoring	**Sample:** N-Methylformamide	**Sampling time:** ES (end of shift)
	Reference limits: Not industrial exposed BEI (Biological Exposure Index)	 < 0.7 mg/g creatinine 30.0 mg/g creatinine
Biological Effect Monitoring	Blood Urine Spirometry Chest X-Rays ECG	ALT, AST, GGT, ALP, Full blood count Dipstix Yes Yes Yes
Reproductive/ Developmental Effects	Inconclusive evidence	

FURFURAL

Chemical Formula	$C_5H_4O_2$
Synonyms	2-furancarboxaldehyde, 2-furancarbonal, 2-furaldehyde, artificial ant oil, fu-role, pyromucic aldehyde, 2-furyl-methanal, fural, furfurol
CAS number	98–01–1
Description	Fufural is a colorless oily liquid when pure, darkens in light and air. Very soluble in alcohol, ether and benzene. Slightly soluble in water. Pungent almond odor .
Uses	Solvents for oils, synthetic and natural resins, cellulose and derivatives, dyes, polymers and other organic chemicals, intermediates in the production of plastics and insecticides/pesticides, vulcanisation accelerators in the rubber industry.
Metabolism (Includes absorption & elimination)	Furfural is absorbed through the skin, and through inhalation. 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 on about two hours. Furfural is metabolised such that approximately 97% (range, 93–100%) is oxidized 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.
Effects of Occupational Exposure	Exposure is usually to furfural vapors, 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 vapors are strong skin, eye and mucous membrane irritants, and can lead to sensitization and pulmonary oedema. Chronic expo-sure 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 sensitization 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. Dermatitis is caused by skin sensitization with chronic exposure.
Confounders/ Non Occupational Exposure	Significant non-occupational exposure is unlikely, but furoic acid is a natural constituent of human urine with concentrations varying between 4 and 65 mg/g of creatinine, depending on diet. (Furfural is a herbicide and traces can be found in some cereal bran's). It is also present in some essential oils. Mean value in urine tends to be higher in coffee drinkers.

OCCUPATIONAL EXPOSURE

Medical Surveillance	**Medical and occupational history**	
	Physical	As per occupational medical program
	Sentinel health events	Skin, eyes and upper respiratory sensitization
Biological Monitoring	**Sample:** Total furoic acid in urine	**Sampling time:** ES (end of shift)
	Reference limits: Not industrial exposed (BEI)Biological Exposure Index	 <65 mg/g creatinine 200 mg/g creatinine (ES)
Biological Effect Monitoring	Blood Urine Spirometry Chest X-Rays	Urea, creatinine, ALT, AST, GGT, ALP Dipstix Yes Yes
Reproductive/ Developmental Effects	No evidence available	

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N-HEXANE

Chemical Formula	$CH_3 (CH_2)_4 CH_3$
Synonyms	Hexane, Hexyl hydride, n-hexane, Gettysolve-B
CAS number	110–54–3
Description	Hexane is a colorless volatile liquid used as an industrial solvent. It is insoluble in water, but very soluble in ethanol.
Uses	It is a solvent used in the chemical and food industries. Solvent in glues, cements and adhesives for production of footwear and furniture, car tyre retreads and in the extraction of vegetable oils. Common in paints and thinners, as well as being a component of petroleum and petroleum distillates (like solvents and grease removers). Food additive
Metabolism (Includes absorption & elimination)	Hexane is taken up mainly through the lungs. 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.
Effects 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.
Confounders/ Non Occupational Exposure	Non-occupational exposure to n-hexane is uncommon. n-Hexane may be a component of inks, paints, glues and gasoline, products which are sometimes abused to achieve "highs" by inhalation. 2,5-hexanedione is produced by endogenous metabolism with a mean concentration in urine of the general population of 0.45 mg/L, and 0,11 mg/L (German/Japan) and 2,5 hexanedi-one formation is increased by co-exposure to toluene and methyl ethyl ketone.

OCCUPATIONAL EXPOSURE

Medical Surveillance	**Medical and occupational history**	
	Physical	Annual – specific attention to peripheral nervous system
	Sentinel health events	Peripheral neuropathy
	Special investigations	Measure of peripheral neuropathy e.g. EM (electromyography, etc may be of assistance)
Biological Monitoring	**Sample:** 2.5 Hexandione	**Sampling time:** ES (end of shift)
	Reference limits: Not industrial exposed BEI (Biological Exposure Index)	 < 0.36 mg/g creatinine 5.0 mg/g creatinine at end of shift (OHS act) 4.0 mg/g creatinine at end of shift,end of work-week (Lauwerys, Hoet)
Biological Effect Monitoring	Blood Urine Spirometry Chest X-Rays Nerve conduction studies	Confirmation of exposure – n-hexane in blood, full blood count Dipstix, creatinine - Yes Yes
Reproductive/ Developmental Effects		

ISOCYANATES

Chemical Formula	Compounds containing isocyanate group (-NCO)
Synonyms	Toluene diisocyanate(TDI), 4,4-diphenyl methane diisocyanate (MDI), 1,6-hexamethylene diisocyanate (HDI)
CAS number	TDI: Toluene-2,6-diisocyanate/$C_9H_6N_2O_2$ - 584–84–9 Toluene-2,4-diisocyanate/C9H6N2O2 - 91–08–7 MDI: 4, 4-diphenyl methane diisocyanate /$CH_2(C_6H_4NCO)_2$-101–68–8 HDI: 1,6-hexamethylene diisocyanate/$OCN(CH_2)6NCO$ - 822–06–0
Description	Colorless to pale yellow liquid with sharp, pungent odor
Uses	The most commonly used when react with the hydroxyl groups of compounds containing alcohol 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, ad-hesives, and sealants.
Routes of exposure	Exposure is via inhalation through vapor and mists, liquid and particles via eye and skin contact. Diisocyanates can also be ingested. Because of its inherent toxicity, higher volatility, and widespread use, TDI presents a great hazard and exposure can occur at all stages of its manufacture and use.
Metabolism (Includes absorption &elimination)	Inhaled isocyanates are excreted as an amine. In plasma amines undergo a two-phase elimination pattern. The urinary elimination after inhalation is rapid, > 80% is excreted in the urine within six hours after termination of exposure. The total amount of amines excreted in urine within 24 hours from start of exposure ranges between 12–21% of the estimated inhaled dose .Free and conjugated forms are found in the urine. The mean half-life in plasma is 21 days for TDI and 10–22 days for MDI chronically exposed, two times as long as for short-term exposure. The estimated medians of the urinary half-life are 18 days for 2,4-TDI and 19 days for the 2,6-TDI when exposure is due to thermal degradation of TDI-based pol-yurethane. Adducts with hemoglobin and albumin are also noted.
Effects 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 sensitization/"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 is exposure is small and brief. TDI can cause severe eye irritation with permanent damage if untreated.
Confounders/ Non Occupational Exposure	None

OCCUPATIONAL EXPOSURE

Medical Surveillance	**Medical and occupational history**	
	Physical	As per occupational medical program
	Sentinel health events	Occupational asthma
Biological Monitoring	**Sample:** Urine Toluendiamine Serum IgE TDI Serum IgE MDI Serum IgE HDI	**Sampling time:** ES (end of shift)
	Reference limits: Not industrial exposed Biological monitoring for industrial exposure Not industrial exposed Not industrial exposed Not industrial exposed	 < 3.6 ug/g creatinine Not available < 0.1 U/mL < 0.1 U/mL < 0.1 U/mL
Biological Effect Monitoring	Spirometry Chest Xray	Yes Yes

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METHANOL

Chemical Formula	CH_3OH
Synonyms	Methyl alcohol, carbinol, wood alcohol, colonial spirit, methyl hydroxide, methylol, pyroxylic spirit, wood naphata, wood spirit
CAS number	
Description	Methanol is a colorless, flammable liquid soluble in water. Crude material may have a repulsive, pungent odor. Fully soluble in water, alcohols, ketones, esters and halo-genayed hydrocarbons
Uses	Serves as a starting material in the manufacture of many chemical products, approximately 40% in wood for the production of formaldehyde, synthesis of methacrylates, methylamines, methylhalides, used as a 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, antidetonant fuel-injection fluid for aircraft. A major use is in the production of methyl tertiary butyl ether (MTBE).
Metabolism (Includes absorption & elimination)	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 vapors. 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 metabo-lites. The elimination is fast and complete. The major elimination pathway is metabolism. The total amount excreted this way accounts for about 70–80% of the absorbed amount. The elimination is a saturable process with elimination half-lives of about 1.5–2 hours) The major metabolite in humans is formic acid, which is responsible for the unique manifestations of methanol poisoning, metabolic acidosis and optic neuropathy.
Effects of Occupational Exposure	Mildly toxic by inhalation. Systemic effects by ingestion and inhalation including optic neuropathy, headache, cough, nausea and vomiting, 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 two to four 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.
Confounders/ Non Occupational Exposure	Formic acid is formed endogenously and is ubiquitous in the environment. Methanol is formed endogenously and may be present in the diet in low concentrations. It is a component of paint removers and solvent-based cleaners. It may be present in al-coholic beverages. It is used in automotive fuel in concentrations as high as 15%. It is a metabolite of a number of methyl esters, such as methyl acetate and ethylene glycol monomethyl ether which may be present in consumer products. Substitute for petrol. Abuse of solvent preparations. Also found in fruit and vegetable, fruit juices, and fermented beverages.

OCCUPATIONAL EXPOSURE

Medical Surveillance	**Medical and occupational history**	
	Physical	Ophthalmology
	Sentinel health events	Optic neuropathy
Biological Monitoring	**Sample:** Methanol in urine Formic acid in urine Methanol in blood	**Sampling time:** ES (end of shift) BS (before shift) at EW (end of work-week) EW (end of work)
	Reference limits: 1. Methanol: creatinine Not industrial exposed BEI (Biological Exposure Index) 2. Formic acid: creatinine Not industrial exposed BEI (Biological exposure index) 3. Methanol in blood Normall Toxic Indication for haemodialysis	 < 2.5 mg/g creatinine 10.5 mg/g creatinine (ES, EW) < 23.1 mg/g creatinine 80 mg/g creatinine(BS, EW) < 0.05 mmol/L > 6.24 mmol/L > 14 mmol/L
Biological Effect Monitoring	Urine Blood	Dipstix (pH) Urea, creatinine, ALT, AST, GGT, ALP

METHYL CHLOROFORM

Chemical Formula	CH_3CCl_3
Synonyms	1,1,1 – Trichloroethane, methyltrichloromethane, alpha – Trichloroethane, tri-chloromethylmethane.
CAS number	71–55–6
Description	Methyl chloroform is a colorless, volatile liquid.
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 pro-cessing and in various formulations including adhesives aerosols, coatings, printing inks, typewriter correction fluid, drain cleaners, shoe polish and as a carrier of aerosols. In industry it is used for cleaning, degreasing and as an extraction solvent.
Metabolism (Includes absorption & elimination)	Inhalation is a major route of absorption. Methyl chloroform is excreted almost entirely unchanged through the lungs. A small fraction of methyl chloroform is oxidized to trichloroethanol (TCOH) and then to trichloroacetic acid (TCAA).
Effects of Occupational Exposure	As for solvents, defattening of skin, narcotic at high levels.
Confounders/ Non Occupational Exposure	Methyl chloroform is used as a solvent in several household products such as spot removers, adhesives and aerosols. Abuse by inhalation has been reported. Trichloroa-cetic acid and trichloroethanol can be found in urine as a metabolite of other chlorinated hydrocarbons (trichloroethylene, chloral hydrate, perchloro ethylene and tetrachloro ethane) which can be found in household products. Work-load plays a significant role in update and elimination.

OCCUPATIONAL EXPOSURE

Medical Surveillance	**Medical and occupational history**	
	Physical	Annual, neurological, skin
	Sentinel health events	Skin, neurological
Biological Monitoring	**Sample:** Trichloroacetic Acid (TCAA) Trichloroethanol Blood Trichloroethane Blood	**Sampling time:** End of shift, End of work-week (ES, EW) Pre shift (PS)
	Reference limits: 1. Trichloroacetic acid: creatinine Not industrial exposed BEI (Biological exposure index) 100.0 mg/g creatinine (ES,EW) (OHS Act) 75.0 mg/g creatinine (ES,EW) (Lauwerys, Hoet) 2. Trichloroethanol blood Not industrial exposed BEI (Biological exposure index) 3. Trichloroethane blood Not industrial exposed BEI (biological exposure index) 4. 0 mg/L at end of shift (EW)	 < 3.5 mg/g creatinine Not detected 1 mg/L (ES, EW) < 0.3 mg/L 0.7 mg/L (PS, EW)
Biological Effect Monitoring	Blood Urine	Urea, creatinine, ALT, AST, ALP, GGT Dipstix

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METHYL ETHYL KETONE (MEK)

Chemical Formula	$CH_3\ COCH_2\ CH_3$
Synonyms	MEK, 2-Butanone, Methyl acetone, Methyl-2-propanone
CAS number	78–93–3
Description	Methyl ethyl ketone is a colorless, volatile liquid that is soluble in water. It has an acetone-like odor.
Uses	Methyl ethyl ketone (MEK) is one of the most widely used solvents in lacquers, paints, adhesives and coatings containing synthetic resins, plastics or rubber. MEK is a solvent widely 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 pollu-tant in shoe factories.
Metabolism (Includes absorption & elimination)	MEK is absorbed via all routes. 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.
Effects of Occupational Exposure	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. 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.
Confounders/ Non Occupational Exposure	Non-occupational exposure to MEK is uncommon; although it is a component of some products used in hobby activities and could be present in some household products. Exposure to 2-butanol, which is metabolized to MEK, may result in the presence of MEK in urine. MEK potentiates the neurotoxicity of hexacarbon compounds as well as liver and kidney toxicity of haloalkanes.

OCCUPATIONAL EXPOSURE

Medical Surveillance	**Medical and occupational history**	
	Physical	As per occupational medical program
	Special investigation	Nervous system
	Sentinel health events	Skin - allergic/contact dermatitis, eyes, upper respiratory system
Biological Monitoring	**Sample:** MEK in urine	**Sampling time:** ES (End of Shift)
	Reference limits: Not industrial exposed BEI (Biological Exposure Index)	 < 0.29 mg/g creatinine 1.4 mg/g creatinine
Biological Effect Monitoring	Blood Urine Spirometry Chest X-Rays ECG	Urea, creatinine, ALT, AST, ALP, GGT DIPSTIX - - -
Reproductive/ Developmental Effects	No evidence available	

METHYL ISOBUTHYL KETONE (MIBK)

Chemical Formula	(CH_3) $CHCH_2$ $COCH_3$ $(CH_3)CHCH_2COCH_3$, $C_6H^{12}O$
Synonyms	MIBK, 4-Methyl- 2-pentanone, Isobutyl methyl ketone, Isopropyl acetone, Hexone, MIK, Hexanone 4-Methyl-2-oxopentane, 2-methyl-4pentanone, 2-methylpropyl methyl ketone, 2-methylvaleraldehyde
CAS number	108–10–1
Description	Methyl Isobutyl Ketone (MIBK)is a colorless, flammable liquid with a mild, pleasant odor.
Uses	Used principally as a solvent for protective coatings, lacquers and varnishes. It is also used as a raw material in the production of antioxidants, as an extraction solvent for metals and pharmaceuticals, in the production of paints and pesticide formulations and as a solvent for adhesives. MIBK is used as a dena-turant in cosmetic products. MIBK is a synthetic flavouring adjuvant. MIBK occurs naturally in plant and animal oils.
Metabolism (Includes absorption & elimination)	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 three hours. Absorbed MIBK is essentially completely cleared out of the system by 90 minutes after exposure. MIBK is metabolised in the liver to water-soluble excretory products. Therefore, urine is the major excretory route for MIBK excretion.
Effects 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.
Confounders/ Non Occupational Exposure	Nil

OCCUPATIONAL EXPOSURE

Medical Surveillance	**Medical and occupational history**	
	Physical	Physical
	Sentinel health events	Enlargement of liver, contact dermatitis
Biological Monitoring	**Sample:** MIBK in urine	**Sampling time:** ES (End of shift)
	Reference limits: Not industrial exposed BEI (Biological Exposure Index)	 < 0.29 mg/g creatinine 1.40 mg/g creatinine
Biological Effect Monitoring	Blood Urine	Urea, creatinine, ALT, AST, GGT, ALP Dipstix
Reproductive/ Developmental Effects	No information available	

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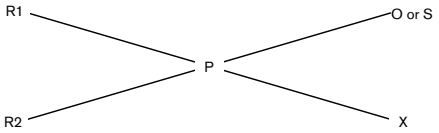
NITROBENZENE

Chemical Formula	$C_6H_5NO_2$
Synonyms	Nitrobenzol; Benzene, nitro-; Oil of mirbane; Oil of bitter Almonds,Nitro-Benzene;
CAS number	98–95–3
Description	Nitrobenzene is a pale yellow oily liquid with pungent odor like almond or shoe polish.
Uses	Nitrobenzene is mainly used in the production of Aniline. It is also used to manufac-ture dyes, oils, drugs, pesticides and synthetic rubber.
Routes of Exposure	Exposure may be through inhalation, ingestion and skin contact
Metabolism (Includes absorption & elimination)	Nitrobenzene absorption is through inhalation and skin contact. It is oxidized to p-aminophenol, which is excreted by the kidney. Exposure causes the formation of methaemoglobin resulting in a functional anemia. As it is heavier than air it may cause asphyxiation in poorly ventilated areas.
Effects 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 two to four 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.
Confounders/ Non Occupational Exposure	Nitrobenzene is used in the production of dyes, rubber formulations and medicinals, residual amounts can be found in the products. It is commonly found in shoe polishes and has been used as an artificial almond flavouring agent. Commercial use in perfumes and as a combustible propellant might be a source of non-occupational exposure.

OCCUPATIONAL EXPOSURE

Medical Surveillance	**Medical and occupational history**	
	Physical	As per occupational medical program
	Sentinel health events	Liver, Kidneys, CNS Anemia and Cyanosis
Biological Monitoring	**Sample** Total p-aminophenol in urine Methaemoglobin in blood	**Sampling time** ES (end of shift) EW (end of work-week) During or end of shift
	Reference limits: 1. Total p-aminophenol: creatinine Not industrial exposed BEI (Biological exposure index) 2. Methaemoglobin in blood Not industrial exposed BEI (Biological exposure index)	 Not determined 5 mg/g creatinine 1.5% of haemoglobin
Biological Effect Monitoring	Blood Urine Spirometry	Full blood count, ALT, ALP, AST, GGT Dipstix Yes

ORGANOPHOSPHORUS [OP] CHOLINESTERASE INHIBITORS

Chemical Formula	 (Here 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 a nitrile)
Synonyms	Azinphos-methyl, Demeton, Diazinon,dichlorvos, Dioxathion, EPN, Ethion, Fenami-phos, Fensulfothion, Fenofos, Malathion, Methomyl, Metyl demeton, Methyl parathion, Mevinphos, Naled, Parathion , Sulfotep, Temephos, TEPP, Carbamates
CAS number	Several
Description	Organophosphorous chemicals are mainly used in pesticides. They generally occur in the air as aerosols and dusts.
Uses	Pesticides
Metabolism (Includes absorption & elimination)	Route of entry: skin contact, inhalation, ingestion (food). The onset and severity of symptoms depend on the chemical structure of the com-pound being used, the amount of toxin to which individual is exposed, route of exposure, rate of metabolic degradation, respiratory rate, ambient temperature, humidity, the use of personal protective equipment. Following oral or respiratory exposure, signs and symptoms appear three hours later, while with dermal exposure, they are delayed to usually 12 hours post-exposure. OPs 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 organophosphorous chemicals are eliminated in the urine in the form of me-tabolites (dialkyl phosphates). About 90% of the compound is eliminated between six and 24 hours after absorption.
Effects 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.
Confounders/ Non Occupational Exposure	There is a considerable intra-individual and inter-individual variability in Erythrocyte or Red Blood Cell (RBC) cholinesterase activity in unexposed persons. Values after expo-sure should therefore ideally be compared to baseline values obtained for the same individual before exposure. Failing that, control measurements for the general population may be used. Non occupational exposure to some organophosphates may occur due to home gardening and household use – cholinesterase inhibition can also be induced by drugs such as chotiophate, neostigimine and physotigmine. RBC cholinesterase (generally reflects the previous 120 days exposure) must be inter-preted with caution in conditions where RBCs are falsely decreased i.e. pernicious anemia, hemoglobinopathies, and in individuals taking anti-malarials.

OCCUPATIONAL EXPOSURE

Medical Surveillance	**Medical and occupational history**	
	Physical	Annual
	Sentinel health events	Acute exposure symptoms,neuropathy
Biological Monitoring	**Sample:** Whole blood cholinesterase activity Pseudocholinesterase –serum(CHE)	**Sampling time:** Discretionary Pre-shift / Post exposure [generally baseline taken during season or peak application period]. True Baseline Level = taken four weeks after non-exposure; ideally two baseline measurements are to be done three to14 days apart; should agree to 15–20% After acute exposure
	Reference limits: A reduction of 30% or more from a basal (pre-exposure) level may indicate organophosphate toxicity/exposure. M&F: 4829-7350 U/L	
Biological Effect Monitoring	Blood Urine ECG	Full blood count, urea, creatinine, electrolytes, ALT, AST, GGT, ALP Dipstix Cardiac arrhythmias
Reproductive/ Developmental Effects	No conclusive information available	

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PARAQUAT

Chemical Formula	$C_{12}H_{14}C_{l2}N_2$
Synonyms	N,N'-dimethyl-4,4'bipyridinium dichloride, Paraquat dichloride, Methyl viologen dichloride, Crisquat, Dexuron, Esgram, Gramuron, Ortho Paraquat CL, Paracol, Pillarxone, Tota-col, Toxer Total, PP148, Cyclone, Gramixel, Gramoxone, Pathclear, AH 501
CAS number	1910–42–5
Description	Toxic chemical – Yellow solid with faint ammonia like odor
Uses	Herbicide-inhibits photosynthesis
Metabolism (Includes absorption & elimination)	Route of entry: inhalation, skin contact, the eyes and ingestion. 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 unmetabolized and excreted unchanged in urine
Effects 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
Confounders/ Non Occupational Exposure	Non occupational exposure to Paraquat is uncommon. Paraquat normally marketed containing a blue dye to prevent it from being confused with coffee, a sharp odor to serve as a warning and has an added agent to cause vomiting when swallowed

OCCUPATIONAL EXPOSURE

Medical Surveillance	**Medical and occupational history**	
	Physical	As per occupational medical program
	Sentinel health events	Poison
Biological Monitoring	**Sample:** Urine	**Sampling time:** After acute exposure
	Reference limits: Qualitative result should be negative to exclude Paraquat exposure	
Biological Effect Monitoring	Blood Urine Chest Lung	Arterial blood gas ,Kidney function Urinalysis X Ray Lung function test
Reproductive/ Developmental Effects	No conclusive information available	

PARATHION (ORGANOPHOSPHATE)

Chemical Formula	$(C_2H_5O)_2$ $PSOC_6H_4NO_2$
Synonyms	Nitrostigmine, Ethyl Parathion, O,O-diethyl O-p-nitrophenyl phosphoro-thianate, phosphorothioate acid, Folidol, Alleron,Etilon, danthion, Niran, paraphos, Rhodiatox, Thiophos
CAS number	56–38–2
Description	Pale yellow liquid when pure. Insoluble in water
Uses	Primarily used as an insecticide on fruit, cotton, wheat, vegetables and nut crops
Metabolism (Includes absorption & elimination)	Route of entry: inhalation, skin contact, the eyes and ingestion. Exposure is generally during manufacture, formulation, and in field application. The hepatic mixed function oxidises metabolize Parathion to Paraoxon. The latter is an active metabolite which inhibits cholinesterase. The esterase's found in plasma and tissue hydrolyse Parathion and paraoxon to alkyl phosphates (diethylthiophosphoric acidor diethyltiophophate (DETP) and dieth-ylphosphoric acid or diethylphosphate (DEP) and the main excretory metabolite of Parathion, p-nitrophenol. Elimination is via urine with 80–90% of absorbed dose eliminated within 48 hours
Effects 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 individual is exposed, route of exposure, rate of metabolic degradation, respiratory rate, ambient temperature, humidity, 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
Confounders/ Non Occupational Exposure	Non occupational exposure to parathion is uncommon. Traces of parathion could occur on food, however such exposure is unlikely. Also see notes for organosphosphorus cholinesterase inhibitors with reference to cholinesterase activity in whole blood

OCCUPATIONAL EXPOSURE

Medical Surveillance	**Medical and occupational history**	
	Physical	As per occupational medical program
	Sentinel health events	Neurological,
Biological Monitoring	**Sample:** Whole blood cholinesterase activity Pseudocholinesterase –serum(CHE) Total p-nitrophenol in urine	**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 are to be done 3 - 14 days apart; should agree to 15 - 20% After acute exposure ES (End of shift)
	Reference limits: 1. A reduction of 30% or more from a basal(pre-exposure) level may indicate organophosphate toxicity/exposure. 2. M&F: 4829-7350 U/L 3. Biological exposure index: 0.5mg/g creatinine	
Biological Effect Monitoring	Blood Urine ECG	Full blood count, urea, creatinine, electrolytes, ALT, AST ALP, GGT Dipstix Yes
Reproductive/ Developmental Effects	No conclusive information available	

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PENTACHLOROPHENOL

Chemical Formula	$C_6C_{l5}OH$
Synonyms	PCP, Penta, Penchlorol, Pentacon, Penwar, Penta, Dowicide, Santophen 20
CAS number	87–86–5
Description	Colorless or white (pure) crystals with a phenolic odor.
Uses	PCP is used as a wood, leather and paper preservative, a pesticide, a disin-fectant, a mildew retardant, a fungicide and a contact herbicide (chlorinated hydrocarbon). Persons may come into contact with it during manufacturing processes and application procedures.
Metabolism (Includes absorption & elimination)	Route of entry: lungs, skin and gastrointestinal tract 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) in 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.
Effects of Occupational Exposure	Short term: Acute exposure to PCP may cause irritation to the eyes and 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 anemia, liver damage, renal damage, cardio-vascular and central nervous system effects. The IDLH is 2.5 mg/m^3
Confounders/ Non Occupational Exposure	Environmental exposure occurs from a number of sources. PCP is used as a preservative for wood, leather and paper, and as a paint-on wood preservative for home use. Significant exposure can occur to occupants of log cabins treated with PCP. It has also been found in low concentrations in drinking water and certain food commodities.

OCCUPATIONAL EXPOSURE

Medical Surveillance	**Medical and occupational history**	
	Physical	As for occupational medical program
	Sentinel health events	Skin, liver, respiratory, aplastic anemia
Biological Monitoring	**Sample:** Total PCP in urine Free PCP in plasma	**Sampling time:** PS (Prior to the last shift, end of work-week) ES (End of shift)
	Reference limits: 1. PCP in urine Not industrial exposed BEI (Biological exposure index) 2. PCP in plasma Not industrial exposed: BEI (Biological exposure index)	 < 0.15 mg/g creatinine 2 mg/g creatinine prior to the last shift of the work-week < 0.40 mg/L 5 mg/L at end of shift
Biological Effect Monitoring	Blood Urine Spirometry Chest X-Rays ECG	Full blood count and diff, urea, creatinine ALT, AST, GGT, ALP, Bilirubin Dipstix Yes Yes Yes
Reproductive/ Developmental Effects	No conclusive evidence available for direct causal effects	

PERCHLOROETHYLENE

Chemical Formula	$CCl_2 = CCl_2$
Synonyms	Tetrachloroethylene, Perc, Ethylene tetrachloride, PCE, Perchlor, tetrachloroethylene, tetrachloreothene, 1,1,2,2-tetrachloroethylene
CAS number	127–18–4
Description	Colorless liquid used mainly as a solvent. May decompose to phosgene and hydrogen chloride gases which are hazardous. Vapor is heavier than air.
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.
Metabolism (Includes absorption & elimination)	Route of entry: inhalation, dermal and eye contact. After absorption, a fraction of perchlorethylene is oxidised to trichloroacetic acid (TCAA). Human ability to metabolize 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 four percent, 16 hours after a single eight hour exposure period has ended. Between one and three pervent 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.
Effects 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
Confounders/ Non Occupational Exposure	Perchlorethylene is not usually present in end-exhaled air and in blood of non-occupationally exposed people. Low concentrations may be found in people who have visited dry-cleaning establishments or who were in contact with recently dry-cleaned clothes. TCAA can be present in urine as a metabo-lite of other chlorinated solvents (trichloroethylene, methyl chloroform and tet-rachloroethane), some of which can be found in household products. Use of chloral hydrate (a sedative hypnotic drug) may be followed by significant amounts of TCAA.

OCCUPATIONAL EXPOSURE

Medical Surveillance	**Medical and occupational history**	
	Physical	Annual as for occupational medical program
	Sentinel health events	Skin, kidney, liver
Biological Monitoring	**Sample:** Trichloracetic acid in urine Tetrachloroethylene in blood	**Sampling time:** (ES) end of shift (EW) end of work-week (PS) prior to last shift (EW) end of work-week
	Reference limits: 1. Trichloroacetic Acid: creatinine Not industrial exposed BEI (Biological exposure index) 4.9 mg/g creatinine (EW)(OHS Act) 3.0 mg/g creatinine (ES,EW) (Lauwerys, Hoet) 2. Tetrachloroethylene in blood Not industrial exposed BEI (Biological exposure index) 1.0 mg/(PS, EW) (OHS Act) 0.5 mg/L (PS, EW) (ACGIH)	 < 3.5 mg/g creatinine Not detected
Biological Effect Monitoring	Blood Urine Spirometry	ALT, AST, ALP, GGT, urea, creatinine and electrolytes Dipstix Yes
Reproductive/ Developmental Effects	No conclusive evidence available for direct causal effects	

PHENOL

Chemical Formula	C_6H_5OH
Synonyms	Carbolic Acid, Hydroxybenzene, Phenic Acid, Phenyl Hydroxide, Phenylic Acid, Phenyl Alcohol, Benzynol
CAS number	108–95–2
Description	Colorless or white solid when pure. It has a sweet and acrid odor
Uses	Commercially as a disinfectant and as an intermediate in chemical syntheses such as nylon and other man-made fibres.
Metabolism (Includes absorption & elimination)	Route of entry: inhalation & especially skin contact. 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 three and a half hours. Less than 1% of inhaled dose is exhaled
Effects of Occupational Exposure	Burns eyes and skin effect in tissue. Absorption may produce cyanosis, shock, weakness, collapse, convulsions, liver and kidney damage (mainly), coma and death (IDLH = 250ppm). Phenol exposure increases the risk of coronary artery disease. Pigment changes of the skin are noted
Confounders/N on Occupational Exposure	Phenol is a product of protein metabolism and its concentrations in the urine can be influenced by diet. Phenol is a common ingredient in antiseptic medi-cines, such as phenol-camphor-petrolatum lotions, antiseptic throat lozenges (these can severely increase measured levels of phenol), calamine lotions and some over-the-counter antacid preparations. Phenol is also presenting disin-fectants and other household chemicals. Phenol is a major metabolite of benzene and exposure to benzene results in the increased excretion in urine.

OCCUPATIONAL EXPOSURE

Medical Surveillance	**Medical and occupational history**	
	Physical	As for occupational medical Program
	Sentinel health events	Skin, respiratory, liver, kidney
Biological Monitoring	**Sample:** Total phenol in urine	**Sampling time:** (ES) end of shift
	Reference limits: Not industrial exposed BEI (Biological exposure index)	 < 20 mg/g creatinine 250 mg/g creatinine
Biological Effect Monitoring	Blood Urine Spirometry Chest X-Rays ECG	Urea, creatinine, ALT, AST, ALP, GGT Dipstix + microscopy Yes Yes -
Reproductive/ Developmental Effects	Nil	

POLYCYCLIC AROMATIC HYDROCARBON (PAH)

Chemical Formula	Benzo-(a)- pyrene (B[a]P)
Synonyms	PAHs are multi-numbered benzenoid-ring compounds. PAHs contain polycyclic aromatic hydrocarbons (PAHs), substituted PAHs, and PAH heterocyclic derivatives.
CAS number	Different CAS number for each compound
Description	The term polycyclic aromatic hydrocarbons (PAHs) refers to a ubiquitous group of several hundred chemically-related, organic compounds. Most of them are formed by a process of thermal decomposition (pyrolysis) and subsequent recombination (pyrosynthesis) of organic molecules. Polycyclic aromatic hydro-carbons have two or more single or fused aromatic/ benzene rings with a pair of carbon atoms shared between rings in their molecules. PAHs are highly lipophilic and therefore very soluble in organic solvents. PAHs enter the environment through various routes and are usually found as a mixture containing two or more of these compounds, e.g. motor vehicle emissions, domestic coal/oil fired heating systems, and industrial sources. The following more common PAHs have been identified as being of greatest concern with regard to potential exposure and adverse health effects on humans and are thus considered as a group (profile issued by the Agency for Toxic Substances and Disease Registry) and include benz(a)anthracene, ben-zo(a)pyrene, benzo(e) pyrene, benzo(b)fluoranthene, benzo(ghi)perylene, benzo(k)fluoranthene, chrysene, dibenz(ah)anthracene, fluoranthene, and coronene.
Uses	Sources of PAHs can be both natural and anthropogenic. Natural sources of PAHs include forest and grass fires, oil seeps, volcanoes, chlorophyllous plants, fungi, and bacteria. High concentrations of PAHs are found in coke ovens, aluminium-reduction plants, steel industry, asphalt industry, creosote impregnating plants, and the gas and petroleum industries. PAHs are not synthesized chemically for industrial purposes. The major source of PAHs is the incomplete combustion of organic material such as coal, oil and wood. They are mostly used as intermediaries in pharmaceuticals, agricultural products, photographic products, thermosetting plastics, lubricating materials, and other chemical industries. General uses are: • Acenaphthene: manufacture of dyes, plastics, pigments, pharmaceuticals and pesticides • Anthracene: manufacture of dyes and pigments; diluent for wood preservatives • Fluoranthene: manufacture of dyes, pharmaceuticals and agrochemicals • Fluorene: manufacture of dyes, pigments, pesticides, thermoset plastic and pharmaceuticals • Phenanthrene: manufacture of pesticides and resins • Pyrene: manufacture of pigments • Asphalt used for the construction of roads, as well as roofing tar • Specific refined products, are used also in the field of electronics, functional plastics, and liquid crystals
Routes of Exposure	The major route of exposure to PAHs in the general population is from breathing ambient and indoor air, eating food containing PAHs, smoking cigarettes, or breathing smoke from open fireplaces. Tobacco smoke contains a variety of PAHs, such as benzo(a)pyrene, and more than 40 known or suspected human carcinogens. For non-smokers the main route of exposure is through food (PAH levels variable). Charring meat or barbecuing food over a charcoal, wood, or other type of fire greatly increases the concentration of PAHs. Some crops, such as wheat, rye, and lentils, may synthesize PAHs or absorb them via water, air, or soil. Water can also contain PAHs since those chemicals can leach from soil into water or they can enter the water from industrial effluents and accidental spills during oil shipment at sea. Occupational exposure occurs in workers breathing exhaust fumes, such as mechanics, street vendors, motor vehicle drivers, as well as those involved in mining, metal working, or oil refining. Routes of exposure include ingestion, inhalation, and dermal contact in both occupational and non-occupational settings. Some exposures may involve more than one route simultaneously, affecting the total absorbed dose (such as dermal and inhalation exposures from contaminated air).
Metabolism (Includes absorption & elimination)	The most studied of the PAHs is benzo[a]pyrene The major route of exposure is via the lungs. The fate depends on the chemical and structural nature, and the dimensions. The PAH particles may dissolve, may be remove by bronchomucociliary action, or may remain in the lung for a long time. The major de-pots for PAHs are adipose tissue and mammary gland. Once absorbed, PAHs enter the lymph, circulate in the blood, and are me-tabolised primarily by the kidneys and liver. PAHs metabolised by the CYP-450 enzyme complex in the liver resulting in hydroxylated metabolites. PAHs are transformed initially to epoxides, which are converted to dihydrodiol derivatives and phenols. Glucuronide and sulfate conjugates of these me-tabolites are excreted in the bile and urine. Glutathione conjugates are further metabolized 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 sulfate. A commonly measured urinary metabolite is 1-hydroxypyrene Metabolism is a prerequisite for hepatobiliary excretion and elimination through the feces, regardless of route of entry. Excretion half-lives in faeces and urine have been reported in animal studies as 22 hours and 28 hours, respectively
Effects of Occupational Exposure	PAHs are photosensitsers i.e. an abnormally high reactivity in the skin and eyes to ultraviolet radiation or natural sunlight. The dermal toxic effects are en-hanced 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 car-cinogenic. 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).

Confounders/ Non Occupational Exposure	Occupational exposure is generally to a mixture of compounds with similar physical and chemical properties. Therefore the major metabolite of PAH exposure used in monitoring is the urine conjugate of 1-hydroxypyrene. 1-hydroxypyrene excretion is higher in smokers than in non-smokers [additive effect - 1 cigarette contains 20–40 ng of benzo(a)pyrene] Urinary 1-hydroxypyrene is not applicable for biological monitoring of extremely low levels of exposure eg. individuals working in the street. Internal exposure to PAHs does not correlate with external exposure levels. For routine monitoring where pulmonary exposure is the main route, pre shift samples on a Monday and post shift samples on a Friday are recommended. When skin exposure is the predominant exposure route, pre shift samples Monday and Friday are recommended. Non-workplace exposure is from diet, smoking, and the burning of wood and coal.

OCCUPATIONAL EXPOSURE

Medical Surveillance	**Medical and occupational history**	
	Physical	As per occupational medical program
	Sentinel health events	Skin, respiratory tract
Biological Monitoring	**Sample:** urine	**Sampling time:** (ES) end of work week
	Reference limits: Not industrial exposed BEI (Biological exposure index)	 < 2.0 ug/g creatinine 2.7 ug/g creatinine (ES) end of shift
Biological Effect Monitoring	Blood Urine Spirometry Chest X rays	Serum U&E, full blood count, liver function test Dipstix – proteinuria, haematuria Yes Yes
Toxicity	IARC classification is Group 1 carcinogen for benzo-(a)-pyrene exposure	

STYRENE

Chemical Formula	$C_6H_5CH = CH_2$
Synonyms	Styrol, Vinylbenzene, Phenylethylene, Styrolene Cinnamene Ethenylbenzene
CAS number	100–42–5
Description	Colorless to light yellow, watery liquid with a sweet pleasant odor. The liquid is flammable with a flash point of 31 deg C. Phosgene may produce on degradation.
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-butadien styrene (ABS), styrene-acrylonitril (SAN) and styrene-butadiene copolymers, in manufacture of surface coatings, use in miscellaneous processes as an elastomer, intermediate, or starting material, during manufacture ofion-exchange resins (styrene-divinylbenzene copolymer)
Metabolism (Includes absorption & elimination)	**Route of entry:** inhalation (main absorption), skin (liquid/ vapor form) one to two percent of inhaled sytrene is exhaled unchanged. Styrene is metabolized to mandelic acid (MA) and phenylglyoxylic acid (PhGA). These are excreted in the urine with a half-life of five to10 hours. Elimination of styrene (less than 1% of the absorbed amount is eliminated as styrene) from the lungs is biphasic with half-lives of 13–52 minutes and four and 20 hours respectively. Elimination of MA (the major metabolite) from urine is biphasic with a half-life of three to four hours and 25–40 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.
Effects of Occupational Exposure	Inhalation causes irritation of the mucous membranes (@ 300ppm) (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)
Confounders/ Non Occupational Exposure	Non occupational exposure to styrene is uncommon. Styrene is a component of some do-it-yourself, auto body patching compounds. Mandelic acid and phenylglyoxylic acid are metabolic products of ehtylbenzene, styrene glycol, styrene oxide, phenylgycol, acetophenene, phenylglycerine and phenylamino-acetic acid and other chemicals of a similar chemical structure. Ethanol is an inhibitor of styrene metabolites

OCCUPATIONAL EXPOSURE

Medical Surveillance	**Medical and occupational history**	
	Physical	As for occupational medical program
	Sentinel health events	Skin, CNS, eyes, liver, respiratory tract
Biological Monitoring	**Sample:** Mandelic acid in urine Phenylglyoxylic acid in urine Styrene in venous blood	**Sampling time:** ES (end of shift), (BS) before to next shift
	Reference limits: 1. Mandelic acid: creatinine Not industrial exposed BEI (Biological Exposure Index) Before next shift 300 mg/g creatinine (OHS act) 100 mg/g creatinine (Lauwerys, Hoet) 2. Phenylglyoxylic acid: creatinine Not industrial exposed Before next shift 100 mg/g creatinine 3. Styrene blood Not industrial exposed BEI (Biological Exposure Index) 0.55 mg/l at end of shift (OHS act)	 < 11 mg/g creatinine At end of shift: 800 mg/g creatinine (OHS act) 300 mg/g creatinine (Lauwerys, Hoet) < 13 mg/g creatinine At end of shift 240 mg/g creatinine < 0.03 mg/L 0.30 mg/L at end of shift (Lauwerys, Hoet)

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TOLUENE

Chemical Formula	$C_6H_5CH_3$
Synonyms	Toluol, Methyl benzene, Methyl benzol
CAS number	108–88–3
Description	A colorless, flammable, colorless liquid with a pleasant benzene-like odor. This is a flammable liquid with vapor heavier than air.
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.
Metabolism (Includes absorption & elimination)	Route of entry: inhalation (primary route), ingestion and skin 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 tri-phasic with half-lives of one and a half minute, 26 minutes and (tentatively) 3.7 hours. The remainder is excreted in the urine after being metabolized to hippuric acid (mainly) and o-cresol (less than 1% of the absorbed amount). Elimination of hippuric acid has a half-life of one to two hours according to American sources, but seven to eight 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 metabo-lites (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 o-cresol is decreased by co-exposure to benzene.
Effects of Occupational Exposure	Inhalation can cause irritation to mucous membranes (eye, nose and throat) and can cause nausea, vomiting, headaches, dizziness and loss of conscious-ness. 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 defat skin causing it 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 de-crease in lung function (FEV1).
Confounders/ Non Occupational Exposure	Toluene is a component of some household products such as paints, paint strippers, glues and some cleaners. Gasoline in USA contains form 5–18% of toluene. Glues and adhesives are sometimes abused to achieve “highs”. Hippuric acid is produced by the metabolism of certain acid foods, such as plums or cranberries or from the pre-servative, sodium benzoate.

OCCUPATIONAL EXPOSURE

Medical Surveillance	**Medical and occupational history**	
	Physical	Annual as for occupational medical program
	Sentinel health events	Skin, CVS, Liver, Kidney, Hearing, respiratory system
Biological Monitoring	**Sample:** Hippuric acid in urine Toluene in venous blood o-Cresol in urine	**Sampling time:** ES (end of shift) ES (end of shift) ES (end of shift)
	Reference limits: 1. Hippuric acid in urine Not industrial exposed BEI (Biological exposure index) 2. Toluene in venous blood Not industrial exposed BEI(Biological Exposure Index) At end of shift Prior to last shift of week 3. o-Cresol in urine Not industrial exposed BEI (Biological exposure index)	 < 1.5 g/g creatinine 2.5 g/g creatinine (OHS act) (ES), 1.5 g/g creatinine (Lauwerys, Hoet) (ES) < 0.005 mg/L 1.0 mg/L (OHS act), 0.5 mg/L (Lauwerys, Hoet) 0.05 mg/L (Lauwerys, Hoet) < 0.3 mg/g creatinine 1.0 mg/g creatinine (OHS) (ES) 0.5 mg/g creatinine (Lauwerys,Hoet) (ES)
Biological Effect Monitoring	Urine Spirometry Chest X-Rays ECG	Dipstix Yes Yes Yes

Reproductive and Developmental effects	Developmental and other birth defects have been noted	Reproductive and Developmental effects
Biological Effect Monitoring	Blood Urine Spirometry Chest X-Rays ECG	Urea, creatinine, ALT, AST, ALP, GGT, Dipstix Yes Yes OMP to decide

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TRICHLOROETHYLENE

Chemical Formula	$CCl_2 = CHCl$
Synonyms	Acetylene trichloride, ethylene chloride, TCE, Triclene, Algylen, Chlorylen, Gemalgene, Trethylene, Trichloran, Trilene 1,1,2-trichlorotheylene
CAS number	79–01–6
Description	A non-flammable colorless liquid that does not occur naturally in the environment. The vapor is heavier than air.
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.
Metabolism (Includes absorption & elimination)	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 metabolized by hepatic mixed function oxidases to chloral hydrate. The latter is rapid 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–hours. Alcohol, caffeine and drugs effects.
Effects 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 coordination 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.
Confounders/ Non Occupational Exposure	TCE is used as a solvent in some household products such as spot removers and adhe-sives. Abuse by “sniffers” has been reported. Water from wells near waste sites may contain concentrations exceeding 1ppm

OCCUPATIONAL EXPOSURE

Medical Surveillance	**Medical and occupational history**	
	Physical	Annual as for occupational medical Program
	Sentinel health events	Eyes, skin, liver, kidneys, CVS, respiratory, CNS
Biological Monitoring	**Sample:** Total Trichloro Compounds (TTCC) Trichloroacetic Acid (TCAA) Trichloroethanol (TCE) Trichloroethylene in blood	**Sampling time:** ES (end of shift), EW (end of work-week) ES (end of shift), EW(end of work-week) ES (end of shift), EW (end of work-week) ES (end of shift), EW (end of work-week)
	Reference limts: 1. Total Trichloro Compounds: creatinine Not industrial exposed BEI (Biological exposure index) Trichloroacetic acid + Trichlorethanol 2. Trichloroacetic acid: creatinine Not industrial exposed BEI (Biological exposure index) 3. Trichloroethanol: creatinine Not industrial exposed BEI (Biological exposure index) 4. Trichloroethylene in blood Not industrial exposed BEI (Biological exposure index)	 Not detected 300.0mg/g creatinine < 3.5 mg/g creatinine 100 mg/g creatinine at (ES) (EW) (OHS Act) 75 mg/g creatinine (ES) (EW) (Lauwerys, Hoet) < 3.5 mg/g creatinine 150 mg/g creatinine at (EW) Not detected 1.0mg/L end of shift end of work-week
Biological Effect Monitoring	Blood Urine Spirometry Chest X-Rays ECG	ALT, AST, GGT, ALP, urea, creatinine Dipstix Yes
Reproductive and Developmental effects		

XYLENE

Chemical Formula	C_6H_4 $(CH_3)_2$
Synonyms	m-Xylene: 1,3-Dimethylbenzene; o-Xylene: 1,2-Dimethylbenzene; p-Xylene: 1,4-Dimethylbenzene All forms: Xylol
CAS number	
Description	Colorless, flammable, watery liquid with an irritating odor.
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.
Metabolism (Includes absorption & elimination)	The main route of absorption is through inhalation. About 60% of the inhaled amount is retained after eight hours of exposure. Dermal absorption through skin contact with the liquid is also significant. Gastro-intestinal absorption (through ingestion) is rapid. An amount of three to six percent of the absorbed Xylene is exhaled in unchanged form. This elimination route is biphasic with half-lives of one hour and 20 hours. About 95% of elimination occurs through the urine, after being metabolized 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, form whence elimination proceeds slowly.
Effects 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).
Confounders/ Non Occupational Exposure	Xylenes are common ingredients in paints, varnishes, thinners and some adhesives used in the home.

OCCUPATIONAL EXPOSURE

Medical Surveillance	**Medical and occupational history**	
	Physical	Annual: as for occupational medical program
	Sentinel health events	Eyes, CNS, Blood, Liver, Kidneys, Skin
Biological Monitoring	**Sample:** Methylhippuric acid in urine Xylene in blood	**Sampling time:** ES (end of shift) last 4 hours of shift DS (During Shift)
	Reference limits: 1. Methylhippuric acid:creatinine Not industrial exposed BEI (Biological exposure index) 2. Xylene in blood Not industrial exposed: BEI (Biological exposure index)	 < 0.012 g/g creatinine 1.5 g/g creatinine at end of shift < 0.01 mg/100mL 0.15 mg/100mL
Biological Effect Monitoring	Blood Urine Spirometry Chest X-Rays ECG	Full blood count, liver function Combur 9 + creatinine - - -
Reproductive and Developmental effects		

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METALS: DIAGNOSIS AND INVESTIGATION OF OCCUPATIONAL EXPOSURE

DIAGNOSIS AND INVESTIGATION OF OCCUPATIONAL EXPOSURE TO METALS: A GENERAL REVIEW

Exposure to metals in the workplace present serious and significant health risks. The hazards that metals present are a function of the toxic properties of metals and include the duration, dose and route of exposure, and health history of the individuals exposed to them.

Controlling and preventing metal 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. It is frequently undiagnosed as a result of poor occupational history taking.

OCCUPATIONAL HISTORY TAKING

Obtaining such history does not require detailed knowledge of toxicology. In seeking history the health worker should consider all possible exposures that may occur in the community where the patient lives and/or works.

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

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

CLINICAL EXAMINATION AND INVESTIGATIONS

Medical practitioners do not require special skills to diagnose occupational and environmental health problems. A practical approach on examination and tests is useful in day to day practice.

As most metals 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:

- Full Blood count,
- Urine analysis,
- Kidney function, and
- Liver function tests
- Chest X-ray and pulmonary function, ECG, and allergy testing may be performed where relevant.

The determination of metals in blood, urine, and tissues are used to confirm the diagnosis.
It should be noted that generally each metal produces a constellation of symptoms and a clinical picture unique to the metal [Table I]. The tests required for exposures are metal specific.

TABLE I: THE CLINICAL MANIFESTATION/S OF COMMON METAL EXPOSURES AND THEIR INVESTIGATION

METAL	CLINICAL MANIFESTATION/S	BIOLOGICAL MONITORING
Arsenic	Garlic odor on breath and tissue fluids; "rain drop pigmentation" of temples, eyelids, and neck with hyperkeratosis; encephalopathy; peripheral neuropathy ("glove and stocking" distribution); " Mees" lines (white striae on fingernails); cardiac arrhythmias, hepatic and renal damage; haemolytic anemia in the case of arsine gas exposure; known human carcinogen(skin, lungs); hair loss	Urinary arsenic level is the most reliable indicator of recent exposure to arsenic.Arsenic in hair and fingernails can indicate exposure to high levels in the past 6–12 months.
Aluminium	Chronic obstructive airways disease (Shaver's disease); asthma; multiple fractures; osteomalacia, dementia, proximal muscle weakness; microcytic anemia; dermatitis	Aluminium estimation in urine and serum
Antimony	Dermal effects (antimony spots- pustules and eruptions near sebaceous glands); ECG changes (altered T waves); increased blood pressure; pneumoconioses, chronic bronchitis and emphysema; pleural adhesions; inactive tuberculosis	Urine antimony levels
Cadmium	Proteinuria, ulceration of nose; lose of sense of smell; severe back pain and joint pain (waddling gait); Fanconi's syndrome, hypercalciuria with renal stone formation and osteomalacia, emphysema, prostate and lung cancer,	Urine and blood cadmium levels
Chromium (hexavalent)	Bronchitis; pulmonary fibrosis, chronic asthma, lung and skin cancer, ulceration and perforation of nasal septum; "chrome ulcers" on skin and in nose; nasal polyps, rhinitis; sinusitis	Total chromium in urine Chromium in plasma, whole blood, red cell
Lead	Encephalopathy, anemia, abdominal pain (lead-colic often mistaken for an acute abdomen), nephropathy, foot-drop, wrist-drop, reproductive	Urine and blood Lead levels
Manganese	Severe chemical pneumonia from inhalation of fumes (manganism); CNS effects which include Parkinson-like syndrome, bulbar paralysis, extrapyramidal features, multiple sclerosis	Manganese in blood and urine
Mercury	Nausea, bronchial irritation and erosive bronchitis, gastrointestinal and renal tubular necrosis, metallic taste, gingivostomatitis, intention tremor, neuroasthenia, paraesthesia of extremities and face, ataxia, dysarthria, concentric constriction of fields of vision, behaviorial and personality changes, delirium, hallucinations, nephrotic syndrome, hypersensitivity	Total inorganic mercury in urine Total mercury in blood
Nickel	Irritation of nose and sinuses with loss of smell and perforation of nasal septum, asthma, allergic contact dermatitis ("nickel itch") pulmonary fibrosis, reduced sperm count, nasopharyngeal, lung and gastrointestinal tumours; nephrotoxic effect	Urine nickel
Thallium	Usually starts with gastroenteritis, followed by a peripheral neuropathy, and thereafter hair loss, Alopecia, neurologic, anorexia, nausea, vomiting, diarrhoea followed by constipation, with a burning sensation of the tongue and stomatitis	Urine thallium Hair and nails for thallium estimation
Vanadium	Chemical pneumonitis, Chronic bronchitis, allergic dermatitis, nervous system effects, cardiovascular effects, eye irritation, lacrimation.	Urine vanadium

A NOTE OF THE EFFECTS OF METAL OCCUPATIONAL EXPOSURE ON REPRODUCTION AND FERTILITY

Effects of occupational exposure on the reproductive system of men and women may become manifest as alterations of 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, while a high exposure dose cab result in miscarriage or infertility. Maternal exposure during pregnancy may disturb fetal development by either directly or indirectly interfering with maternal, placental or fetal membrane functions.

Toxic exposures can induce many wide –ranging effects, such as fetal 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 most studied metals are lead and mercury

TABLE II: REPRODUCTIVE AND FERTILITY EFFECTS OF LEAD AND MERCURY

MERCURY	CLINICAL MANIFESTATION/S	BIOLOGICAL MONITORING
Lead	Reduced sperm quality, reduced fertility, fetal loss	Reduced fertility, fetal loss, preterm birth, low birth weight, birth defects, impaired cognitive development
Mercury	Fetal loss	Reduced fertility, menstrual disorders, fetal loss
Nickel	Reduced sperm count	

METAL OCCUPATIONAL CARCINOGENS

It must be noted that the only metals that have been demonstrated in epidemiological studies to have correlated with an increased cancer incidence in humans are nickel, chromium, arsenic, cadmium and beryllium. There is a great lack of human data in this field of occupational disease.

Below is table III that shows the global consensus with regards to the classification of human carcinogens and in table IV the current information available on the different metals with their carcinogenic classification and the organs know to be affected.

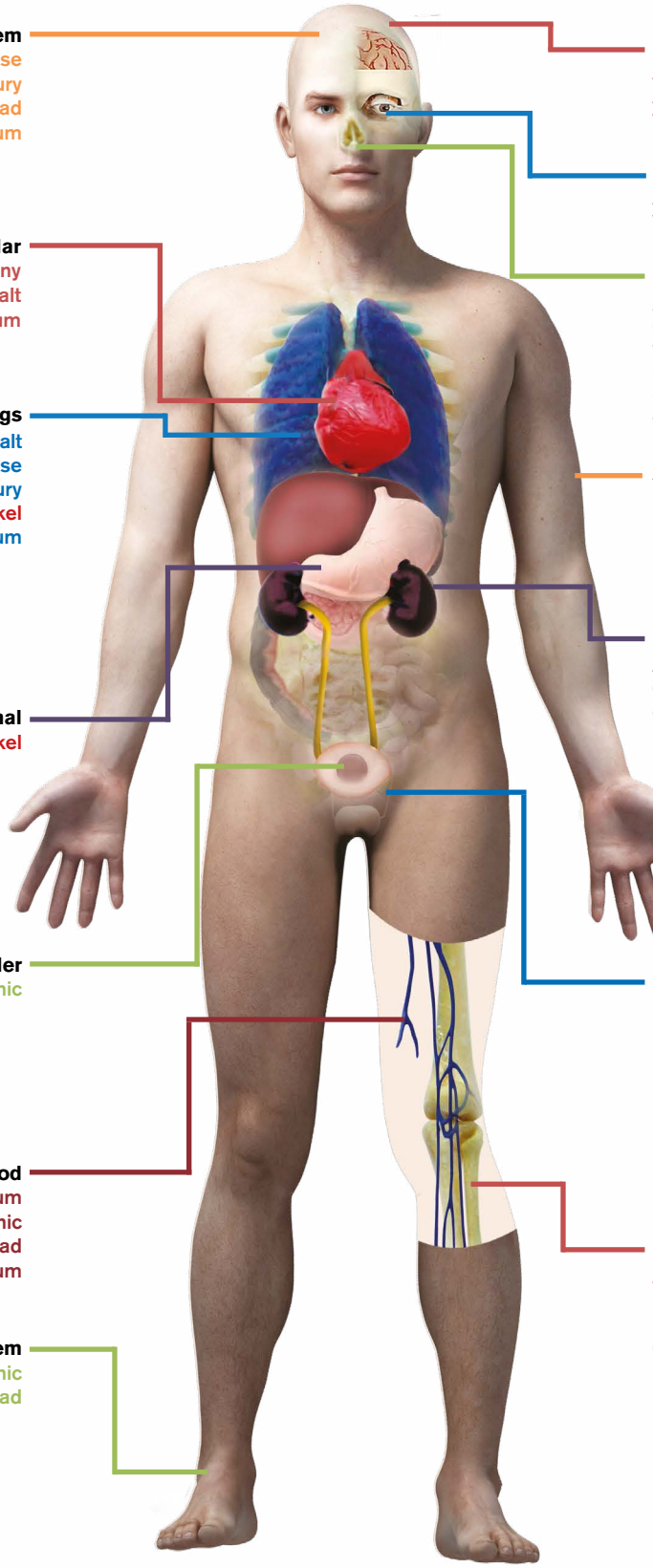
TABLE III: CLASSIFICATION OF METALS AS PER THE INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC)

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

TABLE IV: METAL EXPOSURE, IARC CLASSIFICATION AND AFFECTED ORGAN

METAL	CLASSIFICATION	AFFECTED ORGAN
Arsenic	Group 1	Lung & skin
Aluminium	Not classified	
Antimony	Group 2B	
Cadmium	Group 1	Lung & prostate
CHROMIUM [hexavalent]	Group 1	Nasal cavity ,lung & skin
Lead	Group 2B	
Manganese	Not classified	
Mercury	Group 3	
NICKEL compounds	Group 1	Nasal cavity ,lung & gastrointestinal
Thallium	Not classified	
Vanadium	Group 2B	
Welding fumes	Group 2B	

TARGET ORGANS FOR METAL EXPOSURE



Central nervous system
Manganese
Mercury
Lead
Vanadium

Cardiovascular
Antimony
Cobalt
Vanadium

Lungs
Aluminium
Antimony
*Arsenic
Asbestos
*Cadmium
*Chromium VI
Cobalt
Manganese
Mercury
*Nickel
Vanadium

Gastrointestinal
*Nickel

Bladder
Arsenic

Blood
Aluminium
Arsenic
Lead
Cadmium

Peripheral nervous system
Arsenic
Lead

Hair
Antimony
Arsenic
Thallium

Eye
Antimony
Vanadium

Nasal passages
Arsenic nasal septum
Cadmium
Vanadium
*Chromium VI nasal septum/cavity
*Nickel nasal cavity
Vanadium

Skin
Aluminium
Antimony
*Arsenic
*Chromium VI
Cobalt
Nickel
Vanadium

Kidneys
Arsenic (Arsene)
Cadmium
Chromium VI
Lead
Mercury

Reproductive system
Antimony
Lead
Manganese
Nickel
Mercury
Nickel
*Cadmium-prostate cancer

Bone
Aluminium
Flouride
Lead
Cadmium

*** Carcinogens**

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METAL ALLERGIES IN THE WORKPLACE

Metals have been implicated in causing sensitization and allergic diseases. These patients may present with skin reactions/ dermatitis, eye infections, respiratory symptoms, nasal symptoms, loosening of orthopedic and dental prosthesis-even though 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 on page 70. 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 MELISA (Memory Lymphocyte Immunostimulation Assay) test however does not 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 MELISA test procedure involves the isolation of white blood cells (lymphocytes) from whole blood and then test against allergens chosen according to the patient's occupational history. The blood is then incubated for five days and the lymphocyte reaction is thereafter measured. The level of reactivity is measured as a Stimulation Index (SI). A value over 3 indicates a positive reaction to a given allergen.

MELISA TESTING AT AMPATH

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: 6-8 Citrate tubes

Collection instructions:

- This test is not performed over weekends and must be performed within 24hours 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 FOLLOWING MELISA TESTS ARE AVAILABLE

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

METAL	MNEMONIC
Inorg Mercury	HGIW
Iridium	IRW
Iron	FEW
Lanthanum	LW
Lead	PBW
Manganese	MNW
Methyl Mercury	HGMW
Molybdenum	MOW
Nickel	NIW
Paladium	PDW
Phenyl Mercury	HGPW
Platinum	PTW

METAL	MNEMONIC
Ruthenium	RUTW
Silver	AGW
Thimerosal	THIMW
Tin	SNW
Titanium	TIW
Vanadium	VW
Zink	ZNW
Zirconium	ZRW

ALUMINIUM

Chemical Formula	Al
Synonyms	Aluminium metal, alumna powder, aluminum
CAS number	7429–90–5
Description	Aluminium is a ubiquitous (ever-present) but nonessential metal. Pure aluminium is a light flexible metal with a density of approximately one third that of iron. Aluminium is a good conductor of heat and electricity and easy to weld. When exposed to air or water a thin film of oxide is formed on the surface of the aluminium creating a protective coating that is resistant to corrosion. Aluminium phosphide is a fumigant that releases phosphine and is highly toxic.
Uses	Aluminium is used in alloys with copper, magnesium, zinc, manganese,casting sheet metal foil and welding. Aluminium is also used within antacids and phosphate binders. Aluminium salts are used for antiperspirants and in products used for diaper rash, insect stings, in anti-diarrhea products, vaginal douches and anorectal preparations. Aluminium silicate is found in cosmetics. Aluminium containing food additives are allowed in many food products. Aluminium salts are also used as flocculants during the treatment of drinking water. Aluminium compounds are also an adjuvant in vaccine production.
Metabolism (Includes absorption & elimination)	Ingestion: Inhalation, dermal, ingestion Poorly absorbed via skin, the lungs and intestines. Body burden 30–50 mg. Skeleton 50% and lungs 25% In blood equally distributed between plasma and erythrocytes. Crosses the blood brain barrier but the brain has very low levels compared to other organs. Urine accounts for more than 95% of aluminium excretion. The half life for blood and bone range between one hour and 29 years.
Effects of Occupational Exposure	Long-term occupational exposure to aluminium fumes or fine dusts can lead to accumulation in the body with the skeleton and lungs as main sites. Some studies suggest that occupational exposure to aluminium or aluminium compounds may interfere with neurophysiological and – psychological functions,including cognitive processes. Aluminum-induced pulmonary diseases (aluminosis - Shaver's disease) have been described after inhalation exposure, but they seem to be rare and rather mild. Aluminium has also been implicated in chronic obstructive airways disease and asthma, although all 3 lung diseases have multi-factorial confounders of silica, welding fume and fluorides. Aluminium causes dialysis dementia and osteomalacia in patients on long term dialysis therapy and renal failure. Dialysis dementia comprises central nervous system damage of varying (serum aluminium concentration-dependent) intensity, together with osteomalacia, and microcytic anemia.
Confounders/ Non Occupational Exposure	Aluminium is found throughout the environment, food, medicines, cosmetics, water and exposure can take place through a variety of day to day activities. Contamination potential of samples is high.

OCCUPATIONAL EXPOSURE

Medical Surveillance	**Medical and occupational history**	
	Physical	As per occupational medical program
	Sentinel health events	Respiratory effect (asthma)s, neuropsychological, dialysis associated bone and blood disease, skin – rare contact dermatitis.
Biological Monitoring	**Sample:** Aluminium in serum Aluminium in urine	**Sampling time:** ES (end of shift), EW (end of work week) ES (end of shift), EW (end of work week)
	Reference limits: 1. Aluminium urine Not industrial exposed BEI (Biological exposure index) Occupational exposure to fumes seems to produce higher urinary levels of aluminium than exposure to dust. 2. Aluminium serum Normal Patients on hemodialysis Indicator of Al-induced osteomalacia Clinical signs of Al toxicity	 < 30 ug/g creatinine 150 ug/g creatinine 0–0.2 umol/L 1.6–2.1 umol/L > 3.7 umol/L > 7.4 umol/L
Biological Effect Monitoring	Blood Urine Spirometry Chest X-Rays ECG	Full blood count, urea, creatinine and electrolytes Dipstix Yes Baseline -

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ANTIMONY

Chemical Formula	Sb
Synonyms	Antimony metal, Antimony powder, Antimony oxide, Stibium, Stibine gas
CAS number	7440-36-0
Description	Antimony is a silvery-white, lustrous, brittle, hard metal, scale-like crystals or a dark grey lustrous powder that is found in the earth's crust. It is a non essential metal.
Uses	Antimony isn't used alone because it breaks easily, but when mixed with alloys,it is used in lead storage batteries, solder, sheet and pipe metal, bearings, castings, and pewter. Antimony oxide is added to textiles and plastics to prevent them from catching fire. It is also used in paints, ceramics, and fireworks, and as enamels for plastics, metal and glass. It is incorporated into thermoelectric materials used in nanoparticle technology.
Metabolism (Includes absorption & elimination)	Route of entry: Inhalation, ingestion ,dermal,eye contact Rapidly excreted mainly in urine (four times higher) and faeces. It is excreted in bile after conjunction with gluthathione as well as in urine. A significant proportion of antimony excreted in bile undergoes enterohepatic circulation. Renal excretion is slow. The intestinal absorption of trivalent antimony is lower than the pentavalent compound, but the excretion of the pentavalent compound, which is not bound to erythrocytes, is much faster. Trivalent antimony is mainly bound to erythrocytes, therefore the plasma levels is low. Inorganic trivalent antimony is not methylated in vivo. Antimony has properties and biological activities similar to arsenic, but less toxic.
Effects of Occupational Exposure	Antimony is an irritant of the mucous membranes, eyes, and skin. Severe exposure to antimony trioxide and pentoxide is associated with pulmonary injury where-as antimony trisulphide is considered cardio toxic. Elevated blood pressure and alterations in the T waves have been noted. Acute exposures cause loss of hair, dry scaly skin and weight loss. Chronic exposure causes antimony spots on the skin, mucous membrane irritation, antimony pneumoconiosis(when exposure is to fine antimony dust),chronic bronchitis, chronic emphysema, inactive tuberculosis and pleural adhesions. Mild jaundice has also been reported. It is also described as a haemolytic agent
Confounders/ Non Occupational Exposure	Antimony is found in low levels throughout the environment and food contains very small amounts.

OCCUPATIONAL EXPOSURE

Medical Surveillance	**Medical and occupational history**	
	Physical	As per occupational medical program
	Sentinel health events	Respiratory and cardiac effects, dermatitis and conjuctivitis
Biological Monitoring	**Sample:** Antimony in urine	**Sampling time:** Not critical
	Reference limits: Antimony: creatinine Not industrial exposed BEI (Biological exposure index)	 < 1.6 ug/g creatinine 35 ug/g creatinine
Biological Effect Monitoring	Blood Urine Spirometry Chest X-Rays ECG	ALT, AST, ALP, GGT, full blood count Confirmation of Sb exposure Yes Yes Yes
Reproductive/ Developmental Effects	Non-conclusive effects(menstrual disturbances, spontaneous abortions have been reported) Not classified as a carcinogen	

ARSENIC

Chemical Formula	As
Synonyms	Arsenic Black, arsenic-75, arsenic solid, arsenic metallic, arsenicals, grey arsenic, colloidal arsenic, arsenic gas
CAS number	7440–38–2
Description	Organic and inorganic compounds are white or colorless powders. Arsenic metal is a grey powder.
Uses	• during the manufacture of insecticides, weed killers and fungicides; during use as a wood preservative • during use in the manufacture and handling of calcium arsenate; during use in the manufacture of electrical semi-conductors, diodes and solar batteries • during use as a bronzing or decolorizing addition in glass manufacturing • during use in the production of opal glass and enamels
Metabolism(Includes absorption & elimination)	Route of entry: inhalation, ingestion, skin and eyes. Exposure is mainly to inorganic arsenic. Absorbed arsenic is widely distributed in the body with the highest levels being found in hair, nails and skin and skeleton where the concentration decreases slowly over time. The main route of excretion is through the kidneys and most eliminated within a week. Inorganic arsenic is bio transformed after inhalation or ingestion and detected in urine as (Momonethyl Arsonic Acid MMA) Cacdylic Acid (DMA) and unchanged inorganic arsenic.
Effects 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, anemia, 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 ototoxin, can cause hemolysis, gastrointestinal disturbances, mild jaundice and renal dysfunction. Inorganic arsenic is a known human carcinogen. (Category 1). May cause reproductive disorders such as increasing risk of spontaneous abortions as arsenic can cross the placenta. Prolonged exposure may lead to hair loss
Confounders/ Non Occupational Exposure	Seafood intake is a major confounder as well as drinking water contamination (up to 100 mg/L in urine)It is recommended that shellfish should not be consumed two to three days before urine testing(arsenic levels can increase to >1mg/L)

OCCUPATIONAL EXPOSURE

Medical Surveillance	**Medical and occupational history**	
	Physical	As per occupational medical program
	Sentinel health events	Skin, lung, bladder and lymphatic, cancer, peripheral neuropathy, skin lesions,liver dusfunction haemolytic anemia (arsine) Kidney failure (arsine)
Biological Monitoring	**Sample:** Total arsenic in urine	**Sampling time:** EW (end of work week)
	Reference limits: Not industrial exposed BEI (Biological exposure index) (In the absence of the consumption of seafood for 2 days prior to specimen collection)	< 30 ug/g creatinine 50 ug/g creatinine (total arsenic)
Biological Effect Monitoring	Blood Urine Spirometry Chest X-Rays ECG	Full blood count differential , ALT, AST, GGT, ALP, bilirubin, urea, creatinine Dipstix (proteinuria,hematuria) Yes Yes Yes in selected cases
Reproductive/ Developmental Effects	Can cause spontaneous abortions	

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CADMIUM

Chemical Formula	Cd
Synonyms	Cadmium chloride, Cadmium Oxide, Cadmium Sulphide, "Cadmium and compound" includes Cadmium dust and salts. For compounds of Cadmium.
CAS number	7440–43–9
Description	Chemical element that occurs naturally in the earth's crust. Pure cadmium is a soft-silver white metal. Some cadmium compounds are extremely flammable.
Uses	Alkaline storage batteries, alloys, cadmium vapor lamps, catalyst, ceramics, dyes, electroplating, welding, engraving, glass colouring, metalizing, nuclear reactors, paints, plantings, photometry, silver soldering, welding cadmium alloys.
Metabolism (Includes absorption & elimination)	Route of entry: inhalation, ingestion via acidified food and drinks in cadmium-plated containers. Absorbed via inhalation, ingestion and skin. In normal individuals, two to six percent of ingested and 20–50% of inhaled cadmium will be absorbed and transported to the liver. The liver detoxifies it by combining it with low molecular weight protein, metallothionine. Elimination through the kidneys is very slow, since much of the cadmium-protein complex is reabsorbed and stored. Liver and kidneys contain approximately 50% of the total body burden, the balance being found mainly in the pancreas, salivary glands, thyroid and intestinal mucosa. The metal can be absorbed by its aerosol.
Effects of Occupational Exposure	Inhalation: usually four to 10 hours post exposure – irritation of throat, metallic taste, cough, tightness of chest, chest pain, dyspnoea, chills, sweating, aching in extremities + back, headache, dizziness. Cadmium fumes can result in a severe pneumonitis with pulmonary oedema and death. Delayed reaction may occur one to seven days later – severe dyspnoea, wheezing, chest pain and tightness and coughing, weakness, anorexia, nausea, abdominal pain and diarrhoea, prostration, pneumonitis and pulmonary oedema. Chronic effects: loss of sense of smell, ulceration's nose, shortness of breath (emphysema), kidney damage, mild anemia and prostate cancer (not fully explored epidemiologically) Fanconi's syndrome, hypercalciuria with renal stone formation and osteomalcia. Severe back pain and joint pain. Ingestion: Symptoms usually occur in 15–30 minutes: salivation, nausea, vomiting, abdominal pain, diarrhoea, dizziness, unconsciousness. (Itai-itai disease, Japanese women ingestion of Cd contaminated rice, beans and water produced; severe back pain and joints waddling gait, osteomalacia spontaneous fractures and renal failure).
Confounders/ Non Occupational Exposure	Polluted environments can increase cadmium concentration in urine and blood. Cadmium is widely spread in nature and is found wherever zinc is found. It is found in foodstuffs such as oysters, beef kidney, rice in some areas and shellfish. Smoking – each cigarette contains 1–2 µg of cadmium. Age – cadmium concentration in urine and blood increased with age.

OCCUPATIONAL EXPOSURE

Medical Surveillance	**Medical and occupational history**	
	Physical	As per occupational medical program
	Sentinel health events	Kidney Damage, Fanconi syndrome, Renal stone, Emphysema, Prostate & Lung cancer
Biological Monitoring	**Sample:** Cadmium in urine Cadmium in blood	**Sampling time:** NC (not critical)
	Reference limits: 1. Cadmium urine Not industrial exposed BEI (Biological exposure index) 2. Cadmium blood Not industrial exposed BEI (Biological exposure index)	 < 3.0 ug/g creatinine 10 ug/g creatinine < 5 ug/l 10 ug/l
Biological Effect Monitoring	Blood Urine Spirometry Chest X-Rays ECG	Liver, kidney, full blood count, Dipstix & specific proteinuria Yes Yes -
Reproductive/ Developmental Effects		

CHROME VI

Chemical Formula	Cr (VI)
Synonyms	Chromium Acid; Sodium dichromate; Potassium dichromate; Chromium trioxide; Lead chromate; Calcium chromate; Zinc chromate
CAS number	13333–82–0
Description	Cr is a lustrous, silvery metal. It exists in oxidation states - of II and III states are most common in the workplace. Chromium Trioxide (Cr(III)) is a powerful oxidizing agent and is very corrosive. Hexavalent chromium Cr (VI) is produced in industrial processes when Cr(III) is heated in the presence of mineral bases and atmospheric oxygen (for instance during metal finish-ing processes.) Essential trace element.
Uses	Cr(VI) is used, produced and liberated in chemical intermediates, in the textile industry (in dyes), silk treating, printing, pigments, the leather industry (tanning), photographic fixing, in catalyst for halogenation reactions, in alkylation, chrome electroplating, stainless steel welding, spray painting, cement production and use, in the catalytic crack-ing of hydrocarbons, in fuel additives and in ceramics
Metabolism (Includes absorption & elimination)	Route of entry: inhalation, skin and poorly by the gastro-intestinal tract. (Chromium III is poorly absorbed by all routes). Cr VI is well absorbed through the skin and poorly by the gastro-intestinal tract. It is rapidly changed to Cr III when taken orally. Absorbed Cr VI is rapidly reduced to Cr III (by erythrocytes) and bound to hemoglobin. Cr VI is therefore not detected in urine. The efficacy of detoxification by erythrocytes varies from individu-al to individual. The highest level of Cr is found in liver, kidney, spleen and lungs. Studies of accumulation in the body suggested a plasma pool in equilibrium with three compartments: fast (0.5–12 hours), medium (1–14 days) and slow (3–12 months). Half-life for elimination 7 hours, 15–30 days and 3–5 years. Elimination is mainly by the kidneys, while some excretion also occurs via bile and faeces. Minor routes of excretion include milk, sweat, hair and nails.
Effects of Occupational Exposure	Cr (VI) adversely affects the skin, respiratory tract and kidneys. Skin allergies, chrome ulceration (skin and nose – so called chrome ulcers) and carcinogenetic in the skin and lungs (results from the insoluble nature of Cr(VI)).
Confounders/ Non Occupational Exposure	Dietary intake and smoking are sources as chromium is a naturally occurring metal and a required trace element for humans. Municipal water supplies and ambient air frequently contain traces of chromium. Increased concentrations of chromium have been seen in the urine of populations living near metal refining industries. Urine Cr may also increase in exercise, past chromium exposure, Intake of beer and wine, mineral supplements, and in diabetes.

OCCUPATIONAL EXPOSURE

Medical Surveillance	**Medical and occupational history**	
	Physical	As per occupational medical program
	Sentinel health events	Skin & Respiratory events ,Nose septum Cancers of skin & lung
Biological Monitoring	**Sample:** Total Cr in urine Cr whole blood	**Sampling time:** ES (end of shift), EW (end of work-week)
	Reference limits: 1. Chromium urine Not industrial exposed: BEI (Biological exposure index) 2. Chromium whole blood Not industrial exposed Toxic levels not established.	 0.4–4.0 ug/g creatinine 30 ug/g creatinine 0–1.56 ug/L
Biological Effect Monitoring	Blood Urine Spirometry Chest X-Rays	Full Blood Count,Liver function, Urea, Creatinine, Electrolytes Dipstix Yes Yes
Reproductive/ Developmental Effects	Limited information on the reproductive effects of Cr VI in humans exposed by inhalation suggest that exposure to Cr VI may result in complications during pregnancy and childbirth	

COBALT

Chemical Formula	Co
Synonyms	Cobalt metal dust or Cobalt metal fume and Cobalt compounds
CAS number	7440–48–4
Description	Cobalt is a silver-gray to solid black odorless hard metal found throughout the environment
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
Metabolism (Includes absorption & elimination)	Route of entry: inhalation, skin and gastro-intestinal tract. Cobalt is mainly absorbed by inhalation and ingestion. After inhalation the cobalt deposit is based on their aerosol characteristics and absorption is relat-ed to its solubility. Insoluble particles are cleared by phagocytosis or mucocili-ary transport and thus have a low systemic absorption. More soluble forms may enter the blood stream. Absorption of ingested cobalt varies with the amount given. Nutritional status and solubility of the cobalt is also an important factor in absorption. Soluble cobalt that is ingested is mainly transported via blood to liver and kidneys and excreted in urine. Faecal elimination is the primary method of excretion insoluble cobalt. Excretion after dermal exposure is in urine.
Effects of Occupational Exposure	Insoluble cobalt in lungs may cause irritation of the lungs, asthma, pneumonia, wheezing and so called "hard metal lung disease" which is a type of lung fibrosis. Skin exposure can cause skin irritation and contact dermatitis. Soluble cobalt that is absorbed in blood and excreted by urine
Confounders/ Non Occupational Exposure	Dietary intake – mostly in inorganic Vitamin B12

OCCUPATIONAL EXPOSURE

Medical Surveillance	**Medical and occupational history**	
	Physical	As per occupational medical program
	Sentinel health events	Contact dermatitis, Lung disease, Cardiovascular
Biological Monitoring	**Sample:** Total Cobalt in urine Cobalt whole blood	**Sampling time:** ES (end of shift), EW (end of work-week)
	Reference limits: 1. Not industrial exposed BEI (Biological Exposure Index) 2. *Not industrial exposed When the average exposure levels are 0.1 and 0.5 mg/m^3, the estimated blood levels are 10 and 25ug/L respectively. Biological Exposure Index(BEI) is UNKNOWN	 0.6–1.6 ug/g creatinine 50 ug/g creatinine end of shift,end ofworkweek 0.5–3.9 ug/l
Biological Effect Monitoring	Blood Urine Spirometry Chest X-Rays ECG	 Yes Yes Yes
Reproductive/ Developmental Effects	Limited information on the reproductive effects of Cobalt in humans exposed	

FLUORIDE

Chemical Formula	F
Synonyms	Including metallic fluorides, hydrofluoric acid and fluorine
CAS number	7664–39–3
Description	Fluorides are a diverse group of substances, the physical properties of which are dependent on the individual compound. Metallic fluorides are solids of variable solubility in water. Salts of monovalent metals are fairly soluble; salts of divalent metals are sparingly soluble. Hydrogen fluoride, or hydrofluoric acid, is a reactive gas, which readily dissolves in water, reacts with glass and is corrosive. Fluorine is a highly reactive gas, attacking all elements with the exception of oxygen and nitrogen and forming both covalent and electrovalent bonds with metals and other elements
Uses	Mining of minerals, production of aluminium and steel, brick and refractory, fluxes in welding, hydrofluoric acid and fluorine production and uses.
Metabolism (Includes absorption & elimination)	Inorganic fluoride and elemental fluoride are not metabolized in the body, however, organic fluorides are metabolized to some extent and release fluo-ride ion into the body. Fluorides which are absorbed are deposited mostly in the bone. Soluble fluoride is easily absorbed via the lungs and gastrointestinal tract and is taken up by the blood and rapidly distributed to the entire body. About 75% of fluorides in the blood are found in the plasma. In addition to distribution to organs, such as liver, heart and lungs, the principal site of deposition is the bone where 99% of all deposited fluorides accumulate. Binding to bone is re-versible and is dependent on plasma concentrations. The kidney is the major route of elimination with about 50% of the daily intake appearing in the urine. Approximately five to 30% of the amount ingested is excreted in the faeces. Sweat may be a significant route of elimination in hot environments. Fluoride is eliminated in mother's milk. Skin is not a route of absorption unless associated with burns.
Effects of Occupational Exposure	Acute: Fluoride exposure rarely manifest in other ways than irritation. Inhalation: Mainly as dust. Irritation of nose, throat, eyes and respiratory tract-laryngeal spasm, oedema and haemoptysis. Skin contact: hydrogen Fluoride (HF) can cause deep and excruciatingly pain-ful skin burns. Eye contact: Causes severe burning, irritation. Hydrogen fluoride may cause deep seated burns of the eyes. Ingestion: Severe burning and/or perforation of the digestive system which may lead to death. Chronic: Fluorosis of chronic fluoride intoxication has been widely reported to produce fluoride deposition in skeletal tissues of both animals and man. The symptoms found include increased radiographic bone opacity, formation of blunt excrescence of the ribs and calcification of intervertebral ligaments. Dental mottling is also found in cases of fluorosis. Also results in skin scarring, pulmonary fibrosis, cardiac arrhythmias following blood calcium disturbances, Fluoride has a high affinity for calcium often leading to hypocalcaemia. Carcinogenicity: No specific data is available. Teratogenicity: There is inconclusive data Reproductive toxicity: Insufficient information Mutagenicity: Insufficient data
Confounders/ Non Occupational Exposure	Dietary intake must be considered because fluoride is ubiquitous. Most water supplies contain fluorides. Fluoride levels in urine can be temporarily elevated after: • consumption of tea or salt water fish • administration of fluoride containing drugs such as a fluoride supplement (Fluoritabs, Theraflur), decongestants such as fluorvite. • swallowing or using preparations for prevention of dental caries (mouth-washes, pills, toothpaste's, lozenges) Exposure to organic fluoride – containing chemicals such as chlorinated fluoro carbons (Freons), fire extinguishing agents, insecticides, fluorosteroids and some drugs such as inhalation anaesthetic agents or ointments (Flurone). Regions near phosphate and aluminium plants. Some household products and rust removers or naval jelly.

OCCUPATIONAL EXPOSURE

Medical Surveillance	**Medical and occupational history**	
	Physical	As per occupational medical program
	Sentinel health events	Fluorosis – teeth and pelvic ligaments
Biological Monitoring	**Sample:** Fluoride in urine	**Sampling time:** PS (prior to shift) ES (end of shift)
	Reference limits: Not industrial exposed BEI (Biological exposure index)	 0.3 - 2.2 mg/g creatinine 3.0 mg/g creatinine prior to shift (1) 10.0 mg/g creatinine at end of shift (2)

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Biological Effect Monitoring	Blood Urine Spirometry Chest X rays and other ECG	Serum urea, creatinine and electrolytes, calcium, magnesium, phosphate Dipstix for albuminuria Yes for dust exposure Chest Pelvis baseline may be of use Spine-poker back Increased radiology density of joints in hands, feet, knees Yes (changes due to hyperkalaemia and hypocalcaemia)
Toxicity	Numerous instances of acute fluoride poisoning have occurred as a result of accidental or intentional ingestion or inhalation of fluorides. One mass poisoning involved 263 persons, 47 of whom died following in advertant addition of sodium fluoride to food.	

LEAD

Chemical Formula	Pb
Synonyms	Lead flake, C.I. Pigment Metal 4, Pb-S 100
CAS number	7439–92–1
Description	Lead is a naturally occurring bluish-grey metal found in small amounts in the earth's crust. It is used in the production of some types of batteries, ammunition, metal products (such as solder and pipes) ceramic glazes and paint. Chemicals containing lead, such as tet-raethyl lead and tetramethyl lead are used as gasoline additives. Lead can be used for X-ray and atomic radiation protection.
Uses	• Manufacturing of sheeting or pipes where flexibility and resistance to corrosion are required • Cable sheathing • Mining – lead ore • Manufacturing shut metal and foils • Used as a shield for ionising radiation • Paints, enamels and glazes; glass and ceramic industry • Printing industry – typecasting, remelting of type metal • Ingredient in solder • Welding, burning and cutting metal structures • Melting and pouring of lead alloys • Ammunition procedures • Motor industry • Antiknock additives in petrol (tetra alkyl lead) • Car batteries
Metabolism (Includes absorption & elimination)	INORGANIC LEAD Inorganic lead is the most common form of lead found in the work place. It can be absorbed as a fume or as a dust. Fumes are formed when lead is heated to temperature greater than 560°C. The most common way workers are exposed to lead is to inhale workplace air containing lead hence inhalation is the most important route of absorption of lead. It can, however be absorbed by ingestion, which occurs when a worker eats or smokes in a lead contaminated area. Only 10% of swallowed lead is absorbed into the body – the remaining 90% is excreted, but 40% of the amount inhaled is absorbed via the lungs. Of the inorganic lead which is absorbed into the blood, 90% will be deposited into the body tissues and bone structures while 10% will be excreted via the kidney (predominantly) sweat, faeces and breast milk. Biological half life is 28–36 days. If the exposure of the worker to lead decrease the lead will, over time, slowly be released from the bones and tissues, but if exposure remains high then the lead will continue to be deposited. For this reason workers removed from a lead area can continue to have high blood lead levels as the lead is slowly released back into the blood. The taking of blood lead samples will then indicate how much lead is circulating in the body TETRA ALKYL LEAD Tetra alkyl lead and tetra methyl lead are liquid compounds of lead. In some countries still used as antiknock agents. May be absorbed through all the usual routes. Excretion takes place both in urine and faeces. Lead in faeces is derived mainly from non-absorbed lead. Lead in urine therefore provides a much better measure of absorption.
Effects of Occupational Exposure	Abdominal Non-abdominal pain Constipation Vomiting Tiredness Nerve tingling Psycological symptoms Diarrhoea Severe symptoms usually develop at blood lead levels of 70ug/100mL. These include:- Anemia Interference occurs with hemoglobin synthesis as lead inhibits the functioning of the enzymes required to synthesise Hemoglobin. Changes in enzyme levels occur. CNS effects; such as forgetfulness, irritability, headache, peripheral nerve functions harmed should be evaluated on a regular basis Kidney damage Reproductive damage - male and female sterility, abortion and damage to foetus. Colic (very intense periodical abdominal cramps) – particularly in the first two years of exposure Damage to motor nerves (e.g. wrist drop) Hypertension Encephalopathy: severe headache, convulsions, coma delirium and death. EPA classified lead as a probable human carcinogen (group B2).

Confounders/ Non Occupational Exposure	• Contaminated domestic water • Battery leakage • Newspaper printers • Serving of food in lead casserole dishes • Home distilled wine and whisky • Hobbies - lead glass windows; painting • Ingestion of containing lead herbal medicines • Use of cosmetics containing lead • Soldering • Sniffing of petrol fumes • Ingestion of contaminated foods • Home renovations • Natural environment - air, water and soil • Exhaust pipe emissions • Smoking

OCCUPATIONAL EXPOSURE

Medical Surveillance	**Treatment of "LEAD POISONING"** 1. Withdrawal of the worker from a respiratory zone and lead area is usually sufficient. 2. Removal from workplace as indicated in the Lead Regulations (see reference limits table) 3. Examination of employee by Occupational Health practitioner and treatment as indicated. 4. Further blood/urine lead measurements must be taken. 5. Employee can only return to lead area provided lead levels decrease as indicated in Lead Regulations. Health practitioner certifies in writing he or she is fit to return. 6. The incident must be investigated in accordance with state regulations to implement measures to control exposure of worker to lead. 7. Lead poisoning is a notifiable disease under the Occupational Health and Safety Act, and is compensable under the Workman's Compensation Act.	
Biological Monitoring	**Sample:** Tetra alkyl lead: Urine Pb Inorganic lead: Blood Pb	**Sampling time:** DS (during shift) – the 1st sample to be taken within six months of commencement of employment) DS (during shift) – the 1st sample to be taken within 14 days of commencement of employment)
	Reference limits: **1. Tetra alkyl lead: Urine Lead** Lead Regulations 2001 OHS Act 1993 – Men: Urinary lead (ug/L) < 120 120–149 > 150 < 130 Lead Regulations 2001 OHS Act 1993 – Women not capable of procreation: Urinary lead (ug/L) < 120 120–149 >150 <130 Lead Regulations 2001 OHS Act 1993 – Women capable of procreation: Urinary lead (ug/L) < 65 > 75 < 65	 Maximum intervals between tests 6 weeks 1 week Removal from workplace Reinstatement in workplace Lead Regulations 2001 OHS Act 1993 Maximum Intervals between tests 6 weeks 1 week Removal from workplace Reinstatement in workplace Maximum Intervals between tests 3 monthly Removal from workplace Reinstatement in workplace

Biological Monitoring (Continued)	**2. Inorganic lead: Blood Lead** Lead Regulations 2001 OHS Act 1993 – Men: Blood Lead (ug/100 mL) < 20 20–39 40–59 60–70 > 60 < 50 Lead Regulations 2001 OHS Act 1993 – Women not capable of procreation: Blood Lead (ug/100 mL) < 20 20–39 40–59 60–70 65 and over <55 Lead Regulations 2001 OHS Act 1993 – Women capable of procreation: Blood Lead (ug/100 mL) 40 and less >40 <30	 Maximum Intervals between tests 12 months 6 months 3 months According to discretion of Occupational Health Practitioner Remove from workplace Reinstatement in workplace Maximum Intervals between tests 12 months 6 months 3 months According to discretion of Occupational Health Practitioner Remove from workplace Reinstatement in workplace Maximum Intervals between tests 3 months removal from workplace reinstatement in workplace
Biological Effect Monitoring	Blood Urine Psychological/Psychiatric tests Neurological tests ECG	HB (inorganic lead), FBC & ESR, Liver function Kidney function Yes Yes -
Reproductive/ Developmental Effects	Reproductive damage - male and female sterility, abortion and damage to foetus.	

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MANGANESE

Chemical Formula	Mn
Synonyms	Manganese dioxide, Manganese tetroxide, Manganese chloride, "Manganese and compounds" includes Manganese dusts and salts.
CAS number	7439–96–5
Description	Mn compounds exist naturally in the environment as solids in the soils and small particles in the water. The metal is grey-white, resembling iron, but is harder and very brittle.
Uses	Uses of Mn compounds includes: alloys with steel, aluminium and copper, dry cell batteries, glass manufacturing, colouring agent in bricks and pottery, manufacture of fireworks and matches, used in fertilizers, fungicides and may be used during the production of unleaded gasoline.
Metabolism (Includes absorption & elimination)	Route of entry: Inhalation and ingestion. Mn is absorbed through the epithelium of the gastrointestinal and respiratory tracts. For occupationally exposed persons, the lung constitutes the main route of manganese uptake. Mn deposited in the lungs is readily absorbed, where only 3–5% of ingested manganese is absorbed.
Effects 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 syste 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 decreasd libido,impotence and decreased fertility may occur. Acute intoxification 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
Confounders/ Non Occupational Exposure	Mn is a very common compound that can be found everywhere on earth. The uptake of manganese by humans mainly takes place through food. Foodstuffs that contain the highest concentrations are grains, rice, soya, spinach, beans, eggs, nuts, olive oil, green beans and oysters. After absorption manganese is transported through the blood to the liver, kidneys, pancreas and the endocrine glands.

OCCUPATIONAL EXPOSURE

Medical Surveillance	**Medical and occupational history**	
	Physical	As per occupational medical program
	Sentinel health events	Neurological, respiratory effects
Biological Monitoring	**Sample:** Manganese in urine Manganese in blood	**Sampling time:** ES (end of shift), EW (end of work-week)
	Reference limits: 1. Manganese urine Not industrial exposed BEI (Biological Exposure Index) is UNKNOWN Urine Mn results do not correlate with signs or symptoms of Mn toxicity, and are only indicative of exposure. 2. Manganese in blood Not industrial exposed	< 4.5 ug/g creatinine 4.2–16.5 ug/L
Biological Effect Monitoring	Blood Urine Spirometry Chest X-Rays	Full blood count & diff, urea, creatinine and electrolytes, Iron, ALT, AST, Gamma GT Dipstix Yes Yes
Toxicity	Mn is mainly absorbed through the lungs. The gastrointestinal absorption is low (3% on average) it is controlled by homeostatic mechanisms and reduced by concomitant ingestion of calcium. Absorption is higher in females than in males probably because of the sex difference in iron body levels. In blood, manganese is mainly present in red blood cells in which manganese concentration is about 25–fold higher than in serum. Liver is the main site of storage but manganese accumulates also in the kidney and brain, preferentially in the corpus striatum and the globus pallidus where it exerts its main toxic effect following chronic exposure. Mn concentrations in a patient with chronic exposure poisoning were elevated in blood. (75mg/L) but normal in urine. Occupational exposure to Mn is more frequently encountered. CNS symptoms manifestation may only be present after a year of exposure.	

MERCURY (INORGANIC)

Chemical Formula	Hg
Synonyms	"Mercury and Compounds" includes vapor, alkyl, aryl and inorganic mercury compounds
CAS number	7439–97–6
Description	Silver white metal in liquid state at room temperature. Occurs in three oxidation states – Hg^0, Hg^{2+} Hg^{2+}
Uses	Mining, smelting, refining, batteries, gold ore extraction, chloralkaline industries, laboratories, dental amalgams, as part of instrumentation (pressure, mechanism, vacuum pumps, etc.)
Metabolism (Includes absorption & elimination)	Route of entry: inhalation (80% inhaled is absorbed), through the skin and through ingestion blood 50/50 in plasma and red blood cells, distributed mainly to the kidneys (T½ 2 months) and brain (T½ years) Excretion: urine (T½ 40 days 35–90), faeces, glands (salivary, lachrymal, sweat) and exhaled air.
Effects of Occupational Exposure	Acute: Following intense exposure to elemental Hg vapor, lung, damage (bronchial irritations, erosive bronchitis, diffusive interstitial pneumonitis) occurs, as do gastrointestinal and renal tubular necrosis after ingestion of mercuric mercury. Long term Exposure: Element Hg can affect nervous system and kidney, chronic exposure to mercuric Hg causes renal tubular damage. Immunological-based glomerulonephritis can occur. The characteristic tremor may begin in the fingers, eyelids, lips or tongue. Slight tremor has been considered as an early sign of metallic Hg vapor exposure. The tremor is usually accompanied by severe behavioural and personality changes, memory loss, increased excitability, and in severe cases, delirium and hallucinations. This constellation of symptoms is called mercurial erethism. Renal manifestations of Hg exposure range from proteinuria at low exposures through the nephrotic syndrome. Sub clinical effects on the kidney have been 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. The significance of these two enzymes contained in intracellular lysosomes to current and future health o f the worker is uncertain.
Confounders/ Non Occupational Exposure	Hg is ubiquitous in the environment and exposure is via food, air and drinking water. Medical and domestic use of Hg – local disinfectants, skin – lightening soap old broken mirrors. Release of Hg from dental amalgams. Penicillin intake.

OCCUPATIONAL EXPOSURE

Medical Surveillance	**Medical and occupational history**	
	Physical	Neurological, Psychological
	Sentinel health events	Neuro/Psychiatric, kidney, lung, reproductive
Biological Monitoring	**Sample:** Total inorganic Hg in urine Hg in blood (Methyl Hg)	**Sampling time:** Prior to shift ES (end of shift), EW(end of work week)
	Reference limits: 1. Hg in urine Not industrial exposed BEI (Biological Exposure index) It is recommended that urine Hg: creatinine ratios be repeated 3 monthly in case of exposure. 2. Hg in blood Not industrial exposed BEI (Biological Exposure index)	 < 2.8 ug/g creatinine 35 ug/g creatinine < 10 ug/L 15ug/L
Biological Effect Monitoring	Blood Urine Spirometry Chest X-Rays ECG	 Dipstix Microscopy/biochemistry (protenuria) Low and high molecular weight proteins Renal tubular enzymes Yes If clinically indicated No

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NICKEL

Chemical Formula	Ni
Synonyms	Metallic nickel, nickel oxide, nickel subsulfide, nickel carbonyl
CAS number	7440–02–0
Description	Ni is naturally present in the environment. It is a hard, ductile, silvery white metal. It is produced by mining and refining sulphide and oxide ores. Ni is also recycled from scrap metal. Metallic Ni exists either by itself or with other metals. Insoluble Ni is combined with other substances, usually sulphur or oxygen. The combination will not dissolve in water. Insoluble Ni is found mining, refining and welding. Soluble Ni, the combination of Ni and other chemicals, will dissolve in water. Electroplating and Ni refining are industries where soluble Ni is present. Ni Carbonyl, this is a gas formed during refining of Ni.
Uses	• mining and refining of Ni ores • manufacturing 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. • manufacturing products from stainless steel • recycling, handling or using these products • electroplating and electroforming • welding
Metabolism (Includes absorption & elimination)	Routes of Entry: Inhalation ,skin, ingestion Inhalation involves: • Dust (relatively insoluble nickel compounds) • Aerosols derived from solutions (soluble nickel) • Gaseous Ni (usually nickel carbonyl) The most hazardous route of exposure is by inhalation. If soluble Ni is inhaled, it dissolves and moves through the body going mostly to the kidneys and liver. Most absorbed Ni is rapidly eliminated in the urine with little or no effect on the kidney and its acute toxicity is low. Insoluble and metallic Ni remains in the lungs. Therefore, the long-term carcinogenic potential of Ni salts and compounds, and particulate Ni retained in the lungs and upper airways is the impetus for bio monitoring. Workers may also come into skin contact with Ni. Only 5% of Ni which is ingested is absorbed by the body. Skin absorption is a major human exposure route with a large population suffering from contact dermatitis and nickel allergy. Ni half life for blood is one hour and urine between 19.9 and 26.7 hours. Lung clearance can take several months.
Effects of Occupational Exposure	General systemic Effects: Headache, vertigo, nausea, vomiting, nephrotoxic effect and pneumonia followed by pulmonary fibrosis. Respiratory Effects: 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. Skin Effects: Skin contact can produce allergic contact dermatitis or “nickel itch”. This is the most common health problem caused by exposure to Ni. This skin allergy, will affect people both at and away from work. Cancer: 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.
Confounders/ Non Occupational Exposure	Food & water intake (legumes, spinach, lettuce and nuts contain more Ni) Dietary intake can be as high as 200–300 mg/day and oral intake from leaching of Ni from cooking utensils and water piping as high as 1 mg/day. Tobacco smoking can increase levels of Nil.

OCCUPATIONAL EXPOSURE

Medical Surveillance	**Medical and occupational history**	
	Physical	As per occupational medical program
	Sentinel health events	Acute effects, respiratory, skin , lung and gastrointestinal tumours, reduced sperm count
Biological Monitoring	**Sample:** Ni in urine	**Sampling time:** (ES) end of shift
	Reference limits: Not industrial exposed BEI(Biological Exposure Index)	 < 3.0 ug/g creatinine 35 ug/g creatinine
Biological Effect Monitoring	Spirometry Gastrointestinal tract	Yes As per OMP decision
Reproductive/ Developmental Effects	Animal carcinogen. Review IARC website for further up to date information.	

VANADIUM

Chemical Formula	V
Synonyms	Vanadiumpentafluoride, dichloride, trichloride, pentoxide
CAS number	7440–62–2
Description	V is a compound that occurs in nature as a white-to-gray metal, and is often found as crystals. Pure vanadium has no smell. It usually combines with other elements such as oxygen, sodium, sulfur, or chloride. V and V compounds can be found in the earth's crust and in rocks, some iron ores, and crude petroleum deposits. Vanadium pentoxide (VO) is the major commercial form of vanadium.
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 depolarizers • Mining and processing of V-containing ores; extraction from slag • Cleaning and maintenance of furnaces, boilers, and gas turbines
Metabolism (Includes absorption & elimination)	Since V is poorly absorbed from the gastrointestinal tract, inhalation exposures potentially pose the greatest risk. 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. The main route of excretion is the urine with a half life of 15–40 hours. (87.6% in faeces and 12.4% in urine) A 3 compartmental model for V is proposed with half lives of 1.2 hours, 26 hours and 10 days.
Effects of Occupational Exposure	The health hazards associated with exposure to V are dependent on its oxidation state. This product contains elemental V. Elemental V could be oxidized to VO during welding. The pentocide form is more toxic that the elemental form. Acute exposure: The signs and symptoms of acute exposure to VO dust include irritation of the nose and throat; clear or bloody discharge from the nose; conjunctivitis, substernal chest pain; productive and persistent cough; shortness of breath, wheezing, and diffuse or focal chest sounds (chemical pneumonitis); greenish discoloration of the tongue; and itching of the skin. Chronic exposure: The signs and symptoms of chronic exposure to VO dust include chronic, productive cough; increased mucus production; shortness of breath; fatigue; chronic bronchitis; and allergic dermatitis. Nervous system, hematological and cardiovascular effects have been described in various publications with no clear evidence accepted. Animal studies also indicate liver, kidney, mutagenic, carcinogenic and reproductive effects.
Confounders/ Non Occupational Exposure	Food is the major source of exposure to V in the general population. V in air from fossil fuels is possible. Coal and fuel oils may contain significant levels and home burning may increase exposure. Water is not a significant source of Vanadium.

OCCUPATIONAL EXPOSURE

Medical Surveillance	**Medical and occupational history**	
	Physical	As per occupational medical program
	Sentinel health events	Pneumonitis, Bronchitis, Dermatitis, CNS effects,
Biological Monitoring	**Sample:** Vanadium in urine	**Sampling time:** ES (end of shift), EW (end of work-week)
	Reference limits: Not industrial exposed BEI (Biological Exposure Index)	 0.1–0.2 ug/g creatinine 50 ug/g creatinine
Biological Effect Monitoring	Blood Urine Spirometry Chest X-Rays ECG	As per OMP decision Kidney function Yes Baseline As per OMP decision
Reproductive/ Developmental Effects	Positive animal studies Access IARC website for updates regularly	

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REGISTERING A NEW CLIENT

PROCEDURE FOR AN AMPATH MARKETER TO FOLLOW

- Obtain information from the client, e. G. Tests required, frequency, number of tests specimen collection and courier service's needs.
- Rmp permission requested for every new project registered
- Discuss a fee schedule with your manager and or pathologist

PJ0	PJ10 & PJNRPL	> PJ10
No permission	Pathologist permission	Pathologist, RMP & CEO permission

- Send a quotation and SF CLSE 082 to the client

- **Result formats available**

HARD COPY	EMAIL/EPDF	EPDFD	ELECTRONIC Refer to www.ampath.co.za electronic results		
			APR	APM	AMPATH MOBI
Hard copy printed at site closest to client and delivered Registered on route and stop of site Site to be notified of new client	Password required each time to access results	Download decrypted file-enter password once-can then afterwards click on attachment to open results	View, print or save their results on web		View their results on any web enabled phone
			Registered users can download their results into a local database and browse them offline with a web enabled application. Minimum requirements needed to run APM: Ram 256Mb memory • Hard disk space at least 500Mb available • Internet Explorer/Google Chrome • Software: Windows 7 and higher • Net Framework 3.5 or higher Phone Apps-download available from Ampath website-Electronic results: iPad/iPhone & Android version 4+		

ACCOUNT FORMATS AVAILABLE

CONSOLIDATED ACCOUNT	or	CONSOLIDATED ACCOUNT SINGLE INVOICE PER PATIENT

Email completed SF CLSE 082 form to

Client services/provider registration to register on LAB module providerreg@ampath.co.za	Debtors to register on BAR module

- Order referral forms and deliver with a starter box to the client.
- Ensure that the client has the password allocated to the project to open the E mailed results.
- Confirm on LAB and BAR that all the information is correct.
- Purchase Order Numbers – if relevant for payment of account:
  ENSURE that the client obtains a purchase order number before sending in specimens and that this number is written on the referral forms.

AMPATH ELECTRONIC RESULTS

The following electronic services available to you, apart from fax and encrypted e-mail:

APR – AMPATH PATHOLOGY RESULTS

- Allows registered medical practitioners to view, print or save patient results from the web.
- Windows platform only! Features:
- Secure login validation
- No software installation
- Access results from anywhere in the world
- Cumulative reports
- Search functions
- Print PDF report
- Save report to PC/Notebook
- Group practice viewing capabilities

AMPATH MOBI

Ampath Mobi allows registered medical practitioners to view their Ampath results on any web enabled phone including Iphones.

Features of Ampath Mobi include:

- Secure login validation from anywhere
- No software installation and compatible to all web enabled cell phones including Android, Iphones and Ipads.
- Quick new results retrieve
- Search facilities
- Group practice viewing capabilities
- Contact patient from result
- Contact lab support from within patient result

APM – AMPATH PATHOLOGY MANAGER

APM is a downloadable software application, available to registered medical doctors. This application manages pathology results on your PC or server after they have been downloaded.

Features of APM:

- Secure login
- Schedule downloads
- Cumulative View
- Graphs and result trends
- Patient merge
- Backup facilities
- Results flag
- Diagnostic tags

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REPORT FORMATS

INDIVIDUAL REPORTS

PATHOLOGISTS · PATOLOË

Drs Du Buisson, Kramer, Swart, Bouwer Inc./Ing.

AMPATH

Registration number 2007/018337/21

24 Uur kontaknommer
Hour contact number 014 523 9700

PATIENT:	DOCTOR:	SUBMITTING DR:
XXXX XXXX	XXXX XXXX XXXX XXXX XXXX XXXX XXXX XXXX XXXX	XXXX XXXX **COPY DR(s):**

ROUTE:30000:004

Male/ XXXX XXXX
ID : XXXX XXXX
H:Not available
C:
FOLIO:
M/A :
MEMB :
NO :

FINAL REPORT

REQ NO : 98079469

SPEC	:0707:AL01186R	**COLLECTED**	**:07/07/17 0840**
PT LOC	:30000	RECEIVED	:10/07/17 1730
BATCH #	:8969732	PRINTED	:10/10/17 1129

ORDERED: PAH
SAMPLE: U08 Urine Random

COMMENTS: XXXX XXXX

Test	ABN	Result	Reference	Units
BIOCHEMISTRY URINE				
Creatinine Urine		16.2	3.5-23.0	mmol/l
POLYAROMATIC HYDROCARBON				
1-Hydroxypyrene		2.77		ug/l
1-Hydroxypyrene/Creat Ratio		0.8		umol/mol

Reference Limits:
**Not industrial exposed : <1 umol/mol creatinine*
**BEI (Biological exposure index): 1.4 umol/mol creatinine at end of shift*

For Consultation - Doctors only. Please call:
LOCAL PATHOLOGIST : Dr Jan du Bruyn

** End of report **

MEMBER•LID

=>NUWE RESULTAAT SEDERT VORIGE VERSLAG / NEW RESULTS SINCE PREVIOUS REPORT
#BETEKENISVOLLE VERANDERING SEDERT VORIGE VERSLAG / SIGNIFICANT CHANGE SINCE PREVIOUS REPORT *L / *H HOOGS ABNORMALE RESULTATE / HIGHLY ABNORMAL RESULTS

GROUP REPORTS

COMPANY NAME	c12345			
1-Hydroxypyrene:creat ratio				
NAME	**M\F**	**D.O.B**	**May'13**	**May'14**
Employee A	M	18/09/86	0.8	1.8
Employee B	M	28/05/84	1.1	1.9
Employee C	M	26/12/83	0.7	0.6
Employee D	F	24/06/81	0.8	1.8
Employee E	M	23/05/67	1.1	1.9
Employee F	F	02/02/78	0.7	0.6
Employee G	M	04/05/55	0.8	1.8
Employee H	M	29/08/88	1.1	1.9
Employee I	M	20/09/82	0.7	0.6

Reference Range:

Not industrial exposed	<1 umol/mol creatinine
BEI (Biological exposure index)	1.4 umol/mol creatinine at end of shift

NB. Please note that all medical surveillance test results have to be reviewed and actioned by an Occupational Health Practitioner. Patients must consent to tests and the test result must be explained by said practitioner to the patient.

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USEFULL WEBSITES

http://www.ampath.co.za	Ampath
http://www.asosh.org/	ASOSH Web site
http://www.cdc.gov/niosh/homepage.html	NIOSH Web site
http://www.cdc.gov/	CDC Web site
http://www.acgih.org/home.htm	ACGIH Web site
http://www.ilo.org/	ILO Web site
http://www.epa.gov/	EPA Web Site
http://www.hse.gov.uk/	HSE Web site
http://www.wcomp.gov.za/	Compensation Commissioner website
http://www.ccohs.com/	CCOHS Web Site
http://www.fda.gov/	FDA Web Site
http://www.who.org/	WHO Web site
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
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
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