Department of Occupational Safety and Health
Ministry of Human Resources

GUIDELINES ON MEDICAL SURVEILLANCE PROGRAMME AT THE WORKPLACE 2023

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PREFACE

These guidelines may be cited as the Guidelines on Medical Surveillance Programme at The Workplace 2023.

The purpose of these guidelines is to guide, clarify and elaborate on the contents and the procedures of medical surveillance (MS) conducted by the Occupational Health Doctors (OHD) in complying with the requirements of Regulations 27(2), Occupational Safety and Health (Use and Standard of Exposure of Chemicals Hazardous to Health) Regulations 2000 or USECHH 2000. These Guidelines were developed to replace the Guidelines on Medical Surveillance Programme 2001.

Employers are also encouraged to read these guidelines in conjunction with the USECHH 2000 including any amendments, to assist in fulfilling the requirements of Regulations 27(1) for Health Surveillance Programme in a comprehensive and integrated approach.

Employers and employees as well as other occupational safety and health (OSH) practitioners involved in the Medical Surveillance Programme must understand the rationale and the importance of the programme as this will improve their cooperation with the Occupational Health Doctors (OHD) in ensuring success of the programme.

These guidelines consist of two sections: General Guidelines and Specific Guidelines on Medical Surveillance Programmes. The General Guidelines detail out the important components of MS Programmes' procedures. The Specific Guidelines are the detailed guides in conducting MS programme for the listed chemicals hazardous to health (CHTH).

With these Guidelines, which are practical and comprehensive, it is anticipated that Medical Surveillance Programme will be carried out effectively for early detection or prevention of occupational disease and occupational poisoning thereby increasing the organizational productivity and towards healthy working population.

Ir. Hj. Mohd Hatta bin Zakaria

Director General
Department of Occupational Safety and Health
Malaysia

2023

CONTENT

PART A: GENERAL GUIDELINES OF MEDICAL SURVEILLANCE PROGRAMME

ACKNOWLEDGEMENTS xv

GENERAL GUIDELINES 1

TERMINOLOGY AND DEFINITIONS 2

1.0 INTRODUCTION 5

2.0 TOXICOLOGY 13

3.0 CHEMICAL HEALTH RISK ASSESSMENT (CHRA) & EXPOSURE MONITORING (EM) PROGRAMME 35

4.0 BIOLOGICAL MONITORING (BM) AND BIOLOGICAL EFFECT MONITORING (BEM) 45

5.0 HEALTH EFFECTS MONITORING (HEM) 61

6.0 LABORATORY PROCEDURES 74

7.0 GENERAL MEDICAL SURVEILLANCE PROCEDURES 90

8.0 SEARCHING FOR INFORMATION FOR CHTH NOT LISTED IN THE SPECIFIC GUIDELINES 102

9.0 REPORT AND RECORD KEEPING 110

REFERENCES

APPENDICES 491

CONTENT

PART B: SPECIFIC GUIDELINES ON MEDICAL SURVEILLANCE PROGRAMME

ARSENIC	120
ARSINE GAS	132
ASBESTOS	140
AURAMINE	148
BENZENE	154
BENZIDINE & ITS DERIVATIVES	165
BENZIDINE	165
DICHLOROBENZIDINE	172
BERYLLIUM AND ITS COMPOUNDS	178
BIS(CHLOROMETHYL) ETHER	188
CADMIUM	194
CARBON DISULPHIDE	205
CARBON TETRACHLORIDE	214
CHROMIC ACID	223
DIANISIDINE	233
DISULPHUR DICHLORIDE	238
N-HEXANE	243
HEXAVALENT CHROMIUM COMPOUND	251

CONTENT

PART B: SPECIFIC GUIDELINES ON MEDICAL SURVEILLANCE PROGRAMME

ISOCYANATES 260

LEAD 273

INORGANIC LEAD 273

ORGANIC LEAD 282

MANGANESE 290

MERCURY (Elemental and Inorganic Mercury) 301

MINERAL OIL MIST, REFINED MINERAL 313

NAPHTHYLAMINE AND ITS ISOMERS 320

1-NAPHTHYLAMINE 320

ß-NAPHTHYLAMINE 325

NICKEL 330

NITROBENZENE 340

NITRO OR AMINO DERIVATIVES
OF PHENOL AND DIPHENYL 348

ANILINE 348

4-NITRODIPHENYL 355

4-AMINODIPHENYL 360

ORGANOPHOSPHATES 366

ORTHOTOLIDINE 379

CONTENT

PART B: SPECIFIC GUIDELINES ON MEDICAL SURVEILLANCE PROGRAMME

PITCH, TAR, BITUMEN & CREOSOTE 385

POTASSIUM OR SODIUM CHROMATE OR DICHROMATE 392

SILICA (CRYSTALLINE) 403

TOLUENE 413

TRICHLOROETHYLENE 424

VINYL CHLORIDE MONOMER 433

XYLENE 443

ALUMINIUM AND ITS COMPOUND 452

DIESEL 461

FORMALDEHYDE 469

RESPIRATORY IRRITANTS 479

CALCIUM CARBONATE 479

CHLORINE AND CHLORINE PRODUCTS 484

LIST OF FIGURES

Figure 1. Process Flow of MS Procedure 12

Figure 2. Dose-response Curve 15

Figure 3. Summary of ADME 20

Figure 4. Routes of exposure 22

Figure 5. Molecular Basis of Cancer 30

Figure 6. Pathway from Exposure to Occupational Disease 33

Figure 7. CHRA Process Flow 37

Figure 8. Requirement to conduct the MS Process Flow 40

Figure 9. CHRA report information retrieval process flow 41

Figure 10. Results interpretation process flow for urine samples 59

Figure 11. Relationship between HEM and Other Types of Monitoring Parameters 61

Figure 12. Process flow of MRP 99

Figure 13. Exposure Limits for Benzene 491

Figure 14. Exposure Route for Benzene 491

Figure 15. Absorption of Benzene 492

Figure 16. Distribution of Benzene 492

Figure 17. Metabolism of Benzene 493

LIST OF FIGURES

Figure 18. Elimination of Benzene 494

Figure 19. Toxicodynamic of Benzene Exposure 496

Figure 20. Approach to the MS of the Respiratory System 509

Figure 21. Critical information to be obtained from the worker with a suspicion of Occupational Liver Disease 510

Figure 22. Schematic approach to the assessment and diagnosis of Occupational Liver Disease 511

Figure 23. Development of Chemical Related Occupational Disease 517

LIST OF TABLES

Table 1. Degree of responses 14

Table 2. Fundamental concept in toxicology 16

Table 3. Definition of Toxicokinetic and Toxicodynamic 20

Table 4. Summary of Absorption, Distribution, Metabolism and Excretion 21

Table 5. Examples of CHTH and its site of absorption 21

Table 6. Common Routes of Excretion for CHTH 23

Table 7. Effects of Chemical Exposure to Major Organ/System 25

Table 8. Relative ranking of toxicity potency of chemicals 29

Table 9. ICOP on Chemicals Classification and Hazard Communication: Hazard Categories for Carcinogens 31

Table 10. IARC Classification 32

Table 11. CHRA Assessment Forms and its Contents 38

Table 12. Important information for the MS programme retrieved from the CHRA report 42

Table 13. Comparison of BM and BEM with Air Monitoring 45

Table 14. Notations in ACGIH BEI 48

Table 15. Health Effects Monitoring of the Lungs for cases of Hypersensitivity 64

Table 16. Health Effects Monitoring of the Lungs for cases of Fibrosis 65

LIST OF TABLES

Table 17. Health Effects Monitoring of the Central Nervous System 66

Table 18. Health Effects Monitoring of the Reproductive System 67

Table 19. Health Effects Monitoring of the Skin 68

Table 20. Health Effects Monitoring of the Liver 69

Table 21. Health Effects Monitoring of the Kidneys 70

Table 22. Health Effects Monitoring of the Haematology 71

Table 23. Health Effects Monitoring for cases of Cancer 72

Table 24. Definitions of Important Performance Characteristics 82

Table 25. Comparison between Accuracy and Precision 84

Table 26. USECHH Forms for Reporting and Record Keeping 111

Table 27. Metabolites of Benzene 494

Table 28. Potential Effects of Inhaled Irritants 506

Table 29. Pathological Patterns and Morphological Features of Liver Disease Associated with Workplace-related Toxicants 512

Table 30. Summary of Current Clinical Serum Biomarkers of Liver Function and Injury 513

LIST OF TABLES

Table 31. Clinical-diagnostic Types of Occupational Toxic Hepatitis 515

Table 32. Category of Severity Description 516

Table 33. Typical Features of Non-alcoholic Fatty Liver Disease (NAFLD) and Alcoholic Liver Disease 517

Table 34. The Mathias Criteria for Probable Occupational Causation of Contact Dermatitis 521

APPENDICES

Appendix 1: Case Study (Benzene) 491

Appendix 2: Assessment of the Fitness to Wear Respirator 497

Appendix 3: Approach to MS of the Respiratory System 505

Appendix 4: Approach to Diagnosis of Occupational Liver Disease 510

Appendix 5: Swedish Q16 Questionnaire 519

Appendix 6: The Mathias Criteria 521

Appendix 7: MS Recommended Forms 523

Appendix 8: Example of MS Recommended Forms – USECHH 4 538

LIST OF ABBREVIATIONS

ACGIH	American Conference of Governmental Industrial Hygienists
ADME	Absorption, Distribution, Metabolism and Excretion
AF	Adjustment Factor
AP	Action Priority
BEI	Biological Exposure Index/Indices
BEL	Biological Exposure Limit
BEM	Biological Effect Monitoring
BM	Biological Monitoring
BW	Body Weight
CAF	Composite Adjustment Factor
CDC	Centres for Disease Control and Prevention, United States
EM	Exposure Monitoring
CNS	Central nervous system
CLASS 2013	Occupational Safety and Health (Classification, Labelling, and Safety Data Sheet of Hazardous Chemicals) Regulations 2013
CHRA	Chemical Health Risk Assessment
CHTH	Chemicals hazardous to health
CO	Carbon monoxide
DDT	Dichlorodiphenyltrichloroethane

LIST OF ABBREVIATIONS

DES	Diethylstilbestrol
DNA	Deoxyribonucleic acid
DNEL	Derived No-effect Level
DOSH	Department of Occupational Safety and Health
ER	Exposure Rating
HEM	Health Effects Monitoring
HR	Hazards Rating
HT1	Hygiene Technician 1
IARC	International Agency for Research on Cancer
ICOP	Industry Code of Practice
LD	Lethal Dose
LOAEL	Low-observed Adverse Effect Level
LOR	Level of Risk
MEL	Maximum Exposure Limit
MRP	Medical Removal Protection
MS	Medical Surveillance
NADOPOD 2004	Occupational Safety and Health (Notification of Accident, Dangerous Occurrence, Occupational Poisoning and Occupational Disease) Regulations 2004

LIST OF ABBREVIATIONS

NIOSH	National Institute of Occupational Safety and Health, United States
NOAEL	No-observed Adverse Effect Level
OHD	Occupational Health Doctor
PCB	Polychlorinated Biphenyls
PEL	Permissible Exposure Limit
POD	Point of departure
PPE	Personal Protective Equipment
RR	Risk Rating
QC	Quality Control
RTW	Return to Work
SDS	Safety Data Sheet
SQC	Standard Quality Control
STEL	Short Term Exposure Limit
TDI	Tolerable Daily Intake
TLV	Threshold Limit Value
TWA	Time Weighted Average
USECHH 2000	Occupational Safety and Health (Use and Standard of Exposure of Chemicals Hazardous to Health) Regulations 2000

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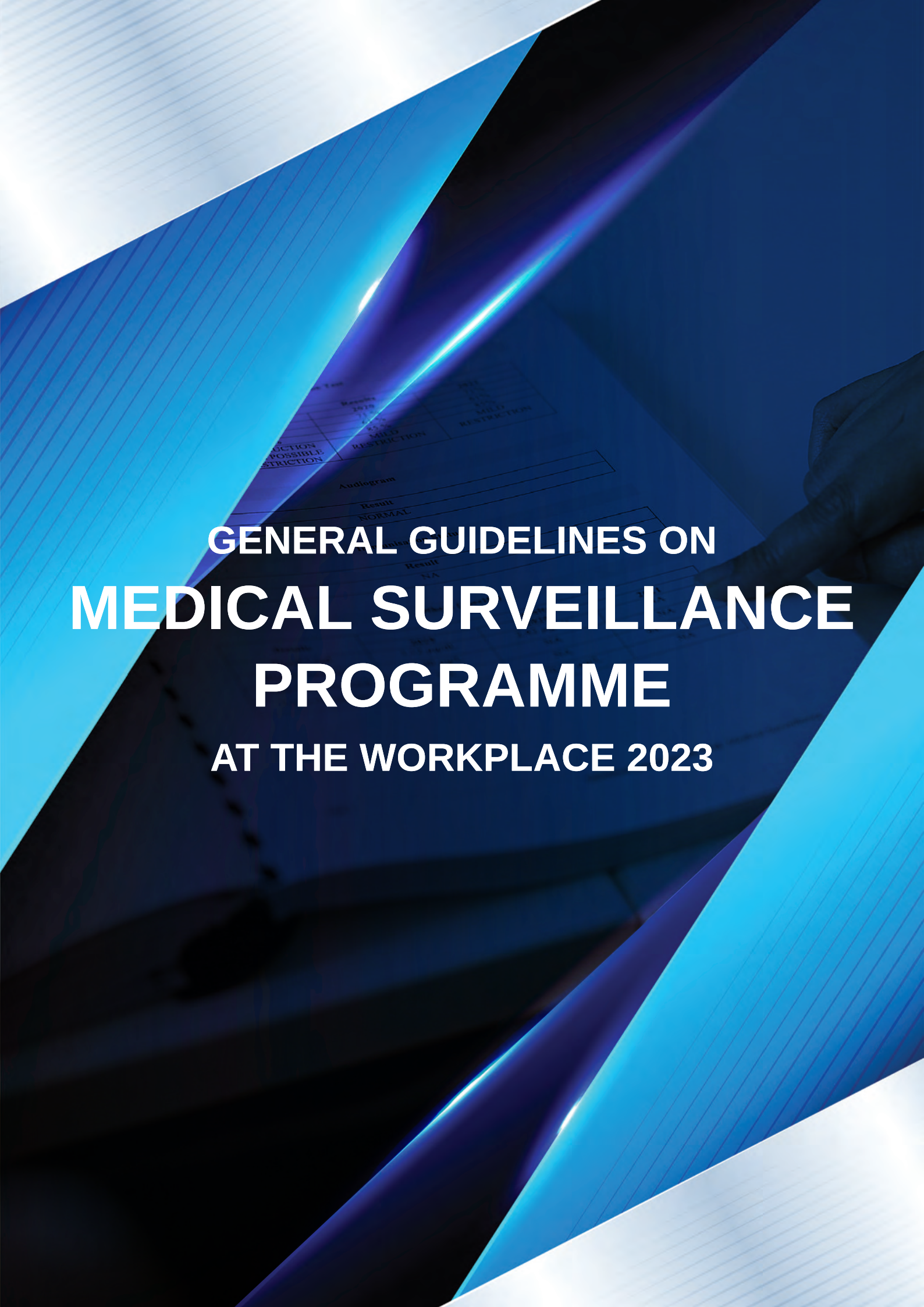


GENERAL GUIDELINES ON MEDICAL SURVEILLANCE PROGRAMME AT THE WORKPLACE 2023

TERMINOLOGY AND DEFINITIONS

Biological Exposure Indices (BEI) are guidance values (ACGIH) for evaluating biological monitoring results. BEI generally represents the levels of determinants that are most likely to be observed in specimens collected from healthy employees who have been exposed to chemicals to the same extent as employees with inhalation exposure at the Threshold Limit Value-Time Weighted Average (TLV-TWA). The BEI represents Biological Exposure Limit (BEL).

Biological Exposure Limit (BEL) is the guidance values for evaluating biological monitoring results, which are adopted from several sources and mostly are BEI from ACGIH.

BEI Determinants are an index of an individual's uptake of a chemical by all routes. In some cases, they correspond to the TLV as a **safe** level without reported health effects (ACGIH). In other cases, they may reflect the highest 5% of levels seen in the general population. The BEI determinant can be the chemical itself; one or more metabolites; or a characteristic, reversible biochemical change induced by the chemical. The specimens used for BEI determinants are urine, blood, or exhaled air.

Biological Monitoring (BM) means the measurement and assessment of agents and/or their metabolites either in tissues, secreta, excreta, expired air, or any combination of these to evaluate exposure and health risk compared to an appropriate reference (CHRA Manual 3rd Edition).

Biological Effect Monitoring (BEM) means the measurement and assessment of the sub-clinical biological effect caused by the hazards (CHRA Manual 3rd Edition). It normally involves measuring biochemical responses that may have health consequences for the individual and may be caused by factors other than occupational exposure.

Ceiling limit means the airborne concentration that shall not be exceeded during any part of the working day as specified in the USECHH 2000.

Chemical means:

a) a substance which is a chemical element and its compounds in the natural state or obtained by any manufacturing process, including any additive necessary to preserve its stability and any impurity deriving from the process used, but excluding any solvent which may be separated without affecting the stability of the substance or changing its composition; or

b) a chemical mixture which is a mixture or solution composed of two or more substances which do not react; for use at a place of work, including an alloy.

Chemicals hazardous to health (CHTH) mean any chemicals or preparations which:

a) are listed in schedules in USECHH 2000;

b) are classified in any hazard class specified under Health Hazards of First Schedule of the Occupational Safety and Health (Classification, Labelling, and Safety Data Sheet of Hazardous Chemicals) Regulations 2013 (CLASS 2013);

c) come within the definition of **pesticide** under the Pesticides Act 1974; or

d) listed in the First Schedule of the Environmental Quality (Scheduled Wastes) Regulations 2005.

Chemical Health Risk Assessment (CHRA) is an assessment that has to be conducted by the employer in his workplace as required by the USECHH 2000.

Exposure Monitoring The exposure monitoring programme is conducted to evaluate the extent of workers' exposure to CHTH and adequacy of existing control measures at the workplace. Exposure monitoring may include air monitoring which is area or personal monitoring, and biological monitoring or a combination of both.

Health Effects Monitoring (HEM) is a process to monitor clinical onset of diseases following an exposure to CHTH.

Maximum exposure limit (MEL) means a fifteen-minute time weighted average airborne concentration limit which is three times the eight-hour time weighted average limit specified in the USECHH 2000.

Medical Removal Protection (MRP) means temporary or permanent removal of the affected employee from a work area in order to prevent him or her from further exposure to chemicals hazardous to health.

Medical Surveillance (MS) means a systematic and periodic assessment of the state of health of an employee upon exposure to chemical hazardous to health which may include biological monitoring or biological effect monitoring.

Occupational Health Doctor (OHD) means a registered medical practitioner who is registered with the Director General of DOSH to conduct MS programmes of employees.

Permissible exposure limit (PEL) means a ceiling limit, or an eight-hour time weighted limit, the maximum exposure limit, or the short-term exposure limit.

Short term exposure limit (STEL) means a fifteen-minute time weighted average airborne concentration limit as specified in the USECHH 2000.

Time weighted average (TWA) limit means an average airborne concentration that shall not be exceeded over a specified period of time as specified in the USECHH 2000.

Validated method means any standard method of sampling and analysis for airborne chemicals evaluated by the Department of Standards Malaysia or any internationally recognized body.

1.0 INTRODUCTION

1.1 INTRODUCTION TO MEDICAL SURVEILLANCE (MS) OF CHEMICALS HAZARDOUS TO HEALTH (CHTH) AT THE WORKPLACE

1.2 OBJECTIVES OF THE GUIDELINES ON MS PROGRAMME

1.3 COMPONENTS OF THE GUIDELINES ON MS PROGRAMME

1.4 DUTIES OF OHD IN THE MS PROGRAMME

1.5 DUTIES OF EMPLOYER

1.6 DUTIES OF EMPLOYEE

1.7 COMPONENTS OF MS PROGRAMME

1.8 PROCESS FLOW IN MS PROGRAMME

1.0 INTRODUCTION

1.1 INTRODUCTION TO MEDICAL SURVEILLANCE (MS) OF CHEMICALS HAZARDOUS TO HEALTH (CHTH) AT THE WORKPLACE

Malaysia has developed to become an advanced and industrialised nation. The introduction of newer processes has led to the use of various chemicals which can often be hazardous to health. MS, if properly implemented, can protect the safety and health of employees exposed and prevent the development of occupational diseases through pre-placement and periodic medical examinations. This is possible by early detection in which necessary intervention may be employed to prevent the progression of the disease.

The Occupational Safety and Health (Use and Standards of Exposure of Chemicals Hazardous to Health) Regulations (USECHH) 2000 and The Manual of Recommended Practice on the Assessment of the Health Risks Arising from the Use of Chemicals Hazardous to Health at the Workplace (3rd edition) assist employers to assess whether there is any significant exposure of the chemicals to the employee which would necessitate MS to be conducted by OHD.

1.2 OBJECTIVES OF THE GUIDELINES ON MS PROGRAMME

These Guidelines are intended to be used as a guide for employers and OHD in performing MS for employees exposed to CHTH. They also aim to highlight important aspects of MS and provide clarification on these elements for effective implementation.

To answer these objectives, the Guidelines are separated into two (2) sections: General Guidelines of MS Programme and Specific Guidelines on MS Programme.

1.3 COMPONENTS OF THE GUIDELINES ON MS PROGRAMME

Section A: General Guidelines of MS Programme details out the important components of MS procedure. This includes terminology and definition, principles of toxicology, CHRA and CEM, BM and BEM, HEM, and laboratory procedures relevant to MS, general MS procedure, searching for information for chemicals not listed in the specific guidelines and report and notification. The aspects discussed in this section aim to clarify and elaborate the processes involved preceding, during and after conducting a MS programme.

Section B: Specific Guidelines on MS Programme is a detailed guide in conducting MS for CHTH listed under USECHH 2000. The guidelines outline the necessary information needed for MS

procedure including PEL and BEL in determining occupational exposure. Other aspects detailed are the physicochemical properties, material use, hazard classification and toxicity which includes sources of potential occupational exposure, route of exposures and toxicokinetic elements separated into absorption, distribution, metabolism, and excretion of the chemical. These revised Guidelines introduce the principle of HEM which lists the acute and chronic effects required for practitioners to understand in assessing the health effects from CHTH exposure. This section includes aspects of the MS programme which are indications for MS procedure, frequency and content of pre-placementand periodic medical examination, biological assessments (if applicable) and the relevant laboratory procedure, criteria for MRP and RTW as well as management and preventive measures.

1.4 DUTIES OF OHD IN THE MS PROGRAMME

Regulation 27, USECHH 2000 stipulates the requirement of MS to be performed by the OHD. The general roles of OHDs in the MS Programme are to:

1) Conduct the pre-placement medical examination (baseline medical data) of employees to assess fitness for work;

2) Conduct the periodic MS;

3) Review the employees' exposure to chemical hazards at the workplace from relevant documents such CHRA, Air Monitoring reports and Biological Monitoring reports;

4) Determine fitness to work with the relevant chemicals including the fitness to wear the Personal Protective Equipment (PPE);

5) Analyse and investigate abnormal MS findings which may include workplace visit;

6) Obtain any medical records of employees from employer if needed;

7) Explain the results of investigations and other relevant findings to employee and employer and specify remedial actions and further follow up;

8) To notify employer and DOSH on MRP within one working day;

9) Notify Occupational Diseases and Poisoning to DOSH and employer as required under the relevant legislation;

10) Submit and present the MS report to the employer within one month upon completion of MS;

11) Submit the required MS report to DOSH within one month upon completion;

12) Assist in the management of Occupational Diseases and Poisoning including treatment, rehabilitation, disability assessment, compensation and RTW;

13) Participate in the training related to chemical health risk at the workplace if needed; and

14) Perform exit medical examinations prior to retirement for employees who have been exposed to the CHTH.

1.5 DUTIES OF EMPLOYER

The duties of employer include to:

1) Appoint an OHD to conduct MS programme;

2) Ensure proper process of the MS Programme and other relevant procedures are adhered to;

3) Ensure the MS Programme conducted as advised by the OHD;

4) Allow and assist the OHD to visit the workplace to investigate and manage occupational disease and poisoning including access to relevant monitoring and other health related data such as CHRA, EM reports, BM reports and others;

5) Cooperate with the OHD in management of the employees, including and not limited to treatment, rehabilitation, disability assessment, compensation and/or RTW;

6) Institute MRP within one working day and RTW of an employee when recommended by the OHD or occupational safety and health officer who is also a medical practitioner;

7) Allow employees under MRP, if necessary to do other work that will not expose them to the CHTH of concern;

8) Notify occupational disease and poisoning to DOSH;

9) Explain the general MS results in the training programme of CHTH;

10) Allow the employee to access his/her own MS records;

11) Review the workplace control measures, including but not limited to organisational control, technical control and emergency response preparedness, and improve them as required;

12) Ensure record keeping and confidentiality of MS records for 30 years and be made available to the appointed OHD; and

13) Maintain proper record keeping of occupational diseases and poisoning.

1.6 DUTIES OF EMPLOYEE

The duties of employee include to:

1) Undergo training on importance of preventing occupational poisoning and disease as instructed by employer;

2) Report early signs and symptoms and signs of disease (including self-examination) to the OHD and management; and

3) Comply with the requirements of MS Programme as stipulated under USECHH 2000.

1.7 COMPONENTS OF MS PROGRAMME

The components of MS Programme include:

1) Pre-placement, periodic and exit medical examination which may include Health Effects Monitoring (HEM), Biological Monitoring (BM) and Biological Effect Monitoring (BEM);

2) Relevant laboratory procedures;

3) Investigation of occupational disease and poisoning including workplace inspections;

4) Notification of occupational disease and poisoning;

5) Medical removal protection;

6) Return to work examination after medical removal protection; and

7) Reporting and record keeping.

1.8 PROCESS FLOW IN MS PROGRAMME

a) Review findings of CHRA and Identification of CHTH requiring MS

The decision on a MS programme is based on the results on CHRA and CEM. It is essential that the OHD review the CHRA and CEM report. All of the details are provided in **Chapter 3.0 Chemical Health Risk Assessment & Exposure Monitoring Programme.** It is also important for OHD to understand the basic toxicokinetic and toxicodynamic principles. This information will assist with the summary of the chemical's different harmful health effects. All details are described in **Chapter 2.0 Toxicology**.

b) CHTH listed in MS Guidelines

The Guidelines on MS Programme describes the MS procedure for all CHTH that require MS (refer to the list of CHTH in **Part B: Specific Guidelines on Medical Surveillance Programme)**. If the CHTH is listed in the guidelines, the MS should be carried out as outlined. If the CHTH is not listed, MS programmes should still be carried out using other reliable sources (all details are provided in **Chapter 8.0: Searching for Information for CHTH Not Listed in the Specific Guidelines.**

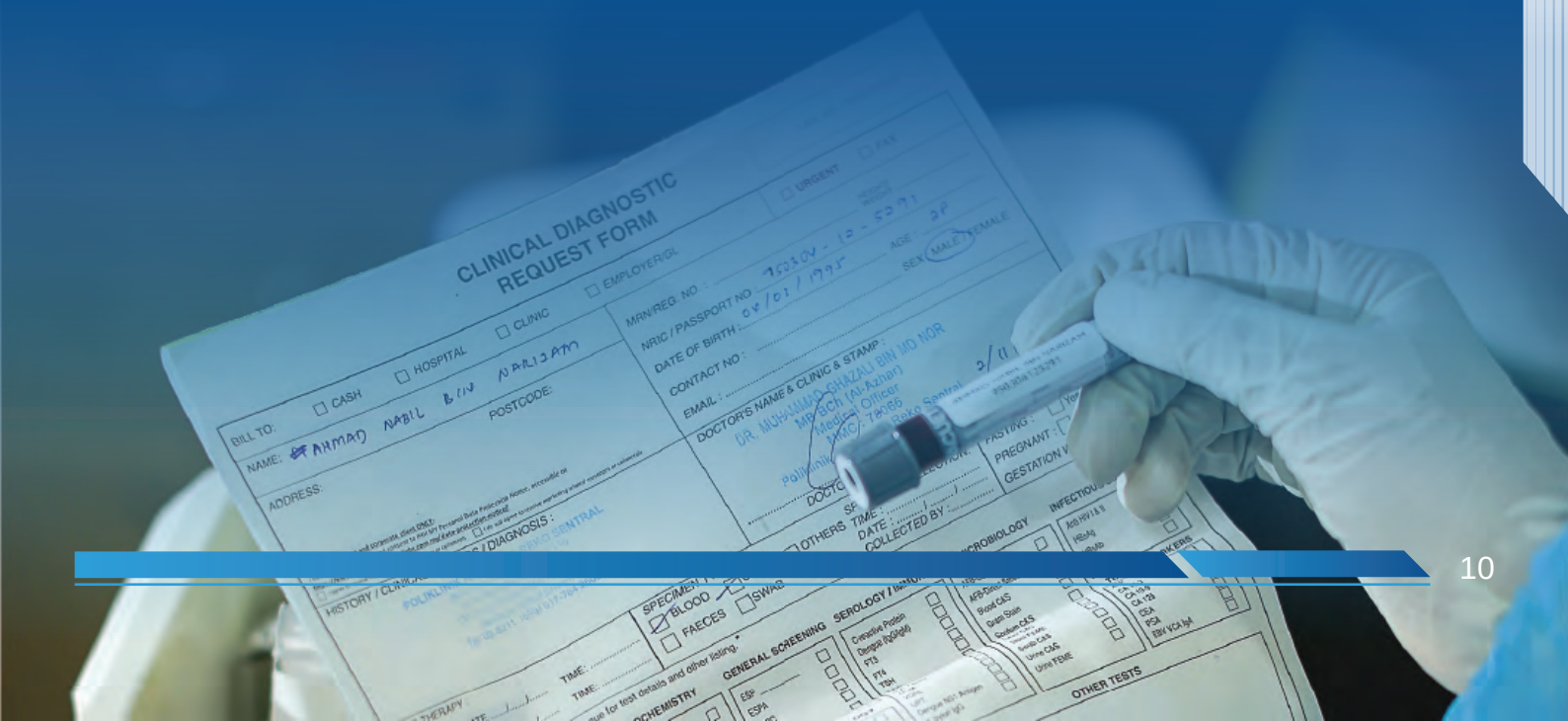
c) Performing Medical Examination and Biological Sampling

An integral part to the MS procedure is the clinical examination and any applicable biological assessments. The following information should be collected to meet these requirements:

- BM and BEM require the collection of BEI determinants. It is critical to understand this BEI determinant as it represents the overall uptake of a chemical by an individual through all routes, including inhalation, ingestion, skin contact, or a combination of these routes. Details are provided in **Chapter 4.0 Biological Monitoring (BM) & Biological Effects Monitoring (BEM).**

- The OHD performing the examination should consider the employee's occupational history, exposure, medical history including previous and/or current illness to detect any medical conditions. All information on HEM can be found in **Chapter 5.0 Health Effects Monitoring (HEM).**

- The sampling procedure should follow the methods given for each chemical or to the laboratory's specific standards. The OHD is responsible for ensuring the integrity of the biological sample in the MS programme. A healthcare practitioner or trained designated personnel must supervise the sample collection. The information is further explained under **Chapter 6.0: Laboratory Procedures.**



d) Analysis of results

Analysis of the medical examination shall conclude whether the findings are abnormal to decide the subsequent steps to be taken in the MS procedure. If the results are abnormal, DOSH must be notified using the notification forms indicated in this guideline **(refer to Appendix 7: Medical Surveillance Recommended Forms)**. The findings must also be shared with the employer and employees. The OHD may recommend the employee's medical removal protection (MRP) based on the requirements listed for each CHTH. The outline on MRP and RTW are explained in **7.4.5 Process Flow of MRP and RTW.** The duration of the medical removal is limited to the time required for the employee's medical condition to normalise and/or when the biomarker of exposure falls below the BEI. All procedures are explained in the **Chapter 7: General Medical Surveillance Procedures.**

e) Reporting and Record Keeping

Record keeping is important in MS to keep track of the employee's medical condition as a result of CHTH exposure. It may also serve as the main reference for recommendations and control measures that may be taken to safeguard the health of the employee throughout the duration of employment and further into the future. A report shall be furnished to the employer and DOSH within the time limit set (within 30 days upon conclusion of the MS) so that workplace conditions may be improved, and steps can be formulated to prevent adverse health effects to the employee. All information is provided in **Chapter 9.0: Report and Record Keeping.**

Figure 1 illustrates the process flow of a MS programme which is recommended to be used as a general guide for OHD.

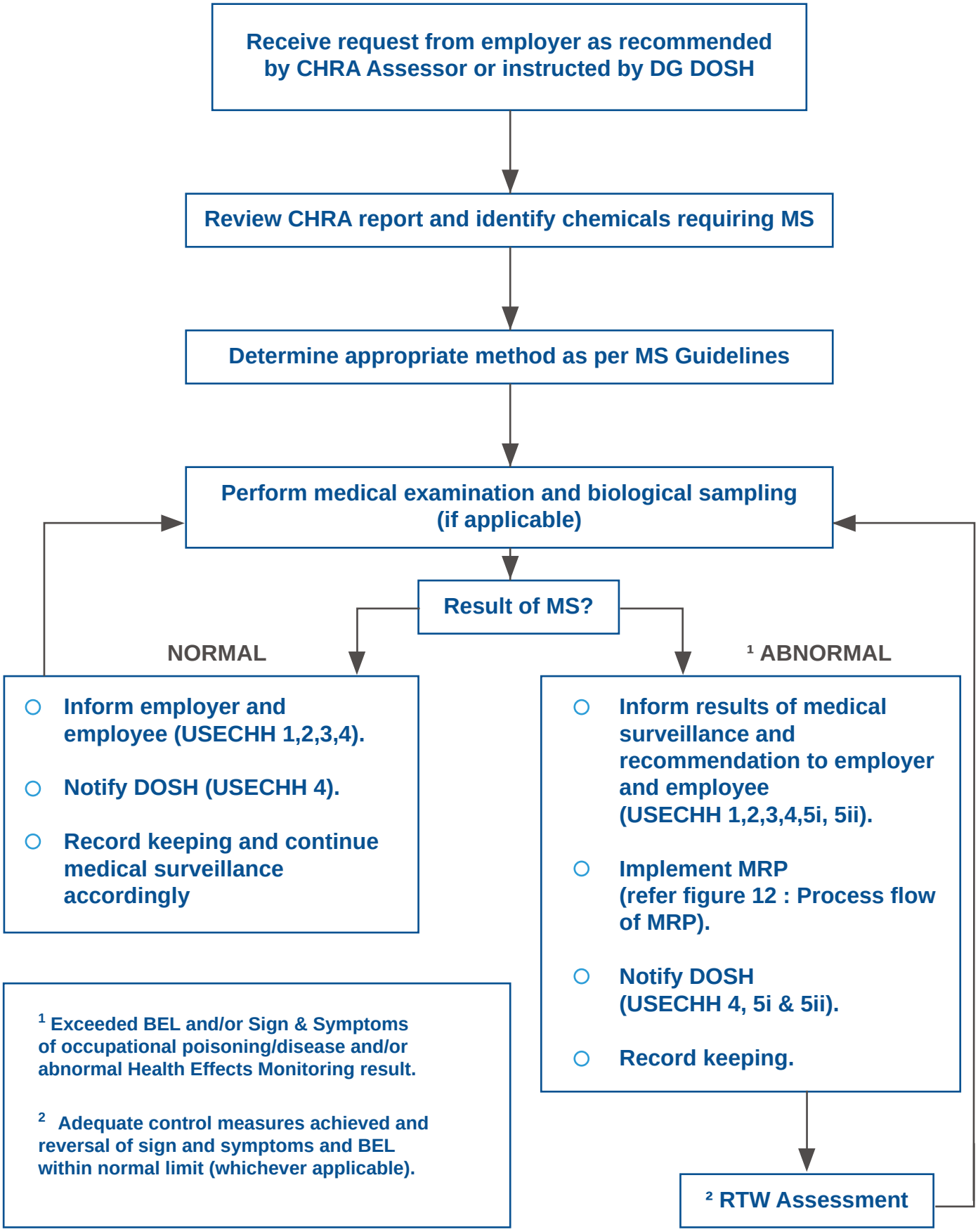
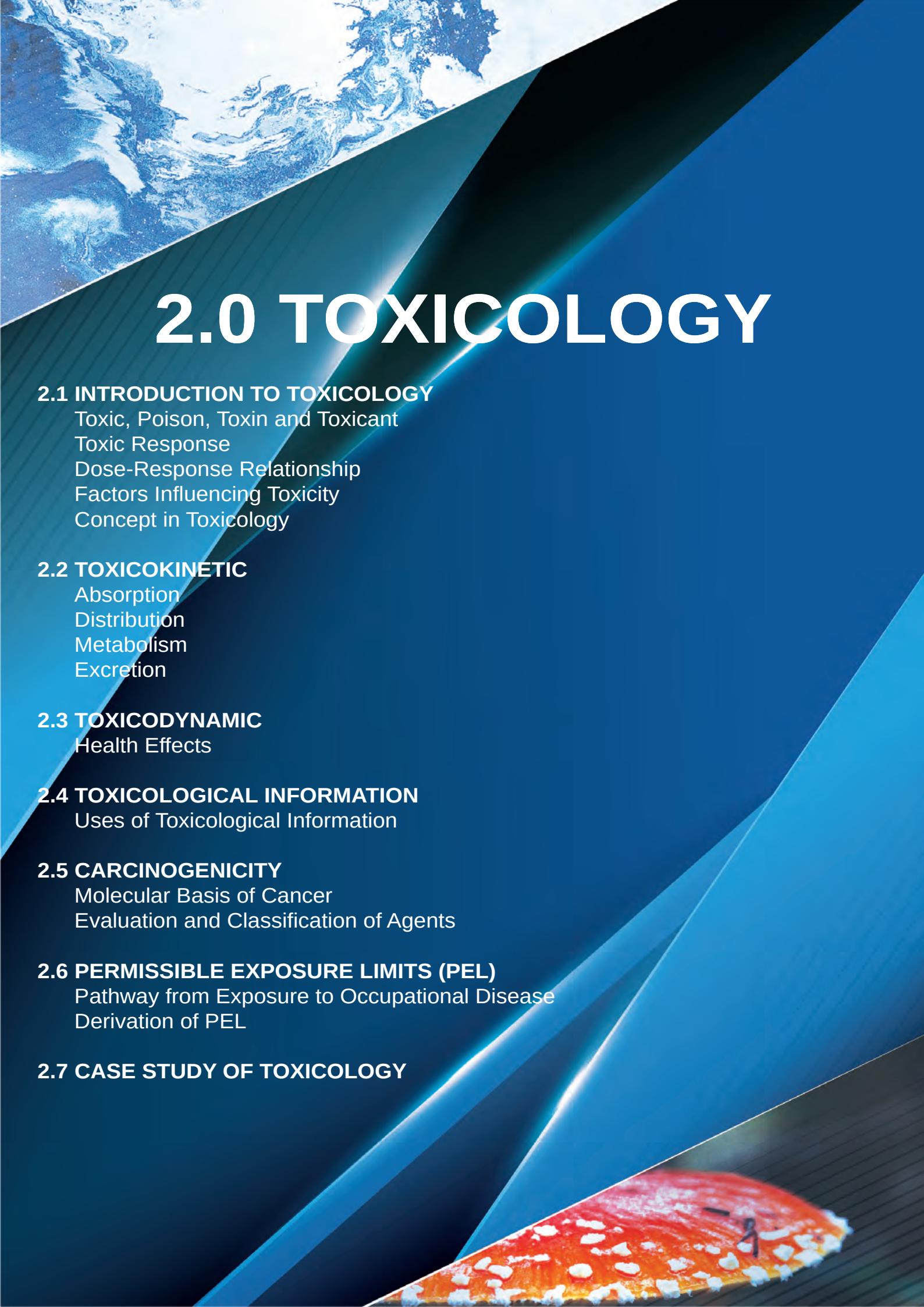


Figure 1. Process Flow of MS Procedure



2.0 TOXICOLOGY

2.1 INTRODUCTION TO TOXICOLOGY
- Toxic, Poison, Toxin and Toxicant
- Toxic Response
- Dose-Response Relationship
- Factors Influencing Toxicity
- Concept in Toxicology

2.2 TOXICOKINETIC
- Absorption
- Distribution
- Metabolism
- Excretion

2.3 TOXICODYNAMIC
- Health Effects

2.4 TOXICOLOGICAL INFORMATION
- Uses of Toxicological Information

2.5 CARCINOGENICITY
- Molecular Basis of Cancer
- Evaluation and Classification of Agents

2.6 PERMISSIBLE EXPOSURE LIMITS (PEL)
- Pathway from Exposure to Occupational Disease
- Derivation of PEL

2.7 CASE STUDY OF TOXICOLOGY

2.0 TOXICOLOGY

2.1 INTRODUCTION TO TOXICOLOGY

Toxicology refers to the study of the adverse effects of chemical, physical, or biological agents on living organisms and the ecosystem, including the prevention and amelioration of such adverse effects.60 The ultimate aim of toxicology is to understand the toxic properties of these agents so that the adverse effects can be prevented by the development of appropriate handling or exposure guidelines.

Toxicology is the cornerstone in all aspects of chemical safety. It uses the power of science to predict what and how chemicals may cause harm and eventually share the information to protect public health. In this respect, the setting of occupational exposure limits to protect employees' health requires critical toxicological information.

2.1.1 Toxic, Poison, Toxin and Toxicant

The term 'toxic' refers to having the characteristic of being able to produce undesirable or adverse health effects at a certain dose. Every known chemical has the potential to be toxic or poisonous if it is present in a sufficient amount.

Poison refers to any agent that can either be a toxin or a toxicant that is capable of producing deleterious responses in a biological system. The term 'toxicant' is often used to refer to toxic substances that are produced by or are a by-product of man-made activities whereas the term 'toxin' is usually used to refer to toxic substances produced naturally.

2.1.2 Toxic Response

A toxic response is an adverse reaction to toxicant and toxin exposure. The toxic responses span a broad biological and physiological spectrum. The toxic responses may range from something relatively minor such as irritation or tearing to a more serious response and permanent disability such as cirrhosis of the liver or liver cancer.

The degree of responses depends upon the dose and the organism as described in Table 1:

Table 1. Degree of Responses

Degree of responses	Description
Allergic reactions	An adverse reaction of the immune system to a toxicant in response to a previous exposure to that chemical or to a structurally similar one. This allergic response is often defined as ‘hypersensitivity’.
Idiosyncratic reactions	Abnormal reactivity can sometimes occur in individuals based on its genetics or other individual sensitivity factors upon exposure to a toxicant.
Immediate vs Delayed Toxicity	The toxic effect of a chemical can develop rapidly or may be delayed for a period of time after chemical exposure(s). Immediate toxicity can be observed straightaway for most toxicants. However, delayed toxicity may take months or years to be recognized.
Reversible vs Irreversible Toxicity	Toxic effects can be reversible or irreversible. This depends on the ability of the biological system to adapt, repair and regenerate upon injury caused by the chemical exposure.
Local vs Systemic Toxicity	Local toxicities are those effects occurring at the contact site upon exposure. For systemic toxicities, the toxicant is usually absorbed and distributed to a distant site from its entry point where the adverse effects are produced.
Interaction of chemicals	An individual may be exposed to multiple toxicants at a given time and these toxicants may have interactions with each other to produce a response.
Tolerance	A reduced response by a person to a toxicant, which occurs when the person is exposed repeatedly, and the body adapts to the continued presence of the toxicant.

2.1.3 Dose-Response Relationship

The dose-response relationship is the fundamental concept in toxicology and the basis for measurement of the relative harmfulness of a chemical. It is a consistent mathematical and biologically plausible correlation between the number of individuals responding to a given dose over an exposure period (Table 2 and Figure 2).

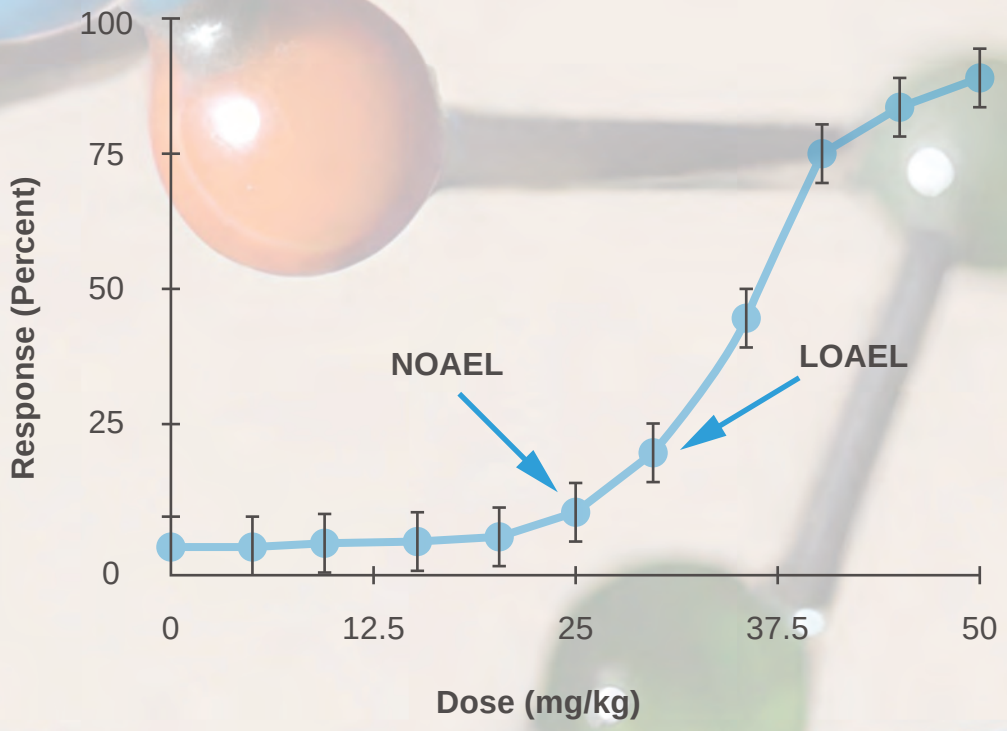


Figure 2. Dose-response Curve
Source: ChemSafetyPRO (2022)[9]

Table 2. Fundamental Concept in Toxicology

Concept Toxicology	Description
Dose	The amount of a chemical/toxicant an organism receives as the result of some exposure. As the dose of a toxicant is increased, the response or effect also increases. The dose is dependent on the concentration and duration of exposure to the toxicant.
Response vs Effect	The terms 'response' and 'effect' are used differently especially in occupational toxicology. 'Response' is used to refer to the proportion of a population showing a specified change whereas 'Effect' is referring to the actual magnitude of the change in the parameter. For example, a number of people showing liver damage upon exposure to chloroform at different concentrations of exposure in a population – this is referred to as response. In the same scenario, the measurement of liver enzyme concentration across various concentrations of chloroform exposure will be referred to as effect.
Dose-Response Curve	The relationship between dose and response or effect is often depicted graphically using log (dose) against response or effect arithmetically or using a probit scale. The dose is usually expressed in mg/kg and depicted on the x-axis, whereas the response is expressed as a cumulative percentage of animal/human that exhibits the toxic effect under study that is expressed on the y-axis of the graph. From the dose-response curve, potency, and efficacy of between chemicals can be compared. In addition, the dose response curve from repeated dose studies may derive the NOAEL or LOAEL of a chemical, which can be used to calculate the OEL (Figure 2).

No Observed Adverse Effect Level (NOAEL)	The dose of a chemical at which no adverse health effects are identified between the study animals or population (the group exposed to the chemical) and the control animals or population (the group with no exposure to the chemical).
Low Observed Adverse Effect Level (LOAEL)	Low observed Adverse Effect Level (LOAEL) is the lowest dose of a chemical at which adverse health effects are identified between the study animals or population (the group exposed to the chemical) and the control animals or population (the group with no exposure to the chemical).

2.1.4 Factors Influencing Toxicity

There are several factors that can influence toxicity of a chemical upon occupational exposure. By knowing these factors, proper workplace management can be planned for the safety and well-being of employees.

- **Duration and Frequency of Exposure**
  - Duration refers to how long the dose is received, whereas frequencyrefers to how often the dose is received. Acute exposures are usually single incidents of relatively short duration (minutes to a few days). Chronic exposures involve frequent doses at relatively low levels over a period of time ranging from months to years. The body can sometimes defend itself against toxic effects when the rate of chemical entry is slower than the rate of detoxification. However, rapid administration with the same dose for the same chemical can produce toxic effects.

- **Routes of Exposure**
  - The toxic effects can be different for the same dose, depending on whether the chemical is inhaled, ingested, applied to the skin, or injected. A dose absorbed through the skin will be deposited in the blood much slower than a dose inhaled through the lungs and transferred directly into the blood.

- **Inter and Intra-species Variations**

  - Inter-species variations

    - The toxic effects exhibited by different species may varygreatly for the same dose received under similar conditions. Since toxicological effects of chemicals on humans are often based on animal studies, the animal model selected should resemble the human physiological processes.

  - Intra-species variations

    - In a population, the toxic response to a chemical may varyfrom the most sensitive to the most resistant group upon exposure to similar dose. Several factors attributed to these variations as follows:

      - Age and maturity – The sensitive groups in general populations are the children and elderly. These groups are more susceptible to toxic effects at relatively lower doses than younger adults.

    - Gender and Hormonal Status

      - Some chemicals are more toxic to one gender than the other. For example, 1,2-dibromo-3-chloropropane, which was previously used as fumigant and pesticide, is well-characterized male reproductive toxicant[54]. Females generally have a higher percentage of body fat than males, therefore they may accumulate more lipid soluble chemicals. Some variations in toxic responses have also been attributed to physiological differences between males and females especially with regards to hormonal status.

    - Genetic Makeup

      - Genetic factors play an important role in influencing individual responses to chemicals. Genetic polymorphisms can sensitize some individuals to a given chemical. For example, individuals with NQO1 polymorphisms have been shown to have a greater risk of aplastic anemia and leukemia as compared to individuals with wild type NQO1 upon chronic exposure to benzene[39].

    - State of Health

      - Health individuals are generally more resistant to toxic effects of chemicals as compared to individuals with poor health. Pre-existing disease or other medical conditions can result in greater sensitivity to toxic agents.

- Environmental Factors
  - Environmental factors such as environmental pollutants and workplace conditions can affect how an individual responds towards a chemical exposure.

- Chemical Combinations
  - Some combinations of chemicals produce different effects from those attributed to each individually.
    - Potentiation describes a situation where a chemical that does not produce a specific toxicity, nevertheless, increases the toxicity caused by another chemical when both are present (0 + 1 = 3).
    - Addition describes a situation where both chemicals produce the magnitude of toxicity of interest equal to sum of both toxicities as exerted by the chemical individually (1 + 1 = 2).
    - Synergism describes a situation when two chemicals that produce the toxicity of interest combined causes a greater-than-additive effect in the anticipated response (1 + 1 = 5).
    - Antagonism describes a situation when a chemical diminished another chemical effect (1 + 0 = < 1).

2.1.5 Concept in Toxicology

The “dose makes the poison” is a well-known concept in toxicology. In other words, any chemical can be toxic if too much is exposed to the body. There are two important aspects of toxicology to fully understand the underlying process leading to the toxic effect from chemical exposure – toxicokinetic and toxicodynamic. Toxicokinetic describes how a toxicant enters the body and reaches a target tissue. Toxicodynamic describes what happens to that tissue once the toxicant reaches an effective dose. Table 3 describes the definition of toxicokinetic and toxicodynamic.

Table 3. Definition of Toxicokinetic and Toxicodynamic

Toxicokinetic	Toxicodynamic
What the organism does with the toxicant	What the toxicant does to the organism

Basically, toxicokinetic is the study of absorption, distribution, metabolism/biotransformation, and excretion (ADME) of the chemical in relation to time. As for toxicodynamic, this process refers to the effects that a chemical has on an organism following a defined exposure (Figure 3).

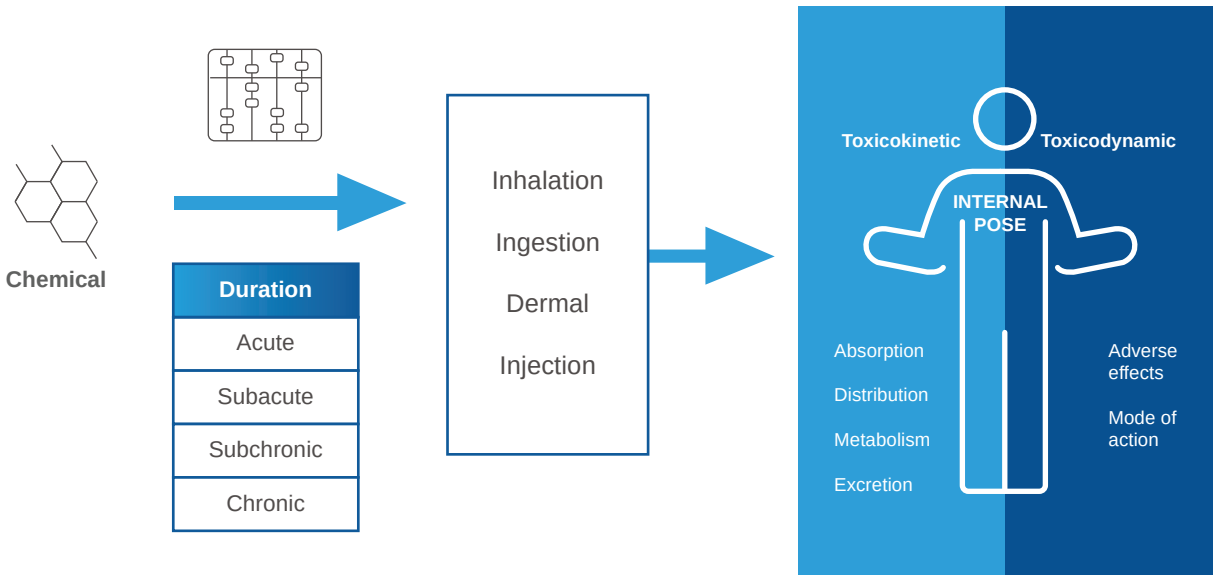


Figure 3. Summary of ADME

Another important concept in toxicology is risk is a function of toxicity and exposure. Herein, risk refers to probability for an adverse effect to occur. Toxicity is the inherent property of the chemical and this characteristic cannot be modified. In contrast, the exposure factor of the chemical can be modulated easily. To lower the risk of chemical injury during handling, the exposure can be controlled and managed by using proper protective equipment.

2.2 TOXIKINETIC

Toxicokinetic refers to the movement and fate of chemicals in an organism. It includes the process of absorption, distribution, metabolism/biotransformation, and excretion (ADME) of the chemical in relation to time in the organism (Table 4).

Table 4. Summary of Absorption, Distribution, Metabolism and Excretion (ADME)

ABSORPTION	Process by which the chemical enters the body (more specifically the blood).
DISTRIBUTION	Movement of the chemical between various compartments in the body.
METABOLISM	Mechanisms by which the chemical is structurally altered into another chemical entity.
EXCRETION	Clearance of chemical and its metabolites from the body.

2.2.1 Absorption

There are several major routes (pathways) by which toxic chemicals gain access to the body. The route of exposure is how the toxic chemical enters or comes into contact with the body. Following exposure, absorption of chemicals eventually into the circulatory system depends on the physiological and anatomical aspect of the entry organ. The same chemical can cause different patterns of adverse effects depending on the route of exposure. The site of absorption that corresponds to the site of chemical exposure is described in Table 5.

Table 5. Examples of CHTH and its Site of Absorption

Site of Absorption	Example
Gastrointestinal tract (ingestion)	Lead absorption via gastrointestinal tract from ingesting lead-painted ceramic dishes and toys. [27]
Lungs (inhalation)	Benzene absorption via the lung from inhaling the solvent vapour. [49]
Skin (topical, percutaneous, or dermal)	Nicotine absorption via skin resulting in green tobacco sickness among employees harvesting tobacco. [10]
Parenteral (intravenous intramuscular injection)	Chemical exposure via injection can occur when handling chemically contaminated items such as broken glass, plastic, pipettes, needles, razor blades, or other items capable of causing punctures, cuts, or abrasions to the skin causing chemicals to enter the bloodstream.

The effect and response are the greatest and most rapid if the chemical exposure via intravenous injection whereas dermal absorption usually exerts the lowest effect and least response (Figure 4).

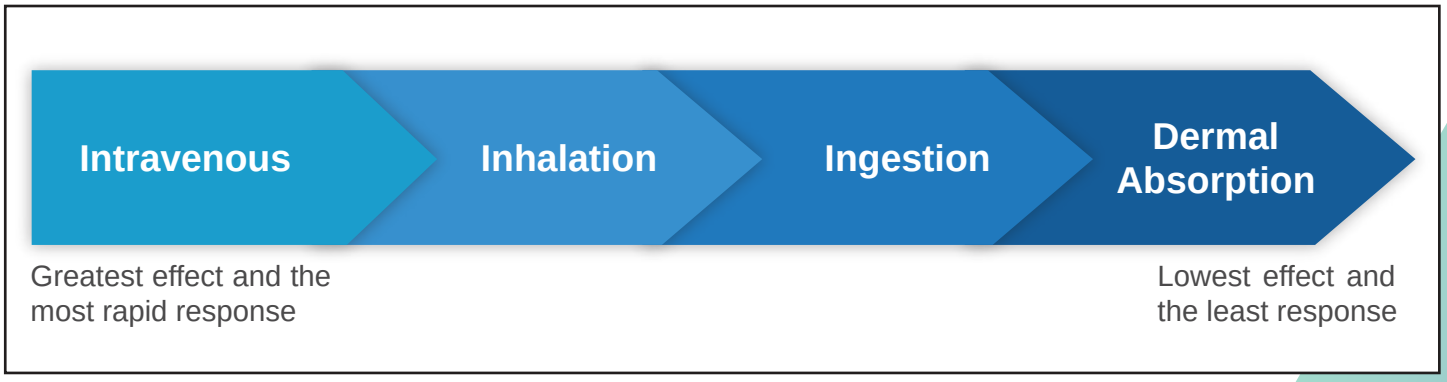


Figure 4. Routes of Exposure

2.2.2 Distribution

Once in the bloodstream or circulatory system, the chemical will be distributed throughout the body. There are several factors that affect the distribution of chemical into various tissues and organs of the body such as:

- **Physical and chemical properties**

  Chemicals that are highly stable and lipophilic, are distributed and concentrated in body fat where they are retained for a long time.

- **Concentration of the absorbed chemical**

  In general, the absorbed chemical will be widely distributed throughout the body according to its concentration in the bloodstream.

- **Blood flow to the tissue or organ**

  Highly perfused tissues or organs are more exposed to the absorbed chemicals.

- **Rate of diffusion out of the capillary bed into the cells or organs**

  Chemical that can easily and readily diffuse through the capillary has a high diffusion rate into the tissue or organ.

- **Extent of protein binding**

  Chemicals that are highly bound to protein such as albumin will remain in the circulatory system, thus giving a low distribution to the tissue or organ.

- **Barriers that inhibit chemical migration**

  The cells in blood brain barriers contain transporters that are able to return the chemical that diffuses into the cell back into the blood circulation. Therefore, the brain is protected from many absorbed chemicals.

2.2.3 Metabolism

Metabolism also known as biotransformation is a process whereby a chemical is altered via enzymatic reaction in living organisms. Generally, the objective of metabolism is detoxification whereby this process promotes excretion of chemicals by enhancing their water solubility. Metabolism can also produce a more reactive metabolite than the parent chemical, a process called 'toxication'.

2.2.4 Excretion

Chemicals are excreted or eliminated from the body via several routes such as renal and faecal excretion, body secretions and exhalation as described in Table 6.

Table 6. Common Routes of Excretion for CHTH

Route of Excretion	Description
Renal excretion	Water soluble by-products / metabolites from chemical metabolism is usually a prerequisite to the excretion of chemicals through urine.
Faecal excretion	Faecal excretion is the second major pathway for elimination of chemicals. It can either be non-absorbed ingested chemicals or biliary excretion of the absorbed chemicals from the gastrointestinal tract.
Body secretions	Some chemicals can be eliminated via saliva, sweat and breast milk.
Exhalation	Absorbed chemicals that are volatile can be eliminated by exhalation from the lungs. For example, some portion of benzene in blood can rapidly be eliminated via exhalation over time.[17]

2.3 TOXICODYNAMIC

Toxicodynamic describes the mechanism of action of chemicals, how they can cause tissue damage, and under what conditions in terms of tissue concentrations and time of tissue exposure/dose do adverse effects on tissue structure and function occur. In a simpler term, this aspect of toxicology refers to the effects that a chemical has on an organism following exposure.

2.3.1 Health Effects

Human health effects caused by exposure to toxic substances fall into two categories: short-term and long-term effects.

- Short-term health effects (or acute effects) have a relatively quick onset (usually minutes to days) after brief exposures to relatively high concentrations of material (acute exposures).
- Long-term health effects (or chronic effects) are those with a long period of time (usually years) between exposure and injury.

These health effects may occur after apparent recovery from acute exposure or as a result of repeated exposures to low concentrations of chemicals over a period of years (chronic exposure). The effect may be local or systemic.

- Local health effects occur at the site of contact between the toxicant and the body. This site is usually the skin or eyes, but includes the lungs if irritants are inhaled or the gastrointestinal tract if corrosives are ingested.
- Systemic health effects are those that occur if the toxicant has been absorbed into the body from its initial contact point, transported to other parts of the body, and causes adverse effects in susceptible organs.

Health effects manifested from acute or chronic exposure are dependent upon the chemical involved and the organ it affects. Most chemicals do not exhibit the same degree of toxicity for all organs. Usually, the major effects of a chemical will be expressed in one or two organs. These organs are known as target organs which are more sensitive to that particular chemical than other organs. Table 7 describes the major organ/ system toxicity that can be adversely affected following chemical exposure.

Table 7. Effects of Chemical Exposure to Major Organ/System

System/Organ	Description
Respiratory Tract	● Inhaled particles vs chemical vapours or gasses ● Particles are small, tiny matters. ● Gas is a state of matter. ● All vapours are gases, not all gases are vapours. ● A vapour will condense if pressurised whereas a gas will not condense if pressurised. ● Inhaled particles settle in the respiratory tract according to their diameters: - 5–30 µ particles are deposited in the nasopharyngeal region. - 1-5 µ particles are deposited in the tracheobronchial region. - Less than 1 µ particles are deposited in the alveolar region by diffusion and Brownian motion. ● In general, most particles 5-10 µ in diameter are removed. ● Certain small inorganic particles settle into smaller regions of the lung and kill the cells which attempt to remove them leading to fibrosis or scarring of the lung. ● Many chemicals used or produced in industry can produce acute or chronic diseases of the respiratory tract when they are inhaled. ● These chemicals can be classified as follows (based on mode of action): - Asphyxiants: CO, CO^2, Helium - Irritants: Ammonia - Necrosis inducers: Ozone - Fibrosis inducers: Asbestos - Allergens: Isocyanates - Carcinogens: coke oven emissions, Arsenic ● Respiratory tract represents a route for chemicals to reach other organs.
Skin	● Absorption of a toxic chemical through the skin can lead to local effects through direct contact, such as irritation and necrosis, and systemic effects. ● Many chemicals can cause a reaction with the skin resulting in inflammation called dermatitis. ● Primary irritants - ethylene oxide, hydrogen chloride, iodine, methyl ethyl ketone, and mercury. ● Photosensitizers - tetracyclines, acridine, creosote, pyridine, furfural, and naphtha. ● Allergic sensitizers – toluene diisocyanate, chromic acid and chromates, cobalt, and benzoyl peroxide.

System/Organ	Description
Eyes	● The eyes are affected by the same chemicals that affect skin, but the eyes are much more sensitive. ● Some chemicals act on eye tissue to form cataracts, damage the optic nerve, or damage the retina. ● These compounds usually reach the eye through the blood having been inhaled, ingested, or absorbed rather than direct contact. ● Examples: - Inorganic lead, methanol leading to retinal damage. - Carbon disulfide, ethambutol leading to optic nerve damage. - Naphthalene, phenothiazines, lens damage (cataract).
Nervous System	● Neurons are readily affected by both simple asphyxiants and chemical asphyxiants because of their need for oxygen. ● Chemicals such as barbiturates reduce respiration and thus reduce oxygen content of the blood to the brain. ● Chemicals such aniline, and benzene reduce blood pressure or flow due to cardiac arrest, extreme hypotension, haemorrhaging, or thrombosis. ● Some compounds damage neurons or inhibit their function through specific action on parts of the cell.
Liver	● Liver injury induced by chemicals has been known as a toxicologic problem for hundreds of years. ● It was recognized early that liver injury is not a simple entity, but that the type of lesion depends on the chemical and duration of exposure. ● Biotransformation of chemicals. ● Acute liver injury - Cell death (carbon tetrachloride) ● Chronic liver injury - Cirrhosis (carbon tetrachloride) - Carcinoma (vinyl chloride)
Kidneys	● The kidney is susceptible to toxic agents for several reasons: - The kidneys constitute 1% of the body's weight but receive 20-25% of the blood flow (during rest). - The kidneys have high oxygen and nutrient requirements because of their workload. - Changes in kidney pH may increase passive diffusion and thus cellular concentrations of toxicants. - Active secretion processes may concentrate toxicants. - Biotransformation is high.

System/Organ	Description
	• Many metals are nephrotoxic including cadmium, chromium, lead, mercury, platinum, and uranium. • Halogenated hydrocarbons such as chloroform, tetrafluoroethylene is nephrotoxic.
Bone Marrow and Blood	• Bone marrow is the source of most components in blood. • Chemicals such as benzene suppress the function of bone marrow and can lead to risk of getting leukemia. • While carbon monoxide combines reversibly with haemoglobin, some chemicals cause the haemoglobin to change such that it cannot combine reversibly with oxygen leading to methemoglobinemia (sodium nitrite, nitrobenzene, trinitrotoluene).
Reproductive System	• Certain chemicals interfere with the reproductive capabilities of both sexes, causing sterility, infertility, abnormal sperm, low sperm count, and/or affect hormone activity. • Male: Anesthetic gases (halothane, methoxyflurane) cadmium, mercury, lead, boron, methyl mercury, DDT, kepone, chlordane, PCBs, dioxin, 2,4-D, 2,4,5-T, carbaryl, paraquat, dibromochloropropane, ethylene dibromide, benzene, toluene, xylene, and ethanol. • Female: DDT, parathion, carbaryl, diethylstilbestrol (DES), PCBs, cadmium, methylmercury, hexafluoroacetone, and anesthetic gases.
Developmental	• Most major structural abnormalities occur during the embryonic period, 5-7 weeks, whereas physiologic and minor defects occur during the foetal period, 8-36 weeks. • Teratogens - Antibiotics: Penicillin, tetracyclines, and streptomycin. - Heavy metals: Methyl mercury, mercury salts, lead, thallium, selenium, and chelating agents. - Azo dyes: Trypan blue, Evans blue, and Niagara sky blue 6B. - Producers of anoxia: carbon monoxide and carbon dioxide.
Mutagenic and Carcinogenic	• Mutagens are agents that cause changes (mutations) in the genetic code, altering DNA. • The changes can be chromosomal breaks, rearrangement of chromosome pieces, gain or loss of entire chromosomes, or changes within a gene. • Chemicals shown to be mutagenic in humans are ethylene oxide, benzene, and hydrazine. • Carcinogenesis can either be genotoxic or non-genotoxic (epigenetic) process.

2.4 TOXICOLOGICAL INFORMATION

Information about chemical hazards and toxic substances is important for better management of these agents for a safer environment including at the workplace. This information can either be obtained from toxicity tests or epidemiological and clinical studies.

- **Toxicity tests**

  In silico toxicology is one type of toxicity assessment that uses computational methods to analyse, simulate, visualize, or predict the toxicity of chemicals. The aim of this type of study is to predict the toxicity of a chemical such as mutagenicity or organ toxicities.[21]

  In vitro toxicology can be defined as the toxicological assessment using non whole animal models. The models used in this area of testing include cell culture, organ culture and bacteria culture. Together with in silico prediction, in vitro toxicity tests represent an alternative to replace animal testing and this is in accordance with the 3Rs principle. This principle involves refining animal use to lessen or avoid pain and distress and enhance animal well-being, reducing the total number of animals required for specific studies, and replacing animals with non-animal systems and approaches.[61]

  In vivo toxicology represents the toxicological assessment using animal models. Toxicity testing in animals is conducted to identify possible adverse effects resulting from exposure to an agent and to develop dose-response relationships.[33]

- **Epidemiological and Clinical Studies**

  These studies represent another means of relating human health effects and exposure to toxic substances. Epidemiological investigations rely upon a human population exposed to a chemical compared to an appropriate, non-exposed group. Statistical analysis will be conducted to determine whether there is a statistically significant association between health effects and chemical exposure. For clinical studies, individual reports of chemical exposure will be examined.

2.4.1 Uses of Toxicological Information

Toxicological information provides a description of the various toxic health effects inherent to the chemical. This information can be used to compare toxicity data, establish exposure guidelines, and establish classification of chemicals as in CLASS 2013.

- **Comparison of toxicity data**

  - Comparing the LD_{50} of chemicals in animals gives a relative ranking of potency or toxicity of each (Table 8).

DDT (LD_{50} for rats = 113 mg/kg) vs ethyl alcohol (LD_{50} for rats = 14,000 mg/kg).

Table 8. Relative Ranking of Toxicity Potency of Chemicals

LD_{50} Translation	mg/kg - bw (body weight) Toxicities LD_{50} rating	Active Constituent
Toxic in very small doses	0.000001	Botulinum Toxin
	0.02	Dioxin
	<1	Brodifacoum, aldicarb
	2	Strychnine, parathion, 1080
	4	Cyanide
	10	Nicotine, abamectin, Vitamin D
	50	Omethoate
	150	Petrol, Pirimicarb
	180	Fluorine
	250	Caffeine
	280	Paraquat dichloride
	408	Diquat dibromide
	639	2,4-D
	3320	Table salt
	5600	Glyphosphate, Simazine
	11900	Vitamin C
Toxic in very large doses	90000	Water

- **Establishing exposure guidelines**
  - Toxicity data from both animal experimentation and epidemiological studies is used to establish exposure guidelines such as PEL, TDI, etc.
- **Classification of chemicals.**

2.5 CARCINOGENICITY

The term ‘carcinogenicity’ refers to the ability of an agent or a toxicant to cause cancer. The agent that causes cancer is called a carcinogen and can be in the form of chemical, biological or physical agent. Carcinogenesis or cancer formation is a complex process that involves gene mutation as a result of chronic exposure to carcinogen.

2.5.1 Molecular Basis of Cancer

Basically, mutations in the somatic cells are a key event in carcinogenesis. These mutations can either be induced by exposure to carcinogen or inherited from certain genetic diseases. The gain of function such as the activation of proto-oncogenes coupled with the loss of function such as the inactivation of tumour suppressor genes lead to uncontrollable growth of cells that forms cancer (Figure 5).

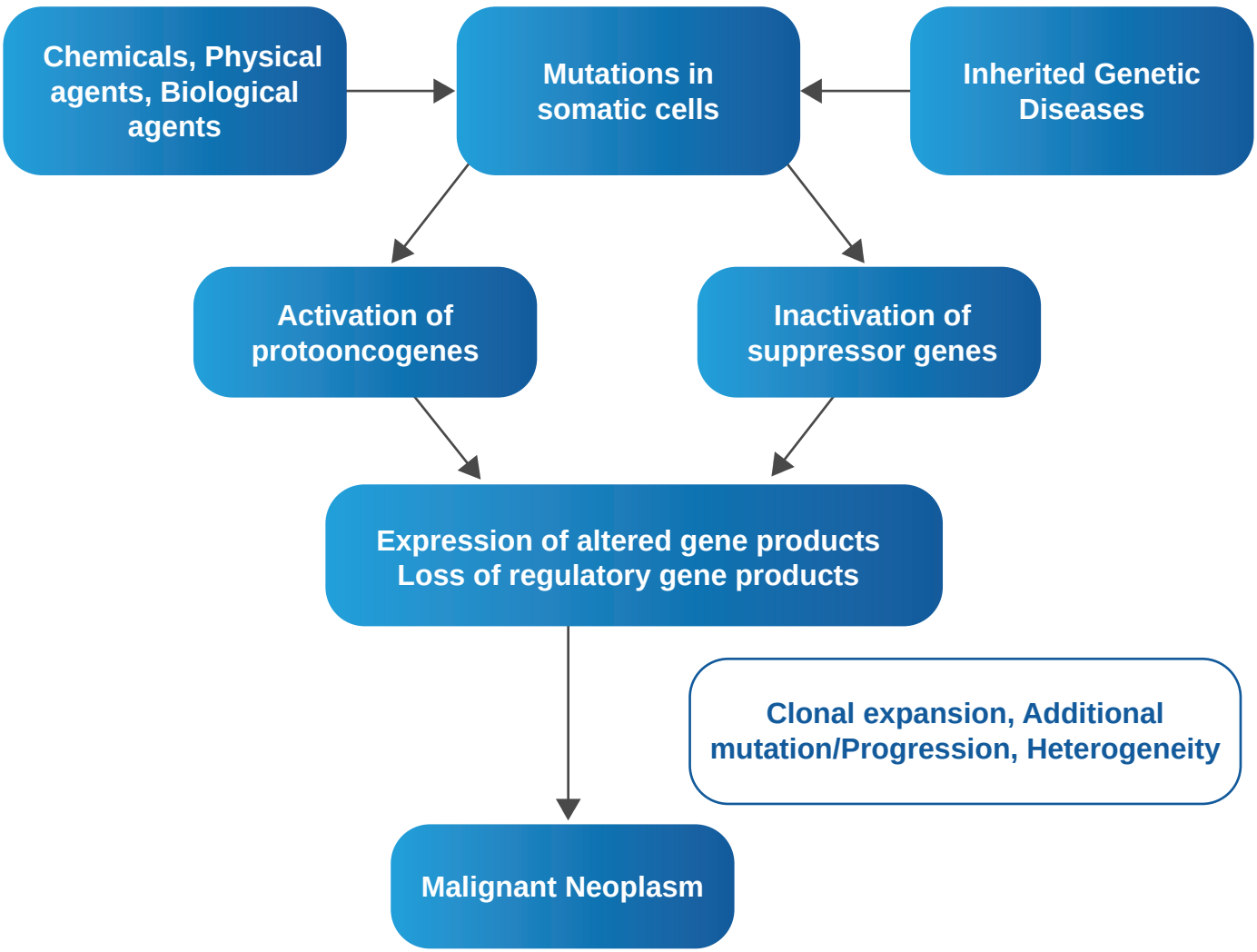


Figure 5. Molecular Basis of Cancer

2.5.2 Evaluation and Classification of Agents

Industry Code of Practice on Chemicals Classification and Hazard Communication 2014 (ICOP on CHC 2014)[15] can be referred to, for guidance on the classification of chemical carcinogens. Criteria for carcinogenicity classification are as in Table 9.

Table 9. ICOP on Chemicals Classification and Hazard Communication: Hazard Categories for Carcinogens

CATEGORY	EVIDENCE	NOTE
1. Known or presumed human carcinogens.	Based on epidemiological and/or animal data.	It may further be distinguished as: 1A & 1B
1A. Known to have carcinogenic potential for humans.	Largely based on human evidence.	Classification in category 1A and 1B is based on the strength of evidence together with additional considerations. Such evidence may be derived from: • Human studies that establish a causal relationship between human exposure to a substance and the development of cancer (known human carcinogen); or • Animal experiments which give sufficient evidence to demonstrate animal carcinogenicity (presumed human carcinogen). On a case-by-case basis, scientific judgement may warrant a decision of presumed human carcinogenicity derived from studies showing limited evidence of carcinogenicity in humans together with limited evidence of carcinogenicity in experimental animals.
1B. Presumed to have carcinogenic potential for humans.	Largely based on animal evidence.	
2. Suspected human carcinogens.	Based on the evidence obtained from human and/or animal studies, but which is	Such evidence may be derived either from limited evidence of carcinogenicity in human studies or from limited

CATEGORY	EVIDENCE	NOTE
	not sufficiently convincing to place the substance in category 1A or 1B, based on the strength of evidence together with other additional considerations.	evidence of carcinogenicity in human studies or from limited evidence of carcinogenicity in animal studies.

Source: ICOP on Chemicals Classification and Hazard Communication (CHC) 2014.

The International Agency for Research on Cancer (IARC) have formulated a system of categories to evaluate the carcinogenicity of an agent to humans. There are 1035 agents that have been classified by the agency whereby 121 agents are in Group 1 (IARC 2022)[22] The IARC classification for chemical carcinogenicity is shown in Table 10.

GROUP	EVIDENCE
1. Agent is carcinogenic to humans.	• Human data strong • Animal data strong
2A. Agent is probably carcinogenic to humans.	• Human epidemiology data suggestive • Animal data positive
2B. Agent is possibly carcinogenic to humans.	• Human epidemiology data weak • Animal data positive
3. Agent is not classifiable as carcinogenic as to humans.	• Human and animal data inadequate

Table 10. IARC Classification Source: International Agency for Research on Cancer

In the case of discrepancy between IARC and ICOP on CHC 2014, unless stated otherwise, ICOP on CHC 2014 takes precedence.

2.6 PERMISSIBLE EXPOSURE LIMITS (PEL)

2.6.1 Pathway from Exposure to Occupational Disease

In occupational settings, exposure is often used as a proxy for dose. The response to a chemical is dependent upon both host factors and dose. The pathway shown in Figure 6 below illustrates from exposure to subclinical disease or to adverse health effect and suggests that there are important modifying factors: contemporaneous exposures, genetic and epigenetic susceptibility, age, gender,

nutritional status, and behavioural factors.[62] These modifying factors can influence whether an employee remains healthy, develops subclinical disease that is repaired, or progresses to frank illness. Workplace health protection and surveillance programmes can reduce exposures, disrupt the exposure-dose pathway, or identify internalized dose and early effects before irreparable disease develops. These programmes help to ensure a safe workplace and a healthier workforce.

Dose is a function of exposure concentration, exposure duration, and exposure frequency. Individual and environmental characteristics can also affect dose. Exposure assessment is the process of quantifying the intensity, frequency, and duration of exposures and their determinants in order to better estimate dose. The enforcement of occupational health standards, the usage of personal protective equipment and the availability of engineering and administrative controls can give some protection to reduce but not eliminate exposure (Figure 6).

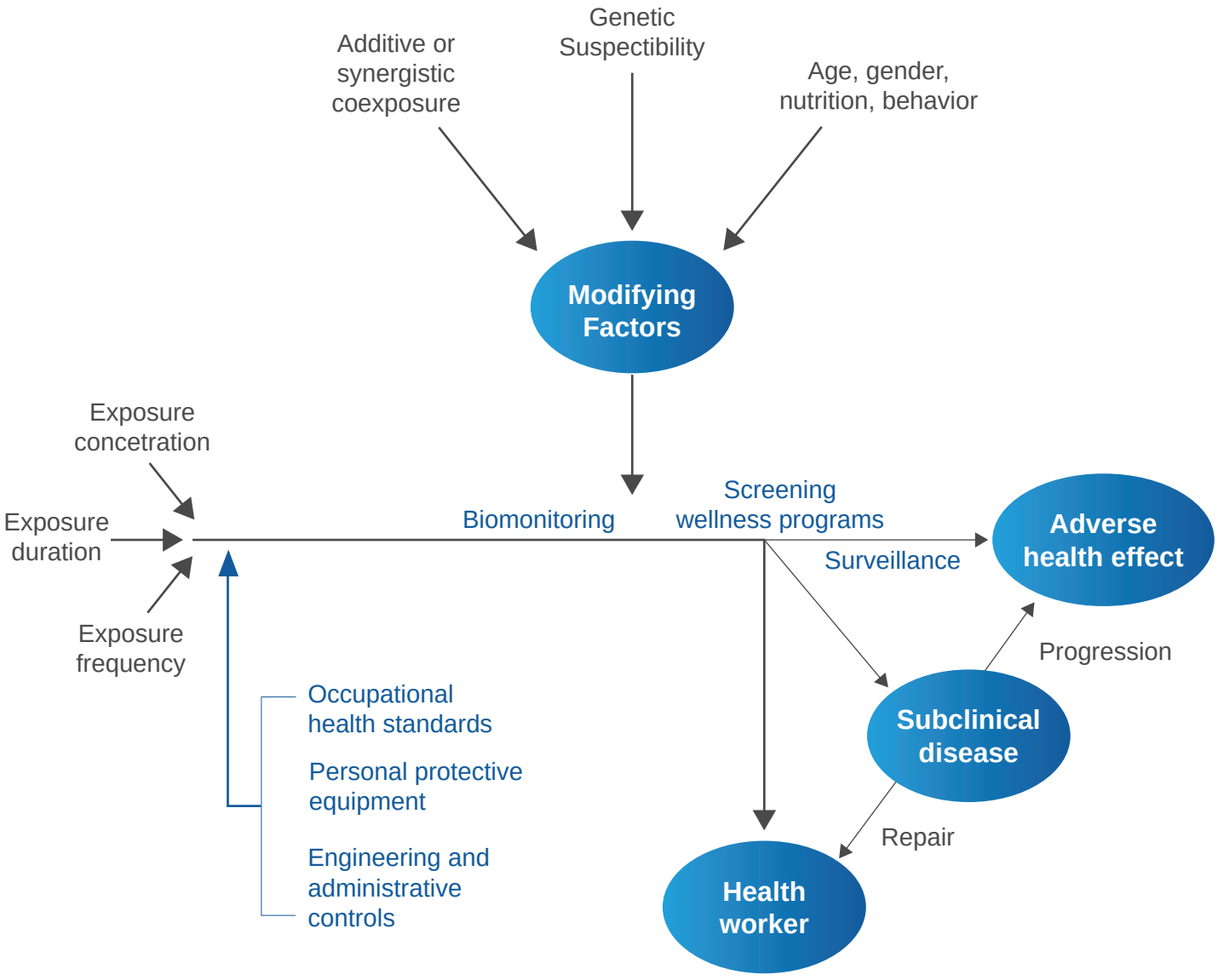


Figure 6. Pathway from Exposure to Occupational Disease

Source: Thorne (2019)

2.6.2 Derivation of PEL

PELs are useful for health professionals to identify toxic substances and their toxic risks in case of human exposure. The derivation of PELs is based on toxicological information such NOAEL from properly conducted animal studies. In the instance where NOAEL is not available, LOAEL can be used by applying an appropriate safety factor.

2.7 CASE STUDY OF TOXICOLOGY

Please refer to Appendix 1 for details on case study of benzene.

3.0 CHEMICAL HEALTH RISK ASSESSMENT (CHRA) & EXPOSURE MONITORING (EM) PROGRAMME

3.1 CHEMICAL HEALTH RISK ASSESSMENT (CHRA)

- Utilization of the CHRA Report
- CHRA Objectives
- CHRA Principle
- CHRA Process Flow
- CHRA Assessment Forms and its Content
- Determination for the Need of MS Programme
- Retrieving information from the CHRA report.

3.2 EXPOSURE MONITORING PROGRAMME (EM)

- Utilization and Validity of the EM Report for CHTH
- EM Methodology
- Types of Air Monitoring
- Utilization of EM Report in MS Programme

3.0 CHEMICAL HEALTH RISK ASSESSMENT (CHRA) & EXPOSURE MONITORING (EM) PROGRAMME

3.1 CHEMICAL HEALTH RISK ASSESSMENT (CHRA)

3.1.1 Utilization of the CHRA Report

Medical Surveillance (MS) programme stipulated under USECHH 2000, is a programme based on a significant risk of exposure to CHTH occurring in the workplace. The risk evaluation is based on the findings of the CHRA. OHD must understand the health risk posed by a CHTH and its contributing components. Therefore, OHD must relate the results of the medical examination and the Biological Monitoring (BM) or Biological Effect Monitoring (BEM) to the workplace risk in order to establish a work-related diagnosis.

3.1.2 CHRA Objectives

The objectives of CHRA are:

- To identify the hazards posed by each CHTH use within the workplace;
- To evaluate the degree of exposure of employees to the CHTH, either through inhalation, dermal or ingestion;
- To evaluate the adequacy of existing control measures; and
- To recommend further appropriate control measures and prioritise actions to be taken to prevent or reduce risks.

OHD must utilize the CHRA report to:

- Obtain the CHTH health hazards information. However, the information may not be adequate for the MS programme. Reference to the specific guidelines and the method for research of hazard information is provided in this guideline.
- Assist in the determination of work-relatedness of a health condition.
- Understand the indication to conduct the MS programme.
- Understand the work unit description.
- Obtain the description of the tasks that expose employees to the CHTH.
- Obtain the information on potential route and extent of the exposure.
- Obtain the information on the control measures being used, the adequacy and the recommendation for improvements.

3.1.3 CHRA Principle

The CHRA is based on this principle:

RISK = HAZARD X EXPOSURE

It comprises hazard identification and the exposure determination and risk evaluation. The assessment is conducted for the inhalation, dermal and ingestion route. For the inhalation route, the hazards and the exposure assessment will be presented as hazard ratings (HR) and exposure rating (ER) of 1 to 5. The higher the rating means the higher the degree of the hazards and exposures. The risk rating (RR) is obtained from the multiplication of both the hazard and exposure rating with a maximum value of 25. Low risk rating has the value of 1 to 4, moderate risk rating has the value of 5 to 12 and high risk rating of 15 to 25.

Level of risk for dermal exposure is categorized into three categories of risk which are low risk (L), moderate risk (M1 & M2) and high risk (H1 & H2).

Conclusion of the risk assessment of a chemical is presented as a level of risk (LOR) and adequacy of the control measures.

3.1.4 CHRA Process Flow

The overall CHRA process flow is shown in Figure 7.

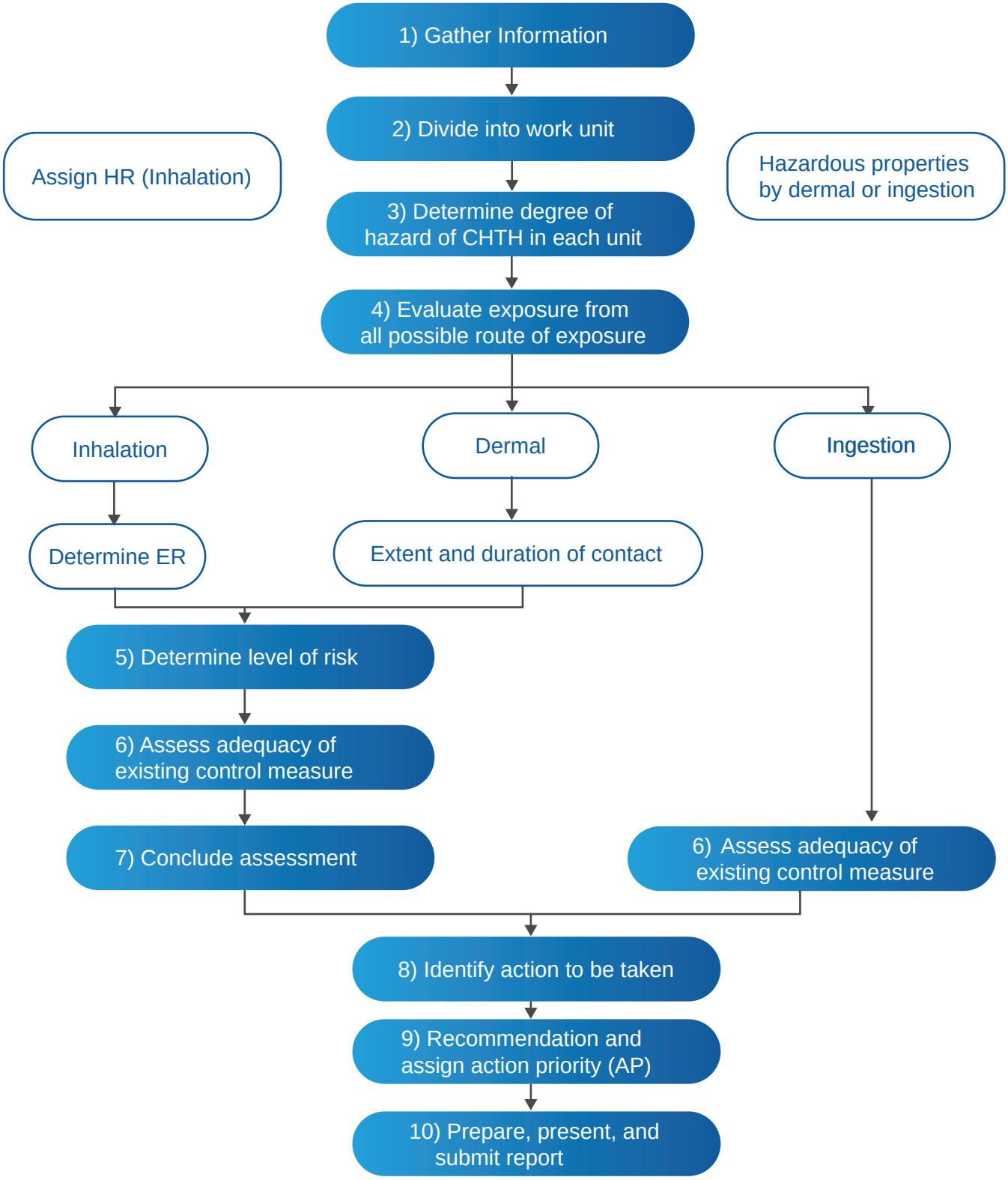


Figure 7. CHRA Process Flow

Source: CHRA Manual Manual 3rd Edition [14]

3.1.5 CHRA Assessment Forms and its Content

The CHRA report is organized according to a standard scientific report. Full assessment information is contained in the assessment forms A, B, C and D will be used to input the information gathered during the assessment. Table 11 shows the CHRA assessment forms and its contents.

Table 11. CHRA Assessment Forms and its Contents

Forms	Contents
A	The information on the work units such as work area, working hours and relevant exposure history. Brief process flow is also noted here; employee health feedback, report on health effects, susceptible conditions, possibility of abnormal exposures, possibility of mixed exposures, possibility of ingestion; and other information. Mixed exposure is the exposure to different chemicals that affect the same target organ. Example: toluene and xylene that affect the central nervous system.
B1	The information about the physical properties of the chemicals and its health hazards posed by all routes of exposure.
B2	The information about the chemicals released by the process or work activities. This form also describes chemicals involved for mixture assessment. Mixture assessment is required when chemicals are mixed and used for the tasks (including dilutions of chemicals).
C1	The information on the tasks and its inhalation exposure evaluation. The conclusion for each chemical will be in the form of Risk Rating (RR) which is from 1 until 25. The higher the RR, the greater the risk.
C2	The information on the tasks and its dermal exposure evaluation. The conclusion for each chemical will be in the form of Level of Risk either low, moderate, or high.
D1	Represents the controls used for each chemical and actions recommended. The conclusion is derived based on the level of risk for both inhalation and dermal exposures. Listed in the Form D1 are also the actions to be taken if the controls are not adequate.
D2	Indicates the adequacy of existing organizational controls and the actions recommended which include safe work system and practices; information, instruction, and training; and personal hygiene.
D3	Indicates the adequacy of existing emergency response preparedness and the actions recommended.
D4	The information on the existing exposure monitoring and MS programme that is applicable in the assessment. Recommendations on the action to be taken regarding these programmes should also be stated in this form.

D5	The specific actions to be taken by the management for the chemical that is known as human carcinogens and respiratory sensitizers. Actions to be taken should also be recommended to control the exposure to these chemicals.

3.1.6 Determination for the Need of MS Programme

As shown in Figure 8, MS Programme is required in the presence of:

1. Significant exposure:

- Air monitoring result is at or above half of 8 hours TWA;
- Air monitoring results exceed ceiling limits (CL), MEL or STEL;
- The chemical pose potential systemic effects through dermal absorption which is indicated as (skin) in Schedule I of USECHH Regulations and the task is performed with a likelihood of dermal contact or absorption;
- Results of biological monitoring exceed BEL.

2. Health effects: Complaints of health effects due to the exposures, based on the known acute and chronic effects of the CHTH.

3. Availability of valid techniques. These methods below, listed according to the effectiveness in detecting ill health effects at early stage:

- BM and BEM (an exposure assessment by determining the internal dose).
- HEM (determining the occurrence of acute and chronic effects).

Determining the need of a MS programme for chemicals when the criteria for the determination may not be confidently applied, requires professional judgement.

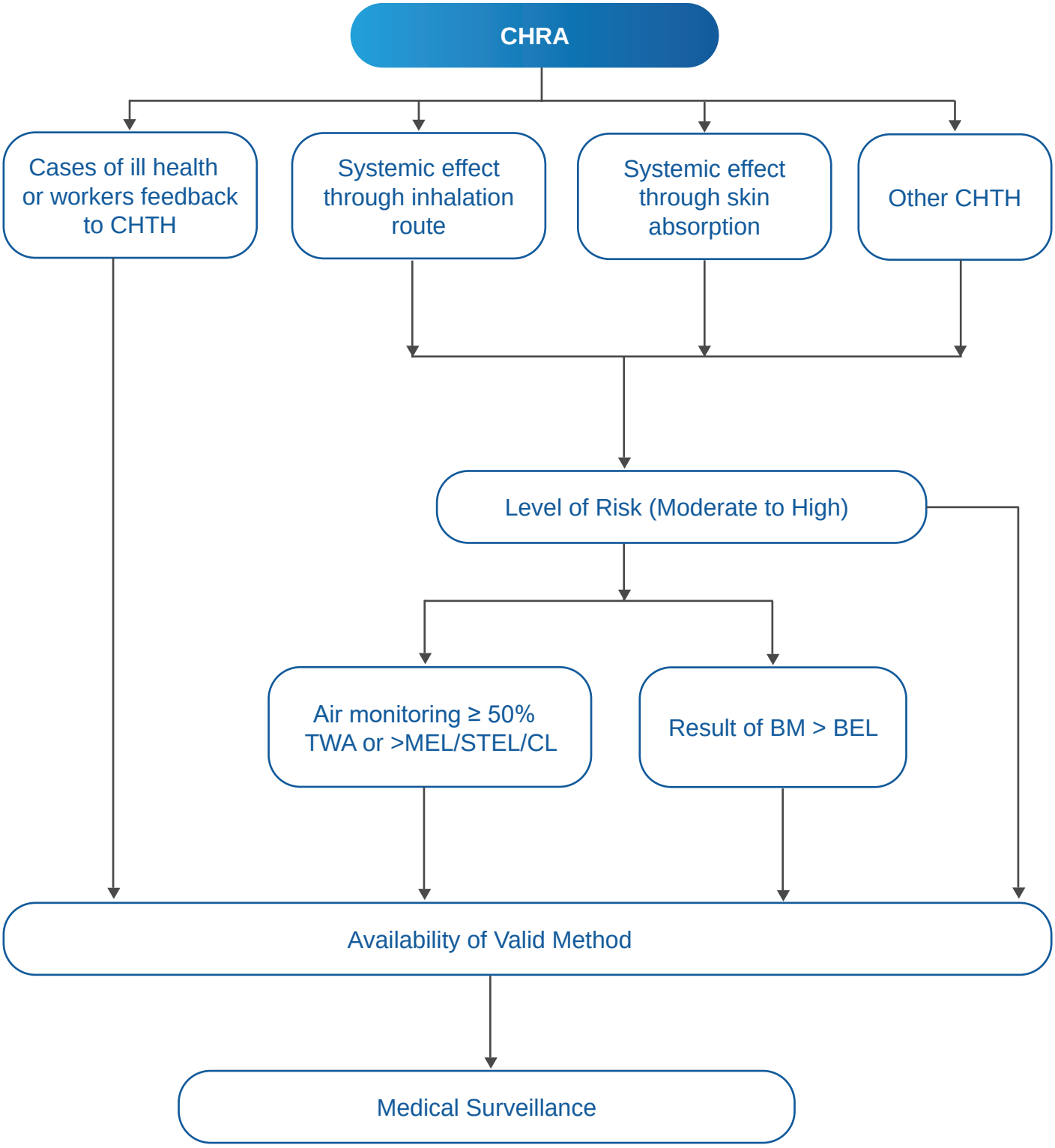


Figure 8. Requirement to Conduct the MS Process Flow

Source: Adapted from CHRA Manual 3rd Edition[14]

3.1.7 RETRIEVING INFORMATION FROM THE CHRA REPORT.

The approach for getting information from the CHRA report process flow is displayed in Figure 9.

- Firstly, the OHD should identify the CHTH that the CHRA has recommended for the MS programme.
- Second, the work unit in concern must be determined. When retrieving information from the CHRA, the OHD should emphasize the chemical and work unit in question. Table 12 provides the relevant information from the CHRA report.

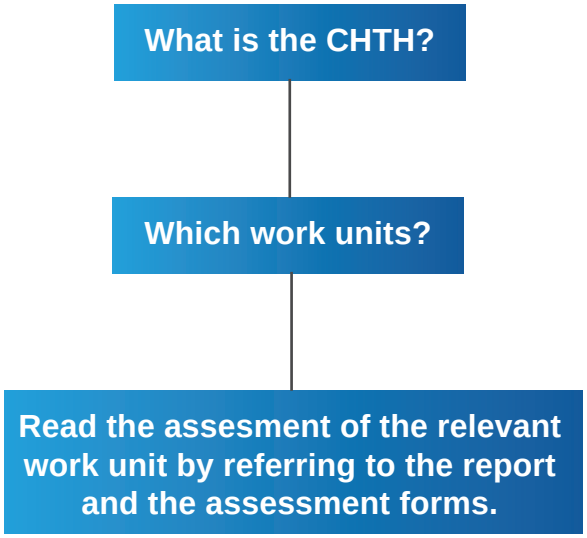


Figure 9. CHRA Report Information Retrieval Process Flow

Table 12. Important Information for The MS Programme Retrieved From the CHRA Report

No	Important information for the MS programme	Information From the CHRA report
1	A chronological summary of all work activities and their duration.	Work unit description and process flow, Form A and C
2	A detailed description of the workplace, of the job and of a typical working day.	The findings, Form A and C
3	An inventory of all chemicals that are present and how they are used. This is to anticipate exposures from other chemicals that can potentiate the health effects.	The findings, Form B and C
4	Details of any measures to limit chemical exposure such as: workplace ventilation and the nature of the protective measures that are taken (requirement to wear special clothing and gloves, the use of respirators, goggles, and other devices).	The findings and the discussions, Form D4
5	Enquiry of exposure monitoring programmes (air and/or biological) and MS programmes are or have been in place and retrieve the result, if necessary, keeping in mind however that compliance with PEL do not necessarily protect all employees from adverse effects.	The findings and the discussions, Form D4
6	Enquire as to whether co-employees have similar symptoms and signs to those of a patient with suspected occupational liver disease. This may involve questioning and even examining co-employees. If several cases come to light, it may be possible to demonstrate an exposure-response relationship.	The work unit's description, Form A
7	Enquire if compensation procedures have been undertaken and results are available.	The work unit's description, Form A

3.2 EXPOSURE MONITORING PROGRAMME (EM)

3.2.1 Utilization and Validity of the EM Report for CHTH

The results of EM are used to determine the need for a MS programme. The air monitoring component of EM represents the employees' inhalation-related exposures. The EM is also used to interpret the results of the BM and BEM. The requirements for airway personal protection are also based on EM data. All EM reports shall be conducted by Hygiene Technician registered with DOSH.

3.2.2 EM Methodology

USECHH 2000 provides that EM is recommended based on the CHRA. The air monitoring component of EM is conducted in line with Airborne Monitoring for CHTH Guidelines 2022 to ensure that employees' exposures to airborne chemicals are within PEL.

3.2.3 Types of Air Monitoring

Area monitoring is done by placing the sampling equipment at the strategic and fixed location of the area, in close proximity to the chemical exposure source. The objectives are to assess the ambient exposure in an area and also the adequacy of the control measures.

Personal monitoring is done by placing the sampling head in the breathing zone of the employee. The sampling will measure the exposure of the employee throughout the sampling duration. The results will be compared with the PEL. The compliance to the PEL is regulated under section 6, 7 and 8 of the USECHH 2000.

The personal sampling results are the basis of the recommendation of a MS programme.

3.2.4 Utilization of EM Report in MS Programme

The EM report includes data on CHTH airborne exposures. OHD should be concerned about the level of exposure in evaluating the work-relatedness of health effects, which may influence the decision regarding MRP and other recommendations. The OHD requires the following information from the EM report:

- Work unit information in the work unit description section of the report.
- Tasks in the work unit description and in the findings section.
- PPE and other control measures. In the work unit description and in the findings section.
- Results in the findings and statistical analysis.
- Discussion about the results.
- Recommendations to reduce the exposures and maintain control.

Below are the lists of factors that can be evaluated by OHD but not limited to:

1. The work unit is the same as the work unit recommended for the MS programme.
2. Determine the work unit and task for EM from the CHRA report.
3. Control measures being used to reduce the exposure of an employee is similar to the control measures listed in the CHRA.

4.0 BIOLOGICAL MONITORING (BM) AND BIOLOGICAL EFFECT MONITORING (BEM)

4.1 INTRODUCTION

4.2 APPLICATION OF BM & BEM

4.3 BIOLOGICAL EXPOSURE LIMIT (BEL)

4.4 APPLICATION OF BEL

4.5 NOTATION IN ACGIH BEI

4.6 CONFOUNDING FACTORS, UNCERTAINTIES AND CONTROLS
- Confounding Factors
- Creatinine Correction

4.7 CONDUCTING BIOLOGICAL MONITORING/BIOLOGICAL EFFECT MONITORING

4.8 BM SAMPLING PROCEDURES
- BM Sampling Time
- BM Sampling Process

4.9 FIELD BLANKS AND OTHER BLANKS

4.10 CHAIN OF CUSTODY (COC) FORM

4.11 IMPORTANCE OF BASELINE SAMPLE

4.12 RESULT INTERPRETATION

4.0 BIOLOGICAL MONITORING (BM) AND BIOLOGICAL EFFECT MONITORING (BEM)

4.1 INTRODUCTION

Biological Monitoring (BM) and Biological Effect Monitoring (BEM) are components of a Medical Surveillance (MS) programme and are used to assess employee's exposure to chemical hazardous to health (CHTH).

4.2 APPLICATION OF BM & BEM

BM serves as a complement to exposure assessment by air monitoring. The interpretation of the BM and BEM data in conjunction with the interpretation of the air monitoring results provides a more accurate representation of actual exposure and risk. Furthermore, this combination may result in discovery of unexpected exposures or routes of exposure, which may help in managing risk. The comparison of BM and BEM with air monitoring is summarised in Table 13.

Table 13. Comparison of BM and BEM with Air Monitoring

BM and BEM	Air Monitoring
Measure the actual body burden caused by CHTH exposure	Measure the airborne level of exposure of employees to CHTH
The finding reflects the total uptake of CHTH by an individual by all routes which are inhalation, ingestion, through the skin or by a combination of these routes	The findings indicate measurement of an individual's inhalation exposure through personal air monitoring, which measures the concentration of CHTH in the air in a person's breathing zone

Source: Adapted from ACGIH TLVs® & BEIs® 2022[1]

Thus, BM are used for these purposes:

a) Part of a Chemical Health Risk Assessment (CHRA) process

b) MS programme

BM is described in the CHRA Manual 3rd Edition (refer to Appendix 13). BM can be considered without the needs for a MS programme provided if there is internal expertise (i.e., trained personnel) who can interpret the results generated from the monitoring. Else employers may seek advice from qualified industrial hygienists. However, the same document refers to the risk assessment process only.

The objectives of BM as exposure assessment as listed in the CHRA Manual 3rd Edition are:

a) To check that control measures are working;

b) To check that work practices are protective; and

c) To check that training is understood and followed.

The above objectives can be also achieved by a MS programme. A BM for exposure assessment as a part of CHRA is not meant to be a periodic programme. A regular and periodic BM, if recommended by the CHRA, shall be considered as a surveillance programme and thus, should be a part of a MS programme.

MS programme objectives in using BM and BEM are:

a) To check that control measures are working;

b) To check that work practices are protective;

c) To check that training is understood and followed;

d) To determine excessive absorption; and

e) To decide on the medical removal protection.

4.3 BIOLOGICAL EXPOSURE LIMIT (BEL)

Biological Exposure Limit is representative of the levels of determinants which are observed in workers exposed to chemicals, exclusively by inhalation at the level of respective Permissible Exposure Limit (PEL). Example of established BEL is Biological Exposure Indices (BEI) by ACGIH which corresponds with their Threshold Limit Value (TLV). Another example is the HSE UK Biological Monitoring Guidance Values (BMGVs) which corresponds with the Workplace Exposure Limit (WEL).

The main reference for this guideline is the **American Conference of Governmental Industrial Hygienists (ACGIH) TLV and BEI 2022.** BEIs are developed by Committee consensus through an analysis and evaluation process. The principal material evaluated by the BEI Committee includes peer-reviewed published data taken from th workplace (i.e., field studies), data from controlled exposure studies, and from appropriate toxicokinetic modelling, when available. The results of animal research are also considered when relevant.

The detailed scientific criteria and justification for each BEI can be found in the **Documentation of ACGIH's Threshold Limit Values and Biological Exposure Indices**. The Documentation provides essential background information and the scientific reasoning used in establishing each BEI. Information given includes the analytical methods, possible potential for confounding exposures, specimen collection recommendations, limitations, as well as other essential information specific for each compound and analyte.

Furthermore, there are BEIs for chemicals for which the TLVs are based on protection against no systemic effects (e.g., irritation or respiratory impairment) where biological monitoring is desirable because of the potential for significant absorption via an additional route of exposure (usually the skin). Therefore, BEIs are used as guidance values for evaluating BM results.

4.4 APPLICATION OF BEL

- BEL does 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 BEL without incurring an increased health risk. The values are inappropriate to use for the general population or for non-occupational exposures. The BEL values are neither rigid lines between safe and dangerous concentrations nor are they an index of toxicity.

- If measurements in specimens obtained from an employee on different occasions exceed the BEL, the cause of the excessive value should be investigated, and action taken to reduce the exposure

- An investigation is also warranted if measurements in specimens obtained from a group of employees at the same workplace and work shift exceed the BEL. It is desirable that relevant information on related operations is included.

- Toxicokinetic and toxicodynamic information is taken into account when establishing the BEL; thus, some knowledge of the metabolism, distribution, accumulation, excretion, and effect(s) is helpful in using the BEL effectively.

- OHD should note that if sequential samples taken early in an employee's exposure career show a marked increase, an overexposure situation might be developing and must be addressed despite the values being below the BEL.

- BEL better predicts health effects than air levels and are based on the levels in the environmentally exposed population.

- The BEL generally indicates a concentration below which nearly all employees should not experience adverse health effects.

- The BEL is not intended for use as a measure of adverse effects or for diagnosis of occupational illnesses.

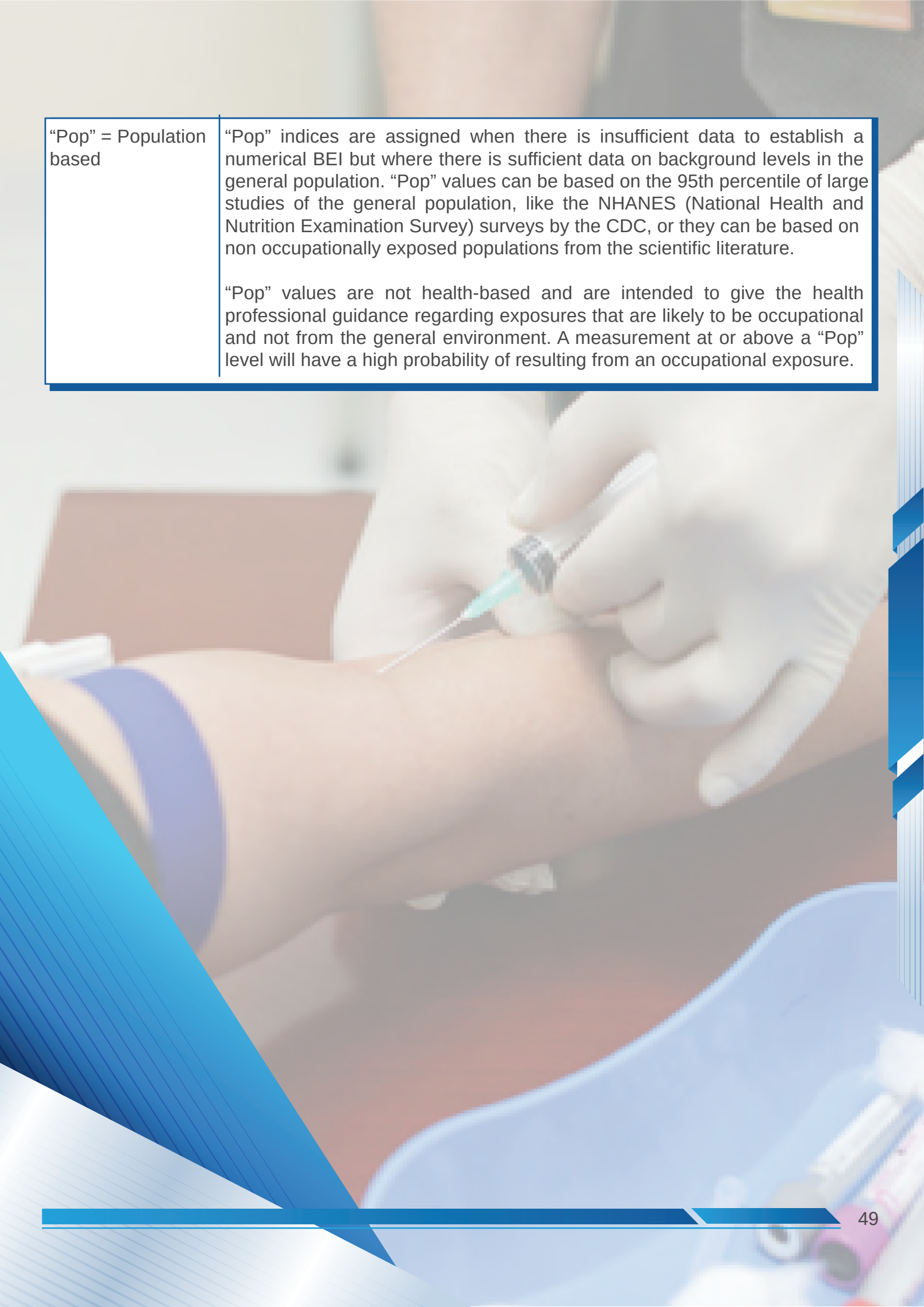
4.5 NOTATION IN ACGIH BEI

When referring to the ACGIH BEI, it is important to understand the notations used in the ACGIH guidelines. Table 14 shows the notations and its meaning.

Table 14. Notations in ACGIH BEI

Notations	Description
"B" = Background (background level)	The determinant may be present in biological specimens collected from subjects who have not been occupationally exposed, at a concentration that could affect interpretation of the result. A "B" notation is assigned to a determinant when the observed 95th percentile value of a random sample, from national population studies, such as the NHANES surveys by CDC, is more than 20% of the BEI. When general population data are not available to make this assessment, the BEI Committee may assign a "B" notation based on its interpretation of the available data in the scientific literature. In this case, the rationale for the notation is provided in the Documentation for the particular Index. Such background concentrations are incorporated in the BEI value.
"Nq" = Non-quantitative	Biological monitoring should be considered for this compound based on the review; however, a specific BEI could not be determined due to insufficient data.
"Ns" = Non-specific	The determinant is nonspecific since it is also observed after exposure to other chemicals.
"Sq" = Semi-quantitative	The biological determinant is an indicator of exposure to the chemical, but the quantitative interpretation of the measurement is ambiguous. These determinants should be used as a screening test if a quantitative test is not practical or as a confirmatory test if the quantitative test is not specific and the origin of the determinant is in question.

“Pop” = Population based	“Pop” indices are assigned when there is insufficient data to establish a numerical BEI but where there is sufficient data on background levels in the general population. “Pop” values can be based on the 95th percentile of large studies of the general population, like the NHANES (National Health and Nutrition Examination Survey) surveys by the CDC, or they can be based on non occupationally exposed populations from the scientific literature. “Pop” values are not health-based and are intended to give the health professional guidance regarding exposures that are likely to be occupational and not from the general environment. A measurement at or above a “Pop” level will have a high probability of resulting from an occupational exposure.



4.6 CONFOUNDING FACTORS, UNCERTAINTIES AND CONTROLS

4.6.1 Confounding Factors

A **confounding factor** may mask an actual association or falsely demonstrate an apparent association between the study **variables** where no real association between them exists.

Confounding factors of the BM results may also be summarised as below:

a) External factors

b) Internal factors (including background levels)

c) Day to day variations

Uncertainties may result from

a. Sampling procedures and transportation (refer section 4.8 and section 6).

b. Laboratory analysis (refer section 6).

The results of BM reflect exposure from all routes, such as inhalation, oral ingestion, and skin absorption, as tempered through determinants half-times that are dependent on the following (external factors):

a) Exposing a chemical and the physical state of its challenge (solid, liquid, or gas). Factors that influence the exposure release of a chemical will also affect the possible uptake into the body.

b) Physical activity (workload) and whether heat stress is present for the employee. This influences the breathing rate of an employee and also his body metabolism.

c) Personal Protective Equipment (PPE), local emission source controls, general workplace controls, and general employee training regarding their use. These are factors that reduce the exposure to the employees.

d) Validity of sampling, storage, transport, and analytical procedures for the determinants. These are possible random and systematic errors of sample handling and analysis.

Other factors that may contribute to the results of the BM (Internal Factors) or Inter-individual Variability:

a) Genetic factors of the employee including gender, race, ethnicity, and family traits. These factors influence the toxicokinetic of a chemical in the body.

b) Homeostatic factors such as body temperature, pH of blood and urine, circadian rhythms, type of symbiotic microorganisms in the body, and integrity of the immune system can also affect the chemical toxicokinetic.

c) Lifestyle controllable factors such as diet; tobacco usage, alcohol, caffeine, and drug intake; and chemical exposures in the home, during commuting, and during recreation. These refer to confounding factors of chemical metabolites.

d) Uncontrollable factors such as medications; medical conditions; shock; depression; schizophrenia; trauma; oxygen lack; ageing; seasonal factors; injury; and environmental factors such as geography; altitude; local temperature and humidity; indoor air pollution; home pollution; commuting mode; types of recreation; and past workplace exposures. Common non occupational confounding factors: examples are seafood's (mercury, arsenic), air pollution (benzene, mercury).

Results may vary day to day due to:

a) Differences in exposure time;

b) Chemical exposure frequency;

c) Chemical exposure intensity;

d) Work practices;

e) PPE wear;

f) Spills and accidents; and

g) Difference in each employee's absorption, metabolism, and excretion of the exposed chemical.

Interpretation of a group's results should consider these factors. Variability between employees in a group is expected due to the above factors.

4.6.2 Creatinine Correction

Creatinine is produced by our bodies at a fairly constant rate. Creatinine is transported by the circulation to the kidneys and excreted in the urine. Creatinine is a waste product found in urine that has been discovered to be a useful indicator of urine concentration. The concentration of urine samples fluctuates throughout the day, which makes analysis difficult. As a result, interpreting the observed value of a chemical in urine is difficult without a method to equalise it (laboratorians call this standardising).

As an example,

- Assume two employees (employee #1 and #2) were exposed to arsenic the day before participating in the BM experiment.
- The employee #1 submitted his urine sample right after getting up in the morning. His urine was dark yellow and had a chemical measurement of 25 ug/L. The laboratory measured the creatinine level in his urine and found that it was 1.25 g/L.
- The employee #2 provided his urine sample later in the day, after drinking several cups of water, and maybe a large, caffeinated soda. His urine sample is almost clear in colour. The laboratory measures the chemical at 17 ug/L and creatinine at 0.85 g/L.
- For employee #1: 25 ug/L ÷ 1.25 g/L = 20 ug/g creatinine
- For employee #2: 17 ug/L ÷ 0.85 g/L = 20 ug/g creatinine
- After correcting for urine concentration, it was discovered that both employees had the same level of chemical (arsenic) exposure. A more accurate assessment of their exposure risks may now be established.

4.7 CONDUCTING BIOLOGICAL MONITORING/BIOLOGICAL EFFECT MONITORING

All employees in a work unit recommended for MS programme in the CHRA report must be examined. The sampling session may take place at a different time than the medical examination session. It is critical to follow the correct sampling time in order to avoid false negative results.

Correct sampling time is crucial for accuracy of the result. False negative results are a common problem for chemicals with short half-life when the correct sampling time is not adhered to.

The following steps are recommended:

a) Reference method - Specific chemical guidelines in this document, or other guidelines.

b) Verify the laboratory on the sampling requirement, transportation, and packaging.

c) Check the laboratory quality assurance programme and the limit of detection.

d) Ensure proper record keeping, i.e., BM sampling forms, consent form, and chain of custody to the laboratory.

e) Report to employer and DOSH.

4.8 BM SAMPLING PROCEDURES

4.8.1 BM Sampling Time

The sampling time of a chemical determinant is influenced by the elimination of half-life and the duration of worker's exposure or shift work. Below are listed the sampling time and elimination half-life recommendations:

a) Very short half-life (less than 1 hour), not practical for routine biological monitoring.

b) Half-life less than 5 hours, end of shifts reflects recent exposure, determinants do not accumulate.

c) Half-life between 5 to 10 hours, collection at end of shifts and of the work week.

d) Half-life of more than 10 hours, levels are cumulative, it reflects recent AND previous exposure

e) Long half-life. Integrate exposure over many years. Specific sampling time is not required

The sampling time is specified in the BEI determinant monitoring of each CHTH and is determined by the duration of retention of the determinant, modified in some cases by practicality (for example, if the peak level is expected several hours after the end of a shift). Substances and determinants that accumulate may not require a specific sampling time. An explanation of the BEI determinant sampling time is as follows:

Sampling Time	Recommended Collection
1. Prior to shift	16 hours after exposure ceases, but before any exposure on sampling day.
2. Prior to last shift	Prior to the last shift of a workweek.
3. Increase during shift	Requires pre-and post-shift sample collection.
4. During shift	Anytime after two hours of exposure.
5. End of shift	As soon as possible after exposure ceases.
6. End of the workweek	After 4 or 5 consecutive working days with exposure.
7. Discretionary/Not critical	At any time.

Important note:

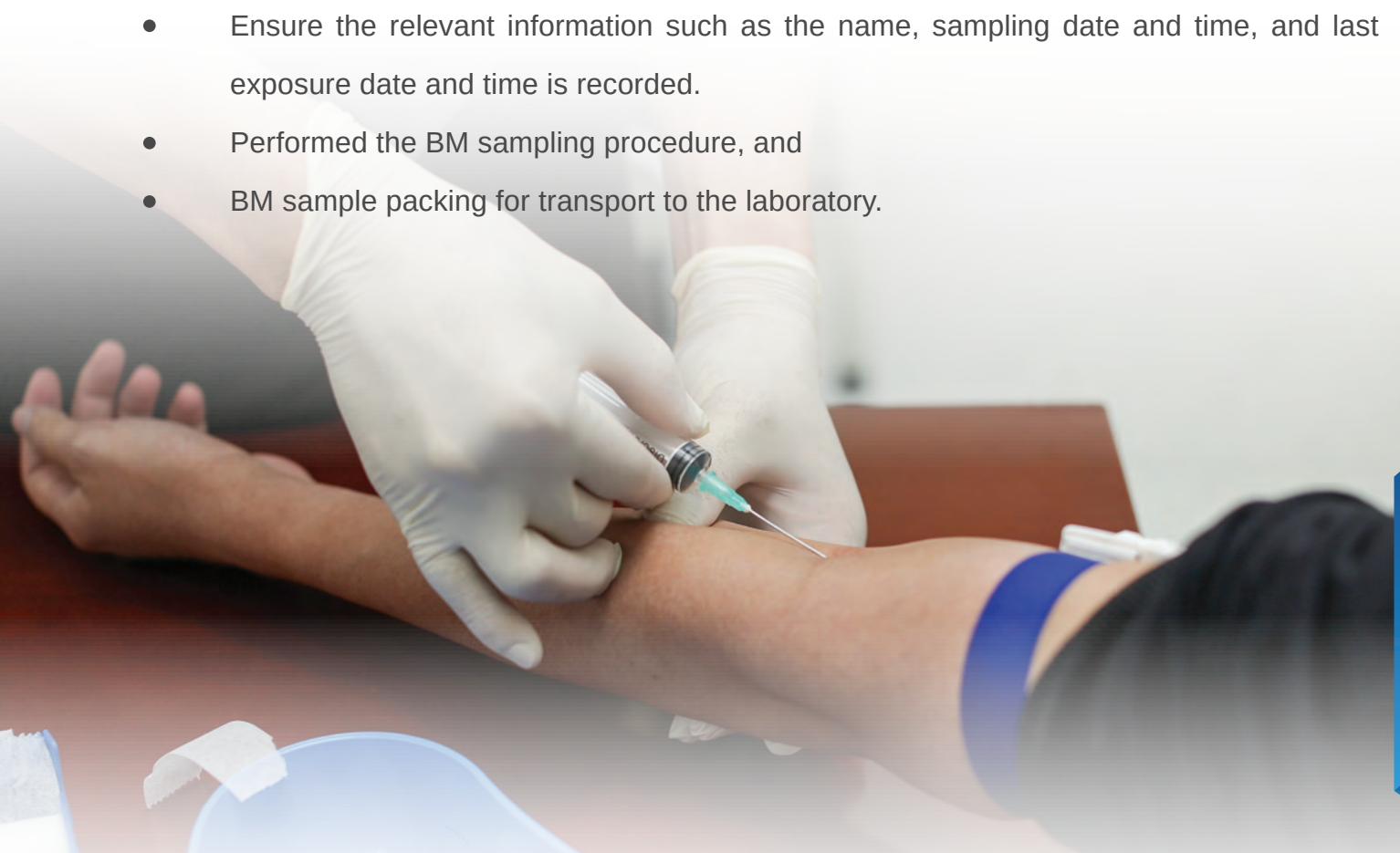
- It is critical that the proper sampling time is within the validity period, in order to avoid lower than actual results or false negative results.
- Detectable results for CHTH with no background level, for samples collected outside of the recommended sampling time period may indicate that the actual exposure was higher. A repeat sample is recommended to determine actual exposure.
- For a MS programme, samples for BM may be taken, separately from the medical examination session, in order to adhere to the recommended sampling time.

4.8.2 BM Sampling Process

OHD needs to ensure that the sample collection is properly done and handled as per these Guidelines.

The sampling process will generally involve these steps:

- Ensure the relevant information such as the name, sampling date and time, and last exposure date and time is recorded.
- Performed the BM sampling procedure, and
- BM sample packing for transport to the laboratory.



Collection procedure for urine sample usually involve these precautions:

1. Sample contamination and shipping are always of concern; it is recommended to contact an analytical laboratory. It will assist in informing the correct containers,preservatives,labelling, holding times, shipping containers, and insulation.
2. It is critical to make adequate preparations before collecting samples, such as removing contaminated clothing, washing hands or skin, showering, and so forth.
3. Adhere to the holding times (the maximum time from sample collection to sample analysis that assures accurate results) and storage conditions may differ for each determinant.
4. Some chemical determinants may require refrigeration immediately following sample collection.

5. Storage conditions also apply to the time the samples remain in shipment to the analytical lab, which may require overnight shipping on ice or dry ice.

4.9 FIELD BLANKS AND OTHER BLANKS

Laboratory will advise on the requirements of field blanks and other blanks as part of the analytical assurance and analytical procedure.

A field blank (container with the appropriate preservative or solvent that is opened, manipulated, and closed in parallel with the sample containers to contain the real samples) is essential for each type of sample to detect any problems in the sampling chain of custody.

Examples of most common sampling errors:

a) Samples were not transported according to the procedure specifications.

b) The sampling time did not meet the requirements. Examples: A post shift sample was collected during pre-shifts.

c) Urine phenol was selected for BM of Benzene where the workplace exposure monitoring results are lower than 5 ppm (urine SPMA is a better option). This could result in false negative results.

4.10 CHAIN OF CUSTODY (COC) FORM

The COC form is usually provided by the laboratory, to ensure validity of the change of the sample custody. This is part of the validation methodology.

4.11 IMPORTANCE OF BASELINE SAMPLE

A baseline sample will usually help in determining true occupational exposures, especially for chemicals with background levels and chemicals with a significant number of confounding factors.

To assess the true occupational exposure, samples need to be collected prior to exposure to establish “baseline” data.

For rapidly metabolised determinants (for example, urine and blood half-times ≤5 hours) this can be accomplished simply by sampling immediately prior to the beginning of the work shift. For example, isocyanates have a short half-life (two to four hours) and samples only reflect recent exposure. The urine samples should be collected immediately post-shift or, if exposure is sporadic, immediately post-exposure.

Baseline samples can reflect holdovers from the previous workday or recent non-occupational exposures.

Pre-placement samples are samples taken before the employee starts work at a new work unit or workplace. If the employee is already exposed in a previous job, the result will reflect previous exposure.

When there is no prior exposure to the chemical, positive results are suspected to be due to non-occupational exposure. This interpretation applies to chemicals with no background levels in the general population.

It is important that the samples are taken when the results are expected to be undetectable, thus applying to chemicals with short half-life and with no background levels.

Importance of baseline samples:

- To provide a level against which the future exposure is compared.
- To identify any previous exposures, irrespective of whether they were occupational or non-occupational.
- To identify individuals who may be exposed to hazardous substances during leisure activities.

Example:

- A baseline sample is part of the BEI determinant protocol for total chromium in urine for water-soluble chromium (VI) fume exposure.
- The urine collected to empty the bladder before the employee begins the shift can be used as the baseline sample. This is because total chromium captures both the Chromium (III) and the Chromium (VI), thus a background level is present.
- Post shift samples when compared with the baseline before shift, will provide the actual exposure of a particular work shift. This procedure can of course be discarded, if we can measure the chromium VI (not the total chromium) in urine samples.

4.12 RESULT INTERPRETATION

There are several important procedures in interpreting BM **urine** sample data.

1) The urine sample acceptance criteria:

- The urine samples should be reviewed, with results discounted if:
  - Creatinine concentration: > 0.3 g/L and < 3.0 g/L

    or

  - Specific gravity: > 1.010 and < 1.030
- Data outside of these reference ranges mean the urine sampling should be discarded and cannot be analysed.
- If values outside the reference range are again obtained, the OHD needs to ascertain whether kidney and liver functions are normal. If these functions are normal, then the out-of-reference-range values can be used. Good judgment should prevail in such cases.

2) Possible false negative results need to be excluded by going through the BM sampling form. The sampling time needs to be within the recommended period.

3) The results should be interpreted with the consideration to the personal protective equipment and consideration of possible confounding factors. For example, a non-detectable result does not indicate that the exposure is insignificant if the employees wore proper personal protective equipment as required by the CHRA recommendation. However, this does indicate that the employee's PPE is effective in avoiding chemical absorption into the body.

Results interpretation process flow for urine samples is shown in Figure 10.

The results interpretations of blood samples do not have specific acceptance criteria. However general precautions such as the sampling time and other procedures still apply.

4.12.1 Urine Sample Results Interpretation Process Flow

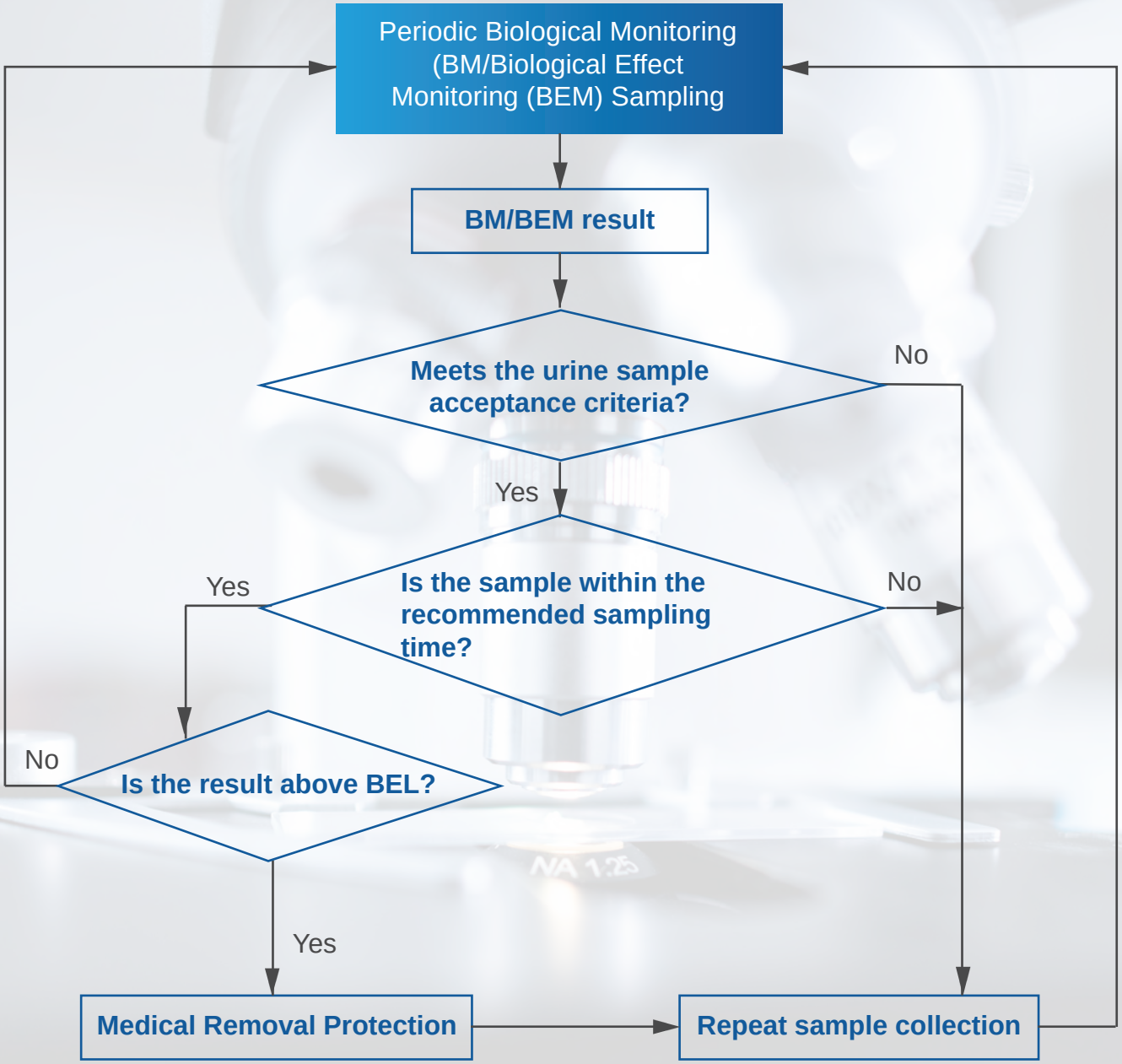


Figure 10. Results Interpretation Process Flow for Urine Samples

Discussed below are the possible outcome of the BM results:

a) Non-detectable results

The OHD should exclude a false negative result as listed below:

- Negative results outside the recommended sampling time, should be discarded and the urine sample repeated.
- Negative results due to non-exposure (chemicals with a short half-life) should be repeated because the objective of the sampling is to determine the internal dose when the employee is exposed to the chemical at workplace and to assess the adequacy of the control measures.

b) Results for CHTH that exceed the BEL

- Review all possible confounding factors and the uncertainties that may affect the results. The OHD needs to determine based on the findings whether there is a need to repeat the sampling.
- Ask the employee about possible failure of the control measures including a breach through the PPE. Examples are the LEV system malfunction and leakages of CHTH from pipelines. Findings should be included in the report to the company.
- Recommend MRP and the RTW assessment date.
- Report to DOSH.
- Provide a report to the company about the findings and the recommendation to improve control measures.

5.0 HEALTH EFFECTS MONITORING (HEM)

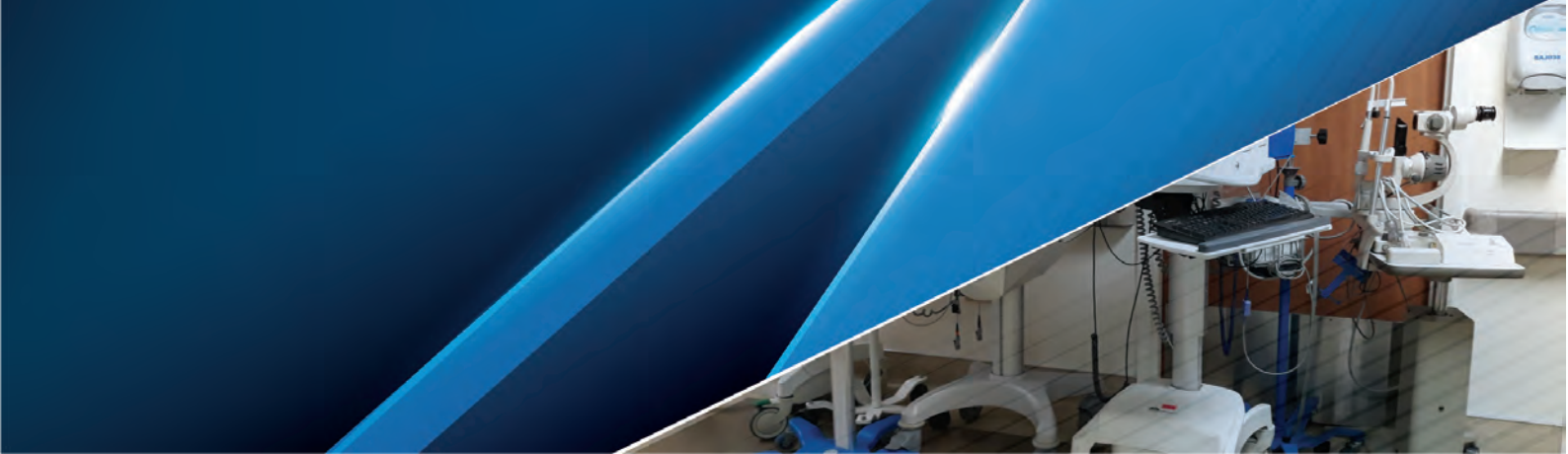
5.1 INTRODUCTION

5.2 GENERAL PRINCIPLES OF HEM

5.3 ROLES OF ACUTE HEALTH EFFECTS DETERMINATION IN MS

5.4 HEM BY TARGET ORGANS

Lungs *refer to Appendix for further information.
Central Nervous System (CNS)
Reproductive System
Skin
Liver
Kidney
Haematology
Cancer



5.0 HEALTH EFFECTS MONITORING (HEM)

5.1 INTRODUCTION

Health Effects Monitoring (HEM) is an important step in MS because:

1) It is the only parameter to monitor health outcome following exposure to CHTH without biomarkers;
2) It is able to show the extent of health insults due to CHTH exposure; and
3) It can identify health insults that may follow an acute high dose exposure to CHTH at work.

Figure 11 displays the adverse outcome pathway following an exposure to CHTH and the role of health effects monitoring in the detection of the health impact.

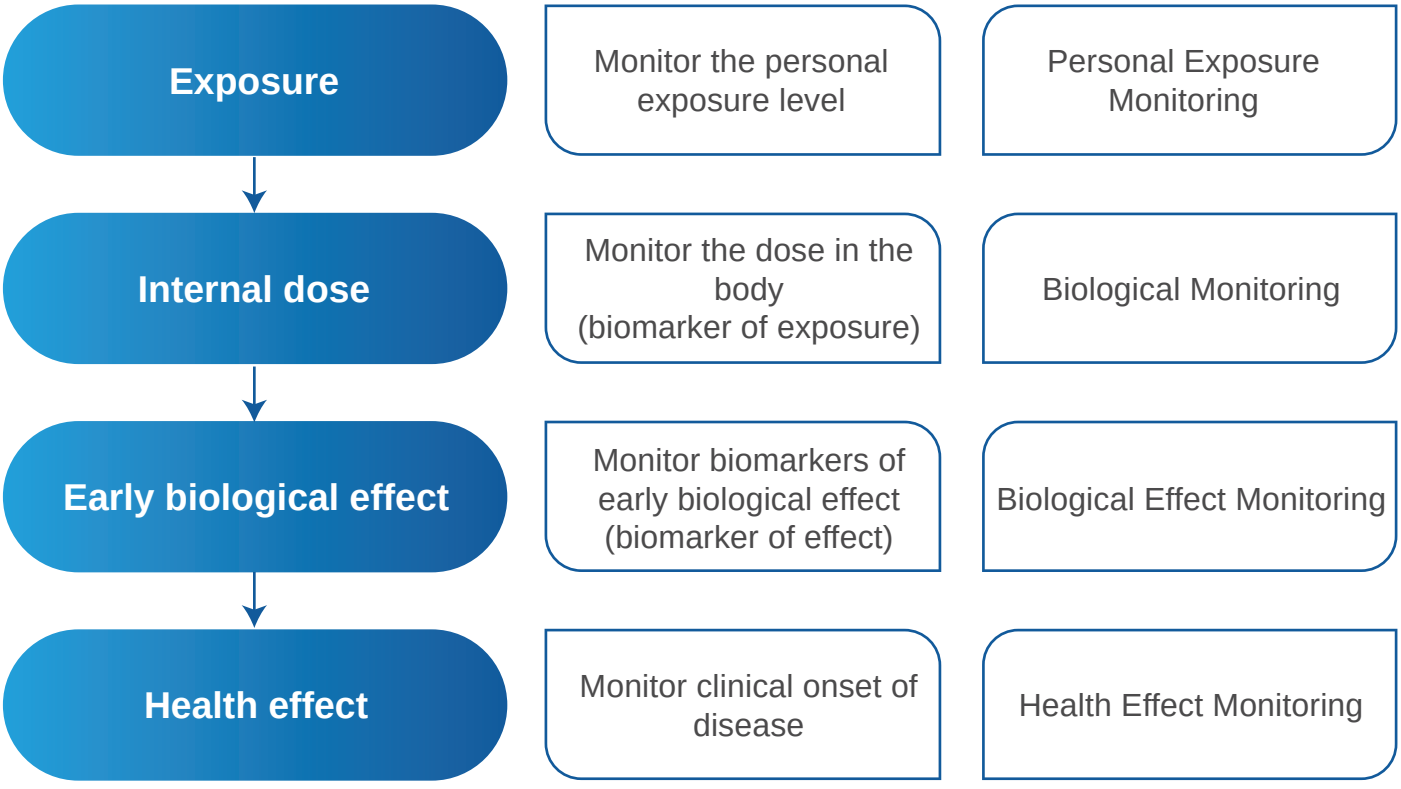


Figure 11. Relationship between HEM and Other Types of Monitoring Parameters

5.2 GENERAL PRINCIPLES OF HEM

- HEM should be able to detect suspected onset of a related disease which shall then be followed-up for confirmation.
- HEM is important for CHTHs without biomarkers of exposure or effect.
- HEM involves the specified duties of OHD which are:
  - Analysis of Occupational Diseases & Poisoning and co-relate with CHRA and/or EM.
  - Investigation of the cause of the Occupational Disease/Poisoning.
  - Notification of Occupational Diseases & Poisoning to DOSH and employer (if applicable).
- It should not be carried out as a perfunctory routine
- The diagnostic criteria of an occupational disease must be fulfilled before it is confirmed and notified. The following criteria are to be considered:

a) The clinical features must fit with the known health effects of the CHTH.

- The health effects that are monitored must be in tandem with specific target organs and toxicity of the CHTH. Knowledge on the toxicokinetics of the CHTH can be useful.
- The symptoms and signs should fit these anticipated health effects.
- Diagnostic tests may be used to support the clinical diagnosis where appropriate.
- Baseline parameters facilitate evidence of changes in health.
- Referral to relevant clinical specialists is made for suspected cases that need clinical confirmation and to rule out other possible non-occupational causes.

b) Indication of sufficient occupational exposure

- Evidence on exposure may be obtained from:
  - The occupational history
  - Exposure monitoring result at the workplace
  - Records of incidents of over-exposure
- The concept of exposure may be used to determine sufficient exposure. The following parameters, if present, may facilitate the decision on the sufficiency of exposure to cause disease:
  - Minimum intensity of exposure
    - The minimum level of exposure that is required to cause disease. Except for carcinogens and allergens where it may not be possible to define the minimum threshold dose.
  - Minimum duration of exposure
    - The shortest exposure period for which disease can occur. Periods of exposure less than this are unlikely to cause disease.
- In some cases, evidence of exposure may be obtained from biological samples such as sputum or fluid from bronchoalveolar lavage (asbestos bodies/fibres).

c) Time interval between exposure and effect consistent with the nature of the disease progression.

- Evidence must be shown that exposure precedes the health effects.
- A past history of the disease does not automatically exclude the possibility of a workplace agent causing current episodes, e.g., work aggravated asthma.
- The following parameters, if present, may facilitate the decision on the consistency of the time interval between exposure and health effects:
  - Minimum induction period
    - The shortest period from beginning of exposure to beginning of disease below which the exposure would have been unlikely to have caused the disease.
  - Maximum latent period
    - The length of time from cessation of exposure, beyond which it is unlikely that any disease can be attributed to the exposure.

d) Consideration of differential diagnosis

- Similar clinical features can be caused by non-occupational conditions. Thus, OHDs need to consider this before diagnosing or excluding an occupational disease found during HEM.
- Additional pointers to the possibility of occupational disease include:
  - worsening of symptoms at work
  - improvement of health condition when away from the workplace
  - clusters of similar cases from the same work area.
- Both occupational factors and non-occupational factors may be synergistic in causing a disease.

5.3 ROLES OF ACUTE HEALTH EFFECTS DETERMINATION IN MS

- History of acute health effects for the specific CHTH are to be determined during MS examination.
- It indicates episodes of short-term high intensity exposures.
- It is not used as an indicator for HEM in a MS programme and it is not a sole basis for medical removal protection.

5.4 HEM BY TARGET ORGANS

A target organ is an organ in the body that is most affected by a specific CHTH, taking into consideration a single or mixed exposure to CHTH. Most of the organs and systems in the body can be a target, but the most commonly referred to as a target organ are lungs, liver, kidney, heart, blood, or circulatory system, brain or central nervous system, reproductive and skin.

Table 15 to 23 explain the examples of the signs and symptoms, monitoring parameters and criteria for referral if specific investigation is needed for the target organs mentioned.

5.4.1 Lungs *refer to Appendix for further information.

Table 15. Health Effects Monitoring of the Lungs for cases of Hypersensitivity

*LUNGS - Hypersensitivity (Occupational Asthma)		
Sign and Symptoms	**Investigation Tools**	**Referral for Specific Investigation**
Hypersensitivity/hyper-responsiveness • Cough, breathlessness, wheezing etc • Clinical and physiological changes show clear relationship with exposure to causal agent: - A sequence of symptoms in direct relation to the work schedule. - Recurrence of symptoms and signs following re-exposure to the same agent	•Spirometry: - Serial PEFR during periods at and away from work •Lung function: - obstructive pattern which is reversible •Lung function and bronchial reactivity may become normal after cessation of occupational exposure	Indication: - When the diagnosis remains in doubt - To determine the precise causative agent Investigations: - Specific inhalation challenge (bronchial provocation test). - Specific lgE antibodies, skin prick or serological testing can be used to assess sensitization.

Table 16. Health Effects Monitoring of the Lungs for cases of Fibrosis

LUNGS - Fibrosis		
Sign and Symptoms	**Investigation Tools**	**Referral for Specific Investigation**
Symptoms: Shortness of breath, cough, fatigue, weight loss, diminished exercise tolerance. Signs: Persistent bilateral late inspiratory basal crepitations, clubbing etc History of sufficient exposure e.g. •Asbestosis: - Min. duration of exposure: 5 years. - Min. induction period: 5 years (shorter in heavy exposure). •Silicosis - Min. intensity of exposure: usually above 50µg.m-3 crystalline free silica. - Min. duration of exposure: 5 years (2 years in accelerated disease).	Lung function tests: •commonly restrictive (asbestosis). •mixed restrictive/ obstructive pattern (silicosis). Chest X-ray: opacities e.g. •diffuse interstitial mainly in the lower lung fields (asbestosis). •bilateral, multiple, discreet rounded usually in the upper zones (silicosis). •Conglomerate ('complicated' silicosis).	Indication: - To confirm the clinical diagnosis. Investigations: - Computerised tomography - Biological samples for exposure investigation - asbestos bodies/fibres in sputum, fluid from bronchoalveolar lavage or lung biopsy if indicated.

5.4.2 Central Nervous System (CNS)

Table 17. Health Effects Monitoring of the Central Nervous System

CENTRAL NERVOUS SYSTEM		
Sign and Symptoms	**Investigation Tools**	**Referral for Specific Investigation**
Starts with CNS symptoms only or progress to Chronic Toxic Encephalopathy: •Type 1: Organic affective syndrome - - Clinical manifestations are depression, irritability, loss of interest in daily activities. •Type 2: Mild CTE - Fatigue, mood disturbances, memory & attentional complaints. Impairment of psychomotor function (speed, attention, dexterity), short term memory and other neuropsychological impairment. •Type 3: Severe CTE - Loss of intellectual ability of sufficient severity to interfere with social or occupational functioning.	**Q16 Checklist -** Neurotoxic Exposure to Organic Solvents (1997 Swedish). (Refer **Appendix 5** for questionnaire) •A tool to identify exposed employees requiring specialist assessment. •Scoring of >6/16: refer for further assessment of toxic encephalopathy and neurobehavioral symptoms. •Scoring of ≤6/16: use other clinical judgement.	Indication**:** •Confirmation of disease by neurologist and a neuropsychologist. •Numerous differential diagnosis (Major depression/Sleep disorders/ Neurodegenerative disorders/ Neurovascular disorders/ Neoplasms/ Metabolic causes) Investigations**:** •Neuropsychological assessment •other exclusion diagnosis *Type 1 -reversible if exposure is discontinued.* *Type 2 - reversible in some cases.* *Type 3 -cases are uncommon and poorly reversible, but nonprogressive once exposure has ceased*

5.4.3 Reproductive System

Table 18. Health Effects Monitoring of the Reproductive System

REPRODUCTIVE SYSTEM		
Sign and Symptoms	**Investigation Tools**	**Referral for Specific Investigation**
Female: •Reproductive cancer signs and symptoms •Menstrual disturbance (e.g., exposure to Dibromopropane) •History of: -Poor pregnancy outcomes: -Spontaneous abortion -low birth weight -preterm birth •Infertility •*Congenital birth defects (e.g., neural tube, cleft lip/palate).	N/A	Referral indication: Any suspected case based on clinical judgement to be referred for further investigation to rule out non-occupational causes.
Male: •Infertility •Reproductive cancer signs and symptoms	N/A	Referral indication: Any suspected case based on clinical judgement to be referred for further investigation (e.g., sperm count) to rule out non-occupational causes.

5.4.4 Skin

Table 19. Health Effects Monitoring of the Skin

SKIN		
Sign and Symptoms	**Investigation Tools**	**Referral for Specific Investigation**
Skin complaint related to work exposure: •The site of exposure: -The anatomical distribution of the skin disease is consistent with cutaneous exposure during the job task. •History: -occupational exposure to a substance known to trigger dermatoses. -The development of the skin lesions is in direct relationship to the work schedule. -There is recurrence of the disease on re-exposure to the same agent.	•Skin examination -In tandem with clinical appearance of occupational skin disease due to irritant, allergen, skin carcinogen, pigmentation and depigmentation agents, oil, tar, and its derivatives. •**Mathias Criteria** for Probable Occupational Causation of Contact Dermatitis. (Refer Appendix 6) -≥4/7 positive response - probable occupational contact dermatitis.	•Indication**:** -to determine the precise causative agent. -when the diagnosis remains in doubt. •Investigations**:** -Immunological tests: ~Skin Patch Test (Allergic CD) ~Skin Prick Test (urticaria) -Skin biopsy (neoplasm).

5.4.5 Liver

Table 20. Health Effects Monitoring of the Liver

LIVER		
Sign and Symptoms	**Investigation Tools**	**Referral for Specific Investigation**
•Symptoms and signs of liver toxicity. •Disorders of the liver ranging from reversible functional abnormalities to severe atrophy. •The pathology of occupational liver disease may include Toxicant-associated steato-hepatitis (TASH), cirrhosis, vascular disease, and neoplasm. •Follows occupational disease diagnostic criteria e.g., concept of exposures.	•Liver function test (monitors subclinical elevation in liver enzyme concentration to liver failure) - alanine transaminase (ALT) - aspartate transaminase (AST) - alkaline phosphatase (ALP) - gamma-glutamyl transferase (GGT) - serum bilirubin, prothrombin time (PT) - the international normalised ratio (INR) albumin etc.	•Referral indication: based on clinical judgement to be referred for further investigation, example: -Liver imaging -Liver biopsy

*Refer to Appendix 4 for further information.

*In the case of liver disorders, OHD are expected to investigate and rule out any other common causes.

5.4.6 Kidney

Table 21. Health Effects Monitoring of the Kidneys

KIDNEY		
Sign and Symptoms	**Investigation Tools**	**Referral for Specific Investigation**
•Severity ranging from subclinical elevation in renal enzyme concentration to renal failure. •The pathology of occupational renal disease may include: -Acute Tubular Necrosis -Glomerulonephritis -Chronic Kidney Disease, neoplasm. •Follows occupational disease diagnostic criteria e.g., concept of exposures.	•Renal function test including: -Serum urea -Creatinine -urine for protein and electrolytes glomerular filtration rate (GFR)	•Referral indication: -Any suspected case based on clinical judgement to be referred for further investigation, example: -Renal imaging -Renal biopsy

*In the case of kidney disorders, OHD are expected to investigate and rule out any other common causes.

5.4.7 Heamatology

Table 22. Health Effects Monitoring of the Haematology

HEAMATOLOGY		
Sign and Symptoms	**Investigation Tools**	**Referral for Specific Investigation**
•As a result of haematotoxicity that includes bone marrow dysplasia, cytolysis, and neoplasm. •This may be manifested by anaemia, leukopenia, thrombocytopenia, leukemia and lymphomas. •Follows occupational disease diagnostic criteria e.g., concept of exposures	•Full blood count and full blood picture	•Referral indication: -Any suspected case based on clinical judgement to be referred for further investigation, example: ~Bone marrow biopsy ~Other relevant investigations to rule out non-occupational causes

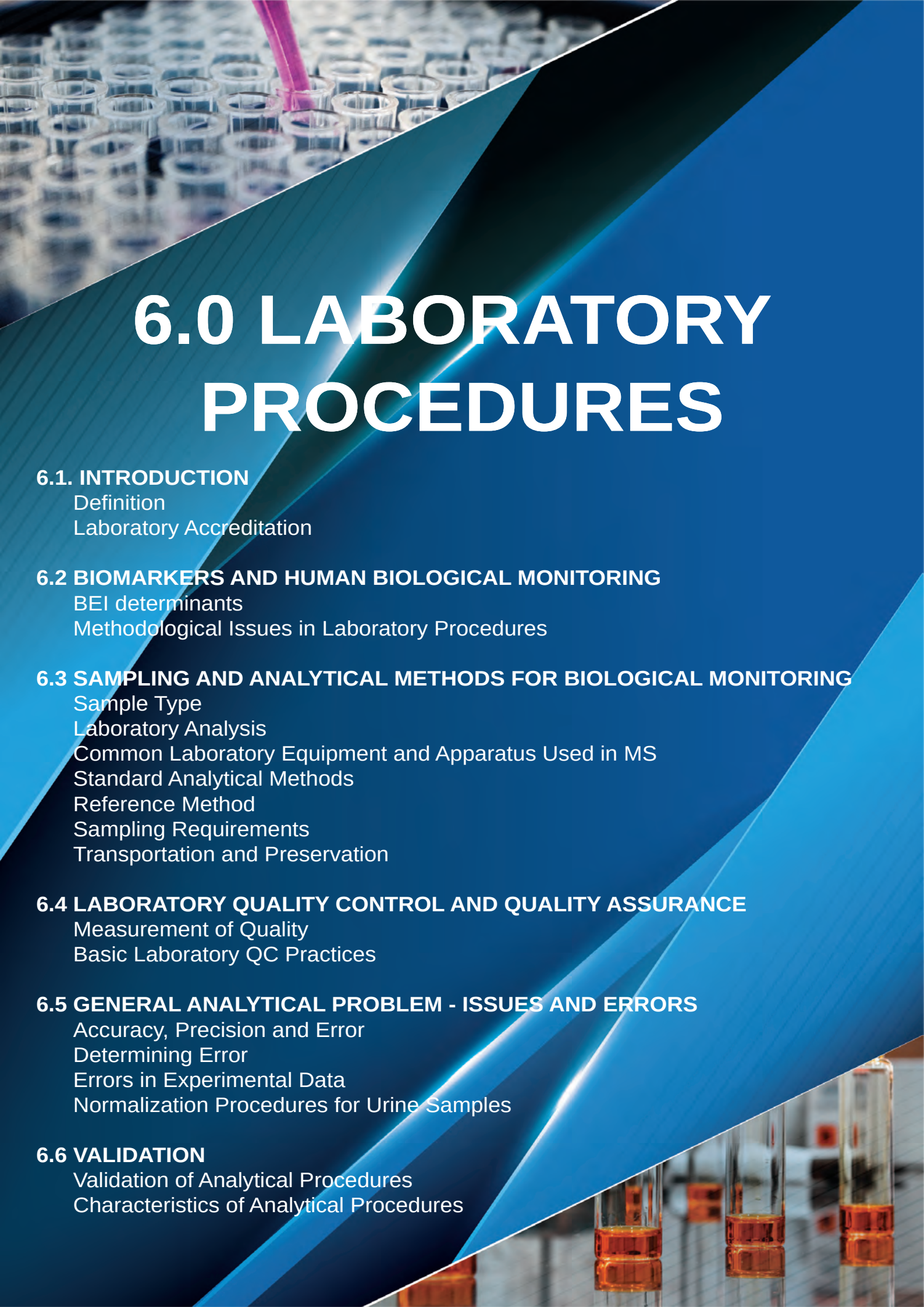
5.4.8 Cancer

Table 23. Health Effects Monitoring cases of Cancer

CANCER		
Sign and Symptoms	**Investigation Tools**	**Referral for Specific Investigation**
•Common clinical features - weight loss and dependent on the body system. •Often difficult to distinguish from those of non-occupational origin. •Some cancers are strongly associated with occupational exposures. e.g., -angiosarcoma of the liver (vinyl chloride monomer). -mesothelioma (asbestos). •May arise in a group of individuals with similar occupational exposure. •Tend to affect young age employees especially if initial exposure to the carcinogen occurs early in their working life.	•Often no unique pathological or histological features	•Referral indication: -Confirmation of clinical diagnosis •Examples of investigations: -Cytological examination of sputum, bronchial aspiration, or bronchial lavage for respiratory cancer. -Histological examination of biopsy specimen. ~e.g., mesothelioma - distinguishing tit from the chief differential diagnosis of secondary adenocarcinoma ~Computed tomography - determining extent of lesion.

***Special notes on carcinogens and respiratory sensitizers**

- Many carcinogens do not have clear dose effects thresholds.
- Respiratory sensitizers also have no clear dose effects thresholds.
- For these CHTH, the determination of the MS requires special considerations and may not necessarily be based on the EM results that exceed the 50% of PEL.
- Careful considerations are necessary on the representativeness of the EM results, anticipation of non-routine exposures and incidence of peak exposures (such as due to leakages and spills).



6.0 LABORATORY PROCEDURES

6.1. INTRODUCTION
- Definition
- Laboratory Accreditation

6.2 BIOMARKERS AND HUMAN BIOLOGICAL MONITORING
- BEI determinants
- Methodological Issues in Laboratory Procedures

6.3 SAMPLING AND ANALYTICAL METHODS FOR BIOLOGICAL MONITORING
- Sample Type
- Laboratory Analysis
- Common Laboratory Equipment and Apparatus Used in MS
- Standard Analytical Methods
- Reference Method
- Sampling Requirements
- Transportation and Preservation

6.4 LABORATORY QUALITY CONTROL AND QUALITY ASSURANCE
- Measurement of Quality
- Basic Laboratory QC Practices

6.5 GENERAL ANALYTICAL PROBLEM - ISSUES AND ERRORS
- Accuracy, Precision and Error
- Determining Error
- Errors in Experimental Data
- Normalization Procedures for Urine Samples

6.6 VALIDATION
- Validation of Analytical Procedures
- Characteristics of Analytical Procedures

6.0 LABORATORY PROCEDURES

6.1 INTRODUCTION

While the technology and instrumentation used for biological monitoring testing have a lot in common with environmental testing laboratories, biological monitoring presents a set of unique challenges. Assaying clinical matrices involves the safe handling of potentially infectious materials, different interferences, and possibly the analysis of metabolites. The greatest differences between biological monitoring and environmental testing involve the interpretation of clinical findings and effective communication of results on an individual and community basis.

6.1.1 Definition

Limit of Detection (LOD): The LOD is defined as the lowest quantity or concentration of a component that can be reliably detected by an analytical method. It is important to know the LOD of the method because this dictates the minimum sampling volume and length of sampling time. The ideal limit of detection should be lower than 1/10th of the exposure standard.

Limit of Quantification (LOQ): LOQ stands for the smallest amount or the lowest concentration of a substance that is possible to be determined by means of a given analytical procedure with the established accuracy, precision, and uncertainty.

6.1.2 Laboratory Accreditation

Laboratory accreditation is a procedure by which an authoritative body gives formal recognition of technical competence for specific tests/ measurements, based on third party assessment and following international standards.

- International Standards Organization (ISO)
- Skim Akreditasi Makmal Malaysia (SAMM)

Laboratories shall be accredited to ensure effective quality management systems, which include but not limited to:

- Qualified lab personnel
- Successful Proficiency Analytical Testing
- Quality control programmes

Specimens relevant to the MS shall be sent to a laboratory that is accredited by the Department of Standards Malaysia or any international accreditation body recognized by the Department of Standards Malaysia. It is recommended to consult Standards Malaysia for the list of accredited laboratories.

6.2 BIOMARKERS AND HUMAN BIOLOGICAL MONITORING

6.2.1 BEI determinants

The sample types of biomarkers are listed below:

A) Blood
B) Urine
C) Breast milk
D) Expelled air
E) Hair
F) Nails
G) Saliva
H) Teeth
I) Meconium
J) Amniotic fluid
K) Adipose tissue
L) Other tissues and fluid

6.2.2 Methodological Issues in Laboratory Procedures

In general, biological monitoring involves measurement of very low levels (e.g., parts per billion), often near the limit of detection. The common methodological issues are summarised below:

1) Analytical technique.

2) Environmental contaminants and controls.

3) Laboratory contamination and quality assurance.

4) Correct choice of biomarker for study design and question.

5) Rationale for selecting environmental chemicals of interest.

6) Coordination with related research - epidemiology, toxicology, pharmacokinetic modelling, exposure assessment.

External contamination is a critical issue, and sources of external contamination deserve special discussion. For example, materials to draw blood or specimen containers may also contain the analyte of interest. Materials used in the lab (paper towels, hand soaps, etc.) may contain the chemical of interest and result in contamination; pesticides sprayed outside the laboratory building may be tracked into the labs, volatilize, and result in contamination; exposing serum to air by repeatedly opening the vial may introduce PBDE-containing dust and falsely elevate results. These are a few examples that have caused measurement inaccuracies. For a laboratory doing biological monitoring measurements, it is imperative to consider such sources of external contamination whereas this is not a consideration (or not to the same extent) for other clinical labs. Pre-screening of materials may be necessary.

6.3 SAMPLING AND ANALYTICAL METHODS FOR BIOLOGICAL MONITORING

The overall reliability of analytical testing depends on a quality management system that ensures proper design and implementation of a series of steps including biological specimen collection, sample pre-treatment, extraction, clean-up, concentration, and instrumental measurement. Analytical method validation confirms that the method is suitable to detect, identify and measure (both accurately and precisely) the target compounds, thus verifying and quantifying method performance. While analytical method validation or verification is a concept applied in all areas of chemical measurements, biological monitoring applications present many unique challenges highlighted in the following sections.

6.3.1 Sample Type

Generally, biomarkers can be used as indicators of hazard, exposure, disease and to determine population at risk. In MS programmes, biomarkers allow for early indication of a disease which help in identifying correct intervention methods to prevent the progress of disease.
Sample type is described as all other elements apart from the analyte of interest. Understanding the sample type and its effect on analysis is crucial when deciding an analytical method. The biomarker of interest, for instance 2,5-hexanedione in BM of n-hexane exposure, is found only in urine samples.

A few common sample types used for MS include:

- Urine
- Blood - whole, plasma or serum
- Hair
- Nail
- Saliva

6.3.2 Laboratory Analysis

Biological samples analysed in the laboratory include but not limited to:

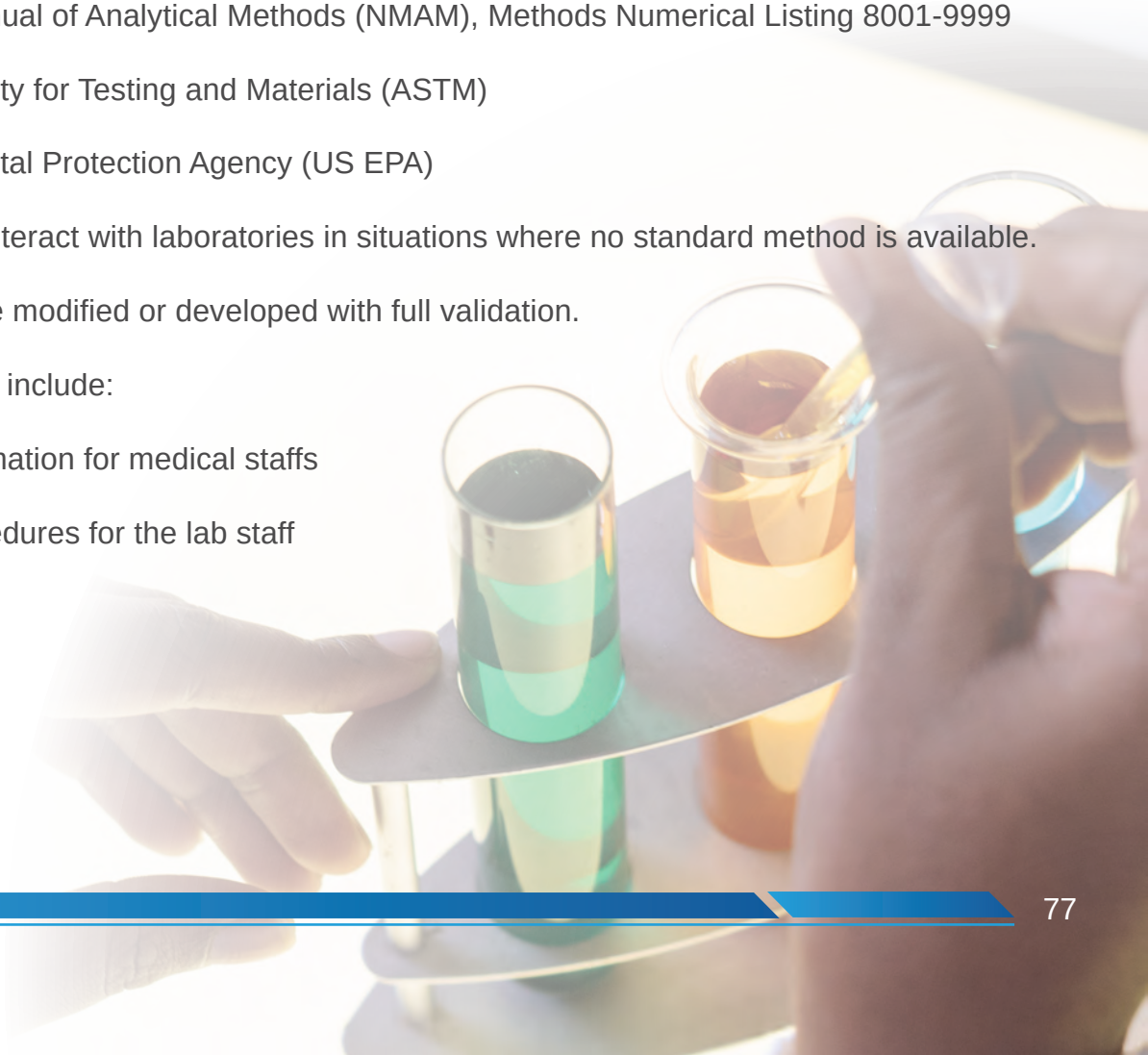
- Metals and other elements
- Gases
- Organics

6.3.3 Common Laboratory Equipment and Apparatus Used in MS

- •Colorimetry
  - ~Titration, UV-vis, ELISA
- •Spectroscopy
  - ~Atomic Absorption Spectroscopy, Atomic Fluorescence Spectroscopy, Inductively Coupled Plasma
- •Chromatography
  - ~LC, GC

6.3.4 Standard Analytical Methods

- •Organizations have developed analytical methods. These methods are evaluated/validated for accuracy.
- •Sources of analytical methods include but not limited to:
  - ~US Occupational Safety and Health Administration (OSHA)
  - ~US NIOSH Manual of Analytical Methods (NMAM), Methods Numerical Listing 8001-9999
  - ~American Society for Testing and Materials (ASTM)
  - ~US Environmental Protection Agency (US EPA)
- •The OHD should interact with laboratories in situations where no standard method is available.
  - ~Methods can be modified or developed with full validation.
- •Analytical methods include:
  - ~Sampling information for medical staffs
  - ~Analytical procedures for the lab staff
  - ~Quality control



6.3.5 Reference Method

The reference method contains information on:

- •Properties of the analyte and threshold limits
- •Sampling
- •Sample preparation for analysis
- •Calibration and Quality control
- •Measurement
- •Applicability
- •Interferences

6.3.6 Sampling Requirements

Once investigators have ensured that the proposed specimen type appropriately reflects the body burden of the biomarker there are a number of considerations in biological specimen collection: containers and tubes, specimen identification and documentation, collection method, shipping, storage, and banking. Specimen collection protocols will provide detailed step-by-step instructions and describe how, where and when specimens are to be collected and transported to the lab. Laboratory personnel should work with the epidemiologist in writing procedures for field staff and instructions for participants on specimen collection. In some situations, laboratory personnel may train field staff in specialized specimen collection procedures to ensure the integrity of the sample for testing.

- •Sample collection for MS will often utilize the following containers:
  - ~Urine container
  - ~Blood container
- •Adhering to sampling requirement is important in biological monitoring. Certain requirements specifically preservation, sample stability and transportation must be followed to ensure sample integrity.

6.3.7 Transportation and Preservation

Potential contamination of the containers can introduce a bias in laboratory measurement. The suitability of a given lot of collection tubes must be assured by the laboratory prior to the collection of specimens. This is particularly important when the laboratory is measuring a chemical or metabolite common in the environment or that could potentially leach from specimen containers.

For traces of heavy metals, such as lead or mercury, some containers are commercially available from lab supply distributors used by clinical/medical laboratories. Some specimen containers are marketed specifically for trace metal analysis and might come with a certificate indicating that they are relatively “metals free.” If a certificate is not available, or if the levels indicated are higher than those measured in the study, additional quality control steps must be taken.

With the exception of trace elements, however, the analytes of interest for clinical measurements are not usually the same ones that are of interest for biological monitoring studies. Therefore, the non-routine uses of blood or urine containers require an additional level of quality control.

Laboratory-performed quality control of lots of containers to be used for specimen acquisition will be required for the majority of methods. The screening procedure must assure that when specimens are collected and stored following laboratory protocol, the contamination introduced by the containers themselves or any preservatives is negligible (i.e., below the detection limit of the analytical method). Pre-cleaning (acid-washing, solvent rinsing) of collection materials may be indicated for some analyses.

Proper collection of specimens to be tested for environmental chemicals serves an important first step in assuring that the final results of laboratory testing are representative of concentrations actually present. Use of specimen collection instructions, field blanks and duplicate samples is recommended to standardize the collection procedure and estimate potential bias that may begin at the specimen collection stage.

Specimen collection instructions detailing collection procedures for specimens for use in a particular study are essential. The instructions should be written, with input from laboratorians and from healthcare providers (phlebotomists, nurses, or physicians) involved in the collection. Step-by-step directions should be specific on where the sample containers are obtained, how they are labelled, what information should be collected prior to the collection, what steps need to be taken to prevent contamination, and where the collected specimen is to be routed for storage or transportation.

Instructions geared towards employees should be written simply and without jargon and should include a phone number where clarifications can be obtained. In some situations, it may be advisable for laboratory staff to train those collecting and transporting specimens to minimize the likelihood of exogenous contamination and to ensure the viability of specimens during transport.

Packaging and shipping of hazardous materials is highly regulated in most countries including Malaysia. Following hazardous material regulations is the responsibility of the shipper. However, for many biological monitoring applications, the laboratory will supply sampling kits and packaging materials. These supplies and their use for a particular mode of transport must be in compliance with regulations listed at the end of this section. In addition, laboratory personnel may have to refer some tests to other facilities and, therefore, must be in compliance with all applicable shipping regulations, including training of personnel required by these facilities. Laboratories that maintain contracted courier services should also be aware of packaging requirements for this type of transport.

Laboratory protocols for storage and handling of specimens should be part of the overall study protocol documents. Unless specimens are being stored for future use, procedures for when and how specimens will be destroyed at the end of the study are needed. Once received at the laboratory, specimens must be stored properly to avoid target analyte and matrix deterioration. While room temperature storage may be appropriate for some sample types (e.g., hair or nail clippings), refrigerated or frozen storage is commonly employed for the majority of common clinical sample types such as urine, whole blood, serum, etc. Storage at temperature below freezing (-20˚C or -70˚C) is generally recommended for long-term storage and for temperature-sensitive analytes such as those to be speciated by oxidation state.

While these storage guidelines are widely followed, it should be noted that there is limited information on the stability of many analytes that may be chosen for biological monitoring studies. The effect of temperature variations during transport, prolonged storage or thawing on the concentration of the majority of analytes has not been studied. These effects remain particularly concerning when plans include specimen storage (i.e., long-term storage for future studies). Analyte stability studies would have to be undertaken to assess suitability of a chosen analyte and storage method for such applications.

In summary, basic handling requirements:

1. Clear labelling, collection day/time is important.
2. Sealed and secured.
3. Do not expose to sunlight; and
4. Storage, packaging, and transportation for samples shall be according to the sampling methodology.

The laboratory shall advise the samples collection containers as described below:

1. Urine - no preservative. Thymol is used in some cases but rarely.
2. Blood - tube comes with preservative e.g., EDTA, heparin, sodium citrate, refer to the blood tube guidelines.

6.4 LABORATORY QUALITY CONTROL AND QUALITY ASSURANCE

Every biological monitoring study must have a quality management system, which ensures the integrity of the samples, the analyses and the data produced. For the analytical portion of the investigation, this includes written Standard Operating Procedures (SOPs) for specimen collection, handling and transport, sample processing, sample analysis, and quality control. Laboratory methods must be internally validated including determination of method accuracy and precision. Chemists must be thoroughly trained in all aspects of the procedures prior to sample analysis. Minimally, each study should have quality control specimens that are analysed concurrently with study samples as well as external assessment of laboratory proficiency.

Quality Assurance (QA): All quality requirements and activities implemented within the quality system of that can be demonstrated to internal management and external parties (customers, government agencies, regulators, and others) to ensure quality requirements can be fulfilled.

Quality Control (QC): The techniques and activities adopted to fulfil quality requirements which involve inspection of quality management.

- Both QC and QA are essential parts of any determination, no matter which matrix is being analysed or which chemical or hazard is being measured.

- Every laboratory, whether it is analysing air or water or dirt or blood, must have a formal written QA/QC programme in place to ensure the validity of the data.

- Although QA/QC falls under the primary responsibility of the laboratory, industrial hygienists, occupational physicians, nurses, and others who rely on contract laboratories for biological monitoring determinations should be aware of basic QA/QC and should use basic QA/QC in their sampling protocols.

- Laboratory SOPs provide the formal documentation of how the laboratory functions including the following: 6.4.1 and 6.4.2.

6.4.1 Measurement of Quality

The major analytical concepts that drive analytical quality management are accuracy, trueness, precision, and traceability. Those concepts can be related to measures of performance, such as bias, standard deviation (SD) or coefficient variation (CV), and measurement uncertainty (MU). To utilize those measures for managing analytical quality, specifications must be defined using goal-setting models to establish limits for the amounts of errors that are allowable.

Those error goals, called analytical performance goals or analytical quality specifications, are used to validate the performance of examination procedures via experimental studies and statistical data analysis, assess performance relative to the desired quality using sigma-metrics, and plan Standard Quality Control (SQC) procedures to ensure attainment of the desired quality in routine production, taking into account the precision and bias observed for the measurement procedure and the rejection characteristics of different control rules and different numbers of control measurements. Operating specifications for precision, bias, and SQC describe the characteristics needed at the bench level to ensure that the desired quality is achieved during routine operation. Finally, quality must be monitored long term to characterize performance, identify problems, and prioritize improvements.

To better understand the different measures and models, Table 24 provides the definitions of critical performance characteristics.

Table 24. Definitions of Important Performance Characteristics

Accuracy	closeness of agreement between a test result and the accepted reference value (ISO 5725-1). Note: The term ‘accuracy’ when applied to a set of test results, involves a combination of random components (imprecision) and a common systematic error or bias component (ISO 5725-1).
Precision (of measurement)	closeness of agreement between indications or measured quantity values obtained by replicate measurements on the same or similar objects under specified conditions (JCGM 200:2012). Note: Measurement precision is usually expressed numerically by measures of imprecision, such as standard deviation, variance, or coefficient of variation under the specified conditions of measurement (JCGM 200:2012).
Trueness (of measurement)	closeness of agreement between the average of an infinite number of replicates measured quantity values and a reference quantity value (JCGM 200:2012). Note: Trueness is expressed numerically using the observed bias.
Bias (of measurement)	difference between the expectation of the test result or measurement results and a true value (ISO 3534-2). Note: Bias is an estimate of the systematic measurement error (JCGM 200:2012).
Traceability (of metrological)	property of a measurement results where the result can be related to a reference through a documented unbroken chain of calibrations, each contributing to the measurement uncertainty (JCGM 200:2012).
Uncertainty (of measurement)	parameter, associated with the result of a measurement, that characterizes the dispersion of the values that could reasonably be attributed to the measurand.
Total error	includes all random and systematic errors that can occur during the total testing process and also includes the combined effect of all precision and bias errors that can affect the accuracy of an analytical result. Note: Total error incorporates error sources from the pre-analytical, analytical, and post-analytical phases of a measurement procedure.

Enrolment in an external proficiency testing programme is the preferred way of confirming the accuracy of laboratory measurement as well as pre-and post-analytical laboratory procedures. External proficiency testing enables a laboratory to establish its accuracy relative to other laboratories measuring the same chemical. Semi-annual testing is sufficient to establish the accuracy of the lab and the entire analytical system. There are many proficiencies testing programmes for inorganic analysis and organic analysis available commercially. Since the field of biological monitoring is not regulated and ever-expanding, proficiency testing programmes do not typically exist for all the possible chemicals. When a programme does not exist, it is advisable for the laboratory to seek out other laboratories doing the same measurement, in order to exchange quality control materials and compare the results. If enough labs are doing the measurement, a round-robin style of testing can be established where each lab measures the same QC material, and the results are compared.

Within the biological monitoring laboratory, a proficiency testing programme for analysts can be established by characterizing some QC material at several concentration levels. Many aliquots of the QC material are then produced and "blinded" by having one person, not associated with the analysis, randomize the vials, and assign their own identification to them. Semi-annually, this person presents vials for analysis to each analyst and then compares the results to the characterized results. This technique mitigates the possibility of an analyst knowingly or unknowingly altering results because the theoretical value is known.

6.4.2 Basic Laboratory QC Practices

It is critical for laboratories to have proper bench (daily) quality control (QC) materials for their analytical systems (e.g., instruments) when analysing samples, particularly for biological monitoring. Otherwise, the analytical processes or instruments could potentially show an erroneous increase or decrease in concentrations due to long term drift of the instrument or the analytical process. QC materials must be well characterized and stable for years, so that long-term QC can be tracked.

Even a small analytical drift can result in an erroneous public health conclusion or decision because the difference over time for a group of people (group mean level) can be small, but it may be statistically significant. Quality control and proper laboratory techniques prevent erroneous conclusions, ensuring that the biological monitoring study results are valid and are scientifically defendable.

Quality control samples comprise part of each analytical run and demonstrate the accuracy of the method during each sample run. Quality control samples are typically the same matrix as the study specimens and have concentrations of target compounds in the low, medium and/or high range of the assay calibration. If available, Certified Reference Materials (CRM) or Standard Reference Materials (SRM) should be used to prepare QC samples. Unfortunately, CRM does not exist for many biological monitoring analytes of interest, and when it does exist, it tends to be expensive. If a commercially available source cannot be located for the chemicals of interest, quality control materials can be prepared in the laboratory using blank matrix.

Blank QC material can be fortified with the appropriate level of the chemicals of interest. Optimally, the solutions used to fortify the QC material should be from a different source than the solutions used to generate calibration standards. If a second commercial source cannot be found, it would be best to produce a second set of stock solutions from neat material. Due to the possibility of endogenous species, the QC material should be characterized after it has been fortified. QC characterization is accomplished with a minimum of 20 analytical runs to produce an average target concentration and a standard deviation from which to derive the limits. The laboratory will have to establish the system by which the QC results are evaluated and accepted or rejected.

6.5 GENERAL ANALYTICAL PROBLEM - ISSUES AND ERRORS

Standard operating procedures (SOPs) serve as essential laboratory documents detailing a complete technique for analytical performance. SOPs are important to ensure operations are carried out correctly, consistently and in a reproducible manner. When used properly, SOPs can improve and maintain quality analytical methods, standardize laboratory performance, and assure quality results. Reliable chemical standards used to prepare calibration and control solutions remain essential to any analytical method. For biological monitoring applications, analytical standards will likely represent a significant cost of the total analysis.

6.5.1 Accuracy, Precision and Error

The definitions for accuracy and precision are compared as seen in Table 25.

Table 25. Comparison between Accuracy and Precision

Accuracy	Precision
● Measure of how close a measurement comes to the actual or true value of whatever is measured. ● Relates to **reproducibility** of results (how similar are values obtained in the same way). ● Bias is also associated with accuracy: ○ Bias is described as the difference between average of measurements and an agreed upon standard value. ○ Cannot be evaluated without a Standard. ○ Add a consistent 'bias factor' to all measurements. ○ Affects all measurements in the same way. ○ Causes of bias include: ~ Error in Laboratory Information System ~ Worn components ~ Instrument improperly calibrated ~ Instrument damaged ~ Instrument improperly used ~ Instrument read incorrectly ~ Part set incorrectly	● Measure of how close a series of measurements are to one another, irrespective of the actual value. ● Relates to **repeatability** of results: ○ Repeatability is described as the variation among repeated measurements. ○ A standard is not required. ○ May add or subtract from a given measurement. ○ Affects each measurement randomly. ○ Issues with repeatability include: ~Measurement Steps >Sample preparation >Setting up the instrument >Locating on the part

6.5.2 Determining Error

"Error" in laboratory analysis is defined as the difference between the true result (or accepted true result) and the measured result. If the error in the analysis is large, serious consequences may result. As reliability, reproducibility, and accuracy are the basis of analytical chemistry;

- Accepted value is the correct value for the measurement based on reliable references.
- Experimental value is the value measured in the lab.
- The difference between the experimental value and the accepted value is called the error.

Error = experimental value - accepted value

The percent error of a measurement is the absolute value of the measured experimental value minus the accepted value divided by the accepted value, multiplied by 100%.

$$\text{Percent error} = \frac{\text{(measured value - accepted value)}}{\text{accepted value}} \times 100\%$$

6.5.3 Errors in Experimental Data

Experimental error is the difference between a measured value and its true value. In other words, it is the inaccuracy or inaccuracies that stop us from seeing an absolutely correct measurement. There are three types of Errors in Experimental Data:

1) Random (indeterminate) Error
- Data scattered approximately symmetrically about a mean value.
- Affects precision - dealt with statistically.

2) Systematic (determinate) Error
- Readings are all too high or too low. Affects accuracy.
- Several possible sources:

i. Instrument Error

a) Need frequent calibration – both for apparatus such as volumetric flasks, burettes etc., but also for electronic devices such as spectrometers.

b) May be minimised by careful recalibration and good maintenance of equipment.

ii. Method Error

a) Due to inadequacies in physical or chemical behaviour of reagents or reactions (e.g., slow, or incomplete reactions)

b) Most difficult error. "True" value may not be known.

c) Three approaches to minimise: analysis of certified standards, use 2 or more independent methods, analysis of blanks.

iii. Personal Error

a) E.g., insensitivity to colour changes; tendency to estimate scale readings to improve precision; preconceived idea of “true” value.

b) Can be constant (e.g., error in burette reading - less important for larger values of reading) or proportional (e.g., presence of given proportion of interfering impurity in sample, equally significant for all values of measurement)

c) Can be minimised by care and self-discipline.

3) Gross Errors

- Usually obvious - give “outlier” readings.
- Detectable by carrying out sufficient replicate measurements.

6.5.4 Normalization Procedures for Urine Samples

- The disadvantage of a spot urine as a specimen is the variation in the concentration of its constituents due to variable worker fluid intake and sweating. This has necessitated the use of normalization procedures.

- The most common approach to normalization is a creatinine correction. Creatinine is a normal constituent of urine that is excreted at a constant rate in people of about constant muscle mass (“lean weight”). The concentration of the marker is divided by the concentration of creatinine in the same sample:

- Common units are milligrams of marker per gram of creatinine; micrograms of creatinine per milligram of creatinine; and milligrams of marker per millimole creatinine.

- There are limits to the use of this correction. It cannot be used if body weight is not relatively constant, or if the worker has kidney damage, for example, excess protein in the urine (proteinuria).

- The correction should be used when creatinine concentration is within the range of 0.3 to 3 g/L, but not outside this range.

- For instance, in the case of diluted urine of 0.2 g/L, the uncorrected marker concentration will be multiplied by a factor of 5, often resulting in a falsely elevated result.

- It is advisable to use a creatinine dipstick just after sample collection at the sampling site to determine if the fresh urine sample is valid or if another sample needs to be collected on another exposure day.

- There is no point to an expensive marker analysis when the sample is invalid.

- Another normalization procedure is by specific gravity, the density of the urine relative to that of water at the same temperature.

- It is advisable to check the specific gravity at the site and time of collection with a dipstick, just as for the creatinine concentration above.

- If the urine specific gravity is greater than 1.015, the creatinine concentration is usually greater than 0.5 g/L.

- This procedure also avoids inconsistent results and unnecessary expense from invalid urine samples.

- The specific gravity correction is not applicable below a value of 1.010. The ACGIH reference specific gravity is > 1.010 and < 1.030, and:

  ~Corrected specific gravity = (observed value × 24) / last two digits of the observed specific gravity

- Samples that are not frozen should have their creatinine or specific gravity checked by the analytical laboratory to ensure sample integrity during storage, transport, and analytical laboratory reception.

- If urine samples need to be frozen, ensure that the bottom part of the sample is cooled first to prevent container breakage, especially for glass and low-density polyethylene containers.

- Creatinine concentration or specific gravity do change after freezing relative to fresh urine. Because labels tend to fall off at or below freezing, the label should be taped securely in a way that does not obscure the label.

6.6 VALIDATION

Method validation remains necessary to confirm that the analytic method is suitable to detect, identify and reliably measure the target compounds in the designated matrix. Method validation is a set of experiments which demonstrate the accuracy, precision, selectivity, and sensitivity of the method. These experiments typically verify specific performance characteristics in order to produce a publication for the method or when a laboratory is adapting a method from another laboratory.

6.6.1 Validation of Analytical Procedures

Validation of physical and chemical analytical methods involves a well-characterized procedure, according to an established SOP, using properly qualified and calibrated instruments. The test should be run a sufficient number of times, using a clearly defined and acceptable reference standard and, if necessary, using different analysts, so that a proper statistical analysis can be performed to determine such things as accuracy and precision, as defined for each type of test. For quantitative measurements, statistics should be able to determine the linearity of the observed response, or to indicate the optimum manipulation to achieve linearity, e.g., log/log plotting. Equally important is the determination of the parallelism between the standard curve and those created by test samples.

6.6.2 Characteristics of Analytical Procedures

Accuracy and precision of each method varies depending on the sample type, the analytical technique, and the analytical standards. Sufficient experiments on sample type-based samples allow determination of intra- and inter-day precision and accuracy. This may also be accomplished with the QC characterization using the 20 analytical runs minimum.

As general guidance, method accuracy within 20% of the theoretical value would be considered sufficient for most applications. Precision at a given concentration level should not exceed 15% of the coefficient of variation. Both the accuracy and precision become worse as the concentrations approach the LOD.

Selectivity of the method establishes that the correct component in a chromatogram is being measured as the chemical of interest. Selectivity is established using all components below:

1) the retention time of the chemical versus the analytical standard.
2) the specific mass being monitored in mass spectrometry.
3) the ion abundance ratio of the chemical by monitoring at least two ions per chemical. If mass spectrometry is not used, or if a satisfactory second ion cannot be established, the retention time of the chemical on two different chromatographic phases can establish selectivity.

Determination of selectivity should be done with control material and actual field samples to properly determine if all interferences have been identified.

Sensitivity of the method is the determination of the LOD. There are many ways to determine a method's LOD. When the method LOD has been determined, it is advisable for the calibration range to include standards at and below this LOD. Typically, only results within the calibration range can be reported by the laboratory, by establishing the calibration range below the LOD, the majority of identified results can then be reported.

Reportable range is also a component of method validation and is generally determined by the analysis of field samples. The calibration range should attempt to include the concentrations expected to be found in an unexposed population as well as the concentrations found in exposed populations. Many times, this range will be too great for the analytical technique, and it may be necessary to analyse a diluted sample when a high result is found.

Stability of the chemical in and out of the sample type will need to be determined, as samples can be collected over a long period and high-priced analytical standards will be used as long as possible. There are several types of stability that need to be addressed: freeze-thaw stability, short-term stability at room temperature, long-term stability in storage conditions and stability of the chemical in solvent as opposed to sample type.

With the analytical chemistry industry constantly improving techniques, the laboratory needs to have a mechanism to update and improve their methods. If significant changes are made to a method, it may be necessary to run a new set of validation experiments to determine the new accuracy, precision, and sensitivity. If the new technique does not significantly impact these parameters, the modified method can be compared to the older method by split-sample analysis. With this technique, a minimum of 40 samples, and preferably 50 samples, are analysed by each method.

7.0 GENERAL MEDICAL SURVEILLANCE PROCEDURES

7.1 MEDICAL EXAMINATION

7.2 TYPES OF MEDICAL EXAMINATION

- Pre-placement Medical Examination
- Periodic Medical Examination
- Return to-Work (RTW) Medical Examination
- Exit Medical Examination

7.3 CONDUCTING MEDICAL EXAMINATION

- Contents of Medical Examination
- Medical History
- Relevant Family History
- Occupational History
- Other History
- History of Training
- History of Health Effects due to CHTH Exposures
- Current Health Effects due to CHTH Exposure
- Physical Examination
- Target Organ Functions Tests
- Assessment of Fitness to Wear Respirator
- Fitness to Work Certifications

7.4 MEDICAL REMOVAL PROTECTION (MRP)

- Pregnancy and Breastfeeding
- Duration of Temporary MRP
- Permanent Removal
- Return to-Work (RTW)
- Process Flow of MRP and RTW

7.5 FREQUENCY OF MS PROGRAMME

7.6 CESSATION OF THE MS PROGRAMME

7.0 GENERAL MEDICAL SURVEILLANCE PROCEDURES

7.1 MEDICAL EXAMINATION

Medical Surveillance (MS) programmes consist of medical examination (assessment of health effects) and Biological Monitoring (BM) or Biological Effect Monitoring (BEM) (assessment of exposure). If a BM or BEM is not available for a CHTH, then the MS will only consist of medical examination for the Health Effects Monitoring (HEM).

Medical examination is a clinical examination performed by OHD on an employee who is required to participate in a MS programme. The medical examination consists of history taking, physical
examination and other relevant tests to monitor the target organ functions.

7.2 TYPES OF MEDICAL EXAMINATION

7.2.1 Pre-placement Medical Examination

Pre-placement medical examination is the medical examination conducted on an employee before commencement of work in:

- a new work unit;
- a new workplace; or
- when he is required to undergo another MS programme for a newly introduced CHTH in the same current work unit.

Careful history of previous exposures to CHTHs should be documented, because the pre-placement medical examination may be considered as the baseline for the MS programme.

7.2.2 Periodic Medical Examination

Periodic medical examination means a medical examination conducted on an employee at a regular interval, as long as the employee remains in the same work unit and exposed to the same CHTH. The periodic medical examination should be conducted at least once in 12 months, or earlier, as recommended by the OHD.

7.2.3 Return to-Work (RTW) Medical Examination

This type of medical examination is conducted before the end date of the Medical Removal Protection (MRP).

It can also be done when the medical examination results are inconclusive, but the employee is allowed to continue his work.

The date of the RTW medical examination should be noted in the USECHH Form 1 and USECHH Form 5. This means that the USECHH Form 1 should be concluded whenever all relevant results are

7.2.4 Exit Medical Examination

The medical examination is conducted whenever an employee stops working at the work unit at risk and not exposed to the CHTH anymore.

This medical examination aims to conclude whether the employee is affected by the CHTH exposure at the end of his job in the work unit. The medical examination should be conducted preferably within 6 months prior to exit from the MS programme.

By comparing the result of the employees upon exit with the result when the employee started working, valuable information on the health status can be obtained. Any changes in the laboratory test result for example, should warrant further investigation to be conducted if the OHD is of the opinion that the changes are due to CHTH exposure. Exit medical examination might not be required if the MS has been performed within the stipulated period to the exit.

7.3 CONDUCTING MEDICAL EXAMINATION

The content of the medical examination is presented in the USECHH 1 (Appendix 7) form and explained in the following subtopics.

7.3.1 Contents of Medical Examination

The contents of the medical examination are listed below:

1) Compilation of information on the personal details, medical history, social history, family history, and occupational history.

2) History of employee's exposure to CHTH, Personal Protective Equipment (PPE) used and high exposure incidents.

3) History of health effects due to CHTH exposure.

4) Clinical assessment which consists of extracting information about current health effects due to the CHTH exposure and conducting physical examination.

5) History of training related to CHTHs and PPE awareness.

6) Conduct target organ function tests such as laboratory investigation, chest x-ray, spirometry and etc. as necessary.

7) Assessment of fitness to wear respirator, where applicable.

7.3.2 Medical History

Current and past medical conditions may impact the fitness to work with a CHTH. For example, chronic liver or kidney disease may affect the metabolism of a CHTH which may result in an increase in the internal dose of the CHTH and may worsen the health conditions of the employee when continuing exposure to the CHTH.

7.3.3 Relevant Family History

Family history is a risk factor for developing diseases such as cancer, autoimmune and cardiovascular disease. For example, a strong family history of autoimmune disease can increase the risk of being diagnosed with the condition when exposed to heavy metal like mercury.

The family history may provide relevant information in the determination of work-related medical conditions due to the CHTH exposure.

7.3.4 Occupational History

Previous CHTH exposures during past and present occupation, inclusive of part time employment may contribute to the current health effects experienced by the employee. Where the employee works before, and the duration of exposures provide valuable information that may explain his current health conditions.

History of exposure of incidents such as spills and splashes at the previous workplace or the current one, provide a complete picture of the previous CHTH exposure.

7.3.5 Other History

Other relevant history may include other important information that was not captured in the above sections such as drug history and dietary intake.

7.3.6 History of Training

Lack or ineffective training in safe chemical handling, recognizing chemical poisoning and proper PPE usage may contribute to the health effects experienced by the employee. Recommendations for training or retraining should be included in the MS report to the company.

Information about the type of PPE worn and problems related to the wear of the PPE by the employee is very important. Problems that resulted in improper wear and fit, may result in excessive exposure to the CHTH and end up with poisoning.

The OHD, should focus on the history of the wear and fit, and look out for issues listed below:

1) History of not wearing the PPE during the tasks that were exposed to the CHTH and the reasons behind it.

2) History of having health effects and smell of the CHTHs that indicate poor seal or inappropriate PPE.

The recommendation section of the MS report should contain actions recommended based on the findings.

7.3.7 History of Health Effects due to CHTH Exposures

History of health effects experienced during and after the handling of CHTH must be obtained. Symptoms of acute health effects experienced by the employee due to CHTH exposure is a sign of overexposure conditions. This history is also contained in the Form A of the CHRA report. However, the sampling size is usually small. MS programme will involve all employees in a work unit, so that all health effects experienced by all the employees can be recorded.

Symptoms of acute health effects due to CHTH are initially manifested through 3 target organs. The target organs are respiratory, skin, and central nervous system.

a) **Health effects on the Respiratory system**

Throat irritation, breathing discomfort, cough, sneeze, and wheeze are the usual signs of the upper respiratory system, which indicates possible overexposure conditions. These conditions usually resolve when the exposure stops. These irritation effects over the long term may cause chronic inflammation and may produce chronic health effects.

Smell of CHTH does not indicate exposures exceeding the PEL. However, for CHTHs with odour threshold which are almost similar, or exceed their PEL, the smell is a possible indication of excessive exposures. Examples are formaldehyde, 1,3 Butadiene and Vinyl Chloride monomer. CHTH such as Xylene, where the odour threshold is far lower than its PEL, the smell is an indicator of exposure but does not necessarily mean excessive exposure.

b) Health effects on the Central Nervous System

Effects on the central nervous system usually are due to central nervous system depression. The symptoms are nausea, vomiting, headache, and giddiness.
Higher exposures of some CHTH may cause higher absorption and cause central nervous system effects. The OHD should not miss these symptoms as they signalled exposures far higher than the PEL.

c) Health effects on the Skin

Skin conditions may indicate exposures. Rash and itchiness are usually the initial complaints. It is a sign of uncontrolled exposure to CHTH capable of skin absorption.

d) Health effects on other target organs

Other relevant exposure history should be gathered and recorded in this section. Other target organs such as the liver, kidney, blood system do not exhibit symptoms and signs of overexposure unless an acute poisoning is taking place.

7.3.8 Current Health Effects due to CHTH Exposure

The OHD should refer to the possible health effects of the CHTH concern in the Specific Guidelines on MS Programme. Systematic history taking is essential as the employee may not report his health conditions in detail.

The health effects need to correlate with the work conditions so that details of uncontrolled conditions can also be elicited. Other health conditions not related to CHTHs exposure should also be included as part of a medical examination.

Positive findings that are CHTH related could mean poisoning due to the CHTH. Confirmed work-related diagnosis or otherwise should be medically removed until the employee's health returns to normal and the OHD is of opinion that he is fit to work with the CHTH again. In the MS report, the OHD should recommend a review of the CHRA, and the company should rectify any lack of control measures. The case should also be reported under NADOPOD 2004.

7.3.9 Physical Examination

Pathognomonic signs of CHTH exposure are rare which present a challenge for the OHD to detect early physical signs related to the CHTHs. OHD should already know the physical signs to look out for, before performing the medical examination. The information can be found in the Specific Guidelines on MS Programme or through OHD's own research.

Physical signs associated with CHTH exposure could be localised or systemic. Local irritation effects to the eyes, upper airways, and the skin, is often a sign of overexposure conditions. Some signs might already normalise, so that it is not captured during the medical examinations. Negative findings do not necessarily exclude significant acute CHTH effects.

Signs of chronic irritation of the upper respiratory tract is possible by looking at the soft tissue of the nose, and throat. Skin examination may reveal signs of irritation and effects to the skin.

Nervous system must be examined properly, as some signs may not be obvious, without a systematic examination. Q16 Swedish questionnaire may be used to suspect central nervous system effect.

Systemic effects or the target organs effects manifest as impairments of functions. CHTH health effects on the skin are usually visible and can be described well. Other signs of medical conditions need to be documented also. Positive signs that are diagnosed as work related should be handled similar to suspected or confirmed poisoning cases.

7.3.10 Target Organ Functions Tests

Some of the examples are:

- Liver function test
- Full blood picture
- Chest X-ray
- Renal function test
- Spirometry

The target organ functions tests are monitored over time to detect worsening of health conditions. Appendix 7: MS Recommended Forms: USECHH 2 are meant for these purposes.

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7.3.11 Reviewing BM Results

OHD need to determine if excessive absorption of a CHTH occurred based on BM result. Determination of poisoning is also critical when reviewing the results. Key difference is that in poisoning cases, the excessive absorption is associated with symptoms and signs of chronic medical conditions attributed to the CHTH of concern.

Excessive absorption of a CHTH will lead to the MRP process while poisoning will also involve the need to notify occupational disease/poisoning as per NADOPOD 2004.

7.3.12 Assessment of Fitness to Wear Respirator

The respirator exerts additional burden on breathing efforts and other factors such as anatomical factors may prevent a good fit necessary for the effective wear of the respirator. Improper use of the respirator can result in health effects due to the CHTH exposure. Thus, assessing fitness to wear a respirator is crucial, as described in Appendix 2: Assessment of the Fitness to Wear Respirator.

7.3.13 Fitness to Work Certifications

Fitness to work certification in a MS programme means a certification of fitness to work with the respective CHTH, made by the OHD during the medical examination.

The fitness to work certification will be either ‘fit’ or ‘not fit’.

“Fit” in the MS programme refers to an employee who is fit to work in his assigned job when there is no excessive absorption based on the biological exposure determinant results of the BM samples, no health effects due to the CHTH exposure, no pre-existing conditions that may be exacerbated by CHTH exposure and the employee is fit to wear respirator when required.

“Not fit” in the MS programme refers to an employee who is not fit to work in his assigned job when the biological exposure determinant is above the BEL, there is evidence of health effects due to CHTH exposure or the employee is not fit to wear respirator for CHTH exposure through the inhalation route. ‘Not fit’ can be either temporary or permanent.

There is a possibility that the diagnosis is NOT conclusive. A referral to a clinical specialist or other investigations may be required.

The OHD should still decide whether the employee can be further exposed to the CHTH while waiting for the full medical examination results. This is based on the possibility of further worsening of the suspected medical condition. Possible worsening warrants MRP. The decision of fitness to work is still required pending further investigation results.

7.4 MEDICAL REMOVAL PROTECTION (MRP)

Medical Removal Protection (MRP) is regulated under Regulation 28, USECHH 2000. The process flow of the MRP is presented in Figure 12 below. Under MRP, an affected employee could be removed from a work area temporarily or permanently to avoid further exposure to CHTH.

An employee should be removed within one working day when a medical condition which places the employee at increased risk of health impairment from exposure to CHTH is detected based on one or more of the followings:

a) The clinical findings;

b) Target organ function test abnormality;

c) BM and/or BEM exceed BEL;

d) Pregnant employees;

e) Breastfeeding employees;

f) Others:
   i. Further exposures to the CHTH may worsen the non-chemical work-related medical conditions. For example, exposure to arsenic may aggravate chronic renal disease.
   ii. A medical condition may affect the accuracy of BM and BEM results. For example, kidney impairment may result in diluted urine leading to SG less than 1.010, and thus, the BM result is not valid.

The employee shall be informed of the reason for his/her MRP and is required to adhere to the instruction given.

7.4.1 Pregnancy and Breastfeeding

The CHTH hazard affecting the foetus and breast-fed children is classified under reproductive toxicity and involves all routes of exposures. The risk includes non-routine exposure and accidental contacts such as spills and splashes.

Risk to the mother is also a risk to the pregnancy and the unborn child, thus the MRP is meant to fully protect the offspring from teratogenic effects irrespective of the degree of exposure. The MRP is also intended to protect breast-fed children from harmful toxicant which may interfere with lactation or may be present in or pass through breastmilk.

MRP should be carried out within one working day after being notified by OHD or an occupational safety and health officer who is also a medical practitioner regarding:

a) employee pregnancy status; or
b) breastfeeding employee.

7.4.2 Duration of Temporary MRP

The following criteria shall be used to determine the duration of MRP:

a) The duration required for the medical conditions to normalize when the cause of MRP is a medical condition.
b) The duration taken for the BEI determinant to normalize. Half-life may be useful in determining the MRP duration.
c) The duration of the pregnancy.
d) Depending on CHTH that can affect lactation and decisions should be made on a case-to-case basis.
e) Workplace hygiene and other controls are improved.

The duration of initial MRP is until the date of next return-to-work assessment (RTW). Further decision of the MRP will be decided during the return-to-work assessment (RTW). DOSH shall be notified of subsequent MRP extensions.

Examples:

- In the case of Lead exposure, the duration of MRP is one month as that is the duration anticipated for significant reduction for the concentration of blood lead.

When the employee is fit to return to work, the employer should be notified again (Refer to **Appendix 7:** MS Recommended Forms). To prevent recurrence, the duration of MRP is also subject to improvement of the workplace conditions which caused the removal in the first place.

7.4.3 Permanent Removal

Permanent removal is recommended for:

a) Permanent medical condition due to the CHTH exposures, which obviously will worsen the condition when further exposed.
b) Permanent medical conditions which are not related to the exposures, but the conditions reduce the degree of tolerance to health effects of the CHTH. Significant renal impairment is an example where the CHTH disposal from the body requires a healthy kidney.

7.4.4 Return to-Work (RTW)

The employee may return to work when:

a) The employee has been examined and found to be free from medical conditions that can be worsened by the exposure to the CHTH.
b) The BEI determinant has normalized.
c) Adequate control measures have been implemented to prevent recurrence.

*For pregnant and lactating mothers, the decisions of RTW should be made on a case-to-case basis.

7.4.5 Process Flow of MRP and RTW

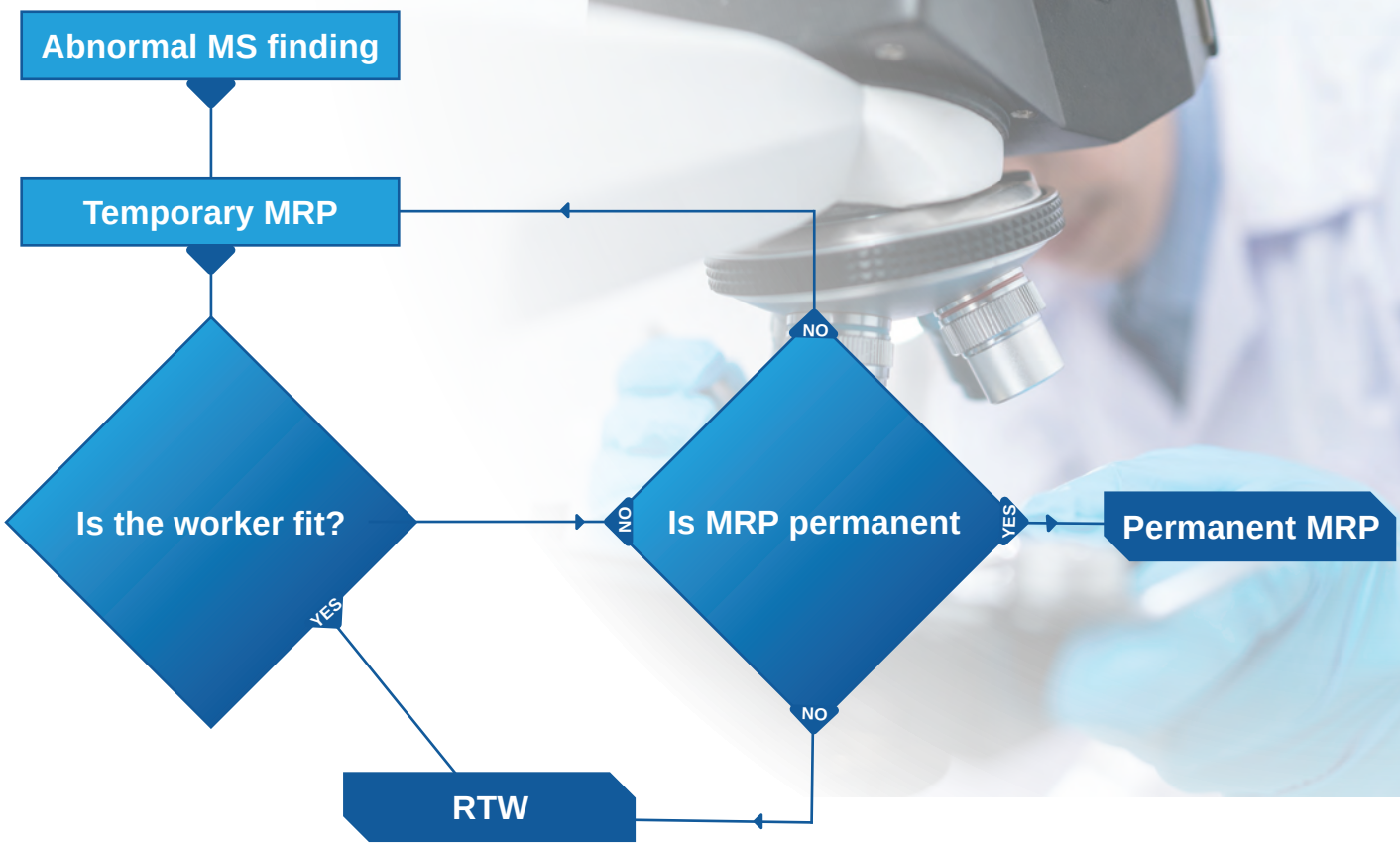

Figure 12. Process Flow of MRP

7.5 FREQUENCY OF MS PROGRAMME

Generally, the MS should be conducted at intervals of not more than twelve months. More frequent MS programme is recommended if there is a case of MRP or there is a concern on the work-related health effects reported during the medical examination whether historically or by medical examination findings. Three monthly or 6 monthly programmes are recommended based on the severity of the concern.

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7.6 CESSATION OF THE MS PROGRAMME

MS is regulated by the USECHH 2000 and initiated by the CHRA. It is a risk-based programme and there will be a point where the MS of the CHTH can be stopped when the control measures are adequate.

A MS is normally stopped when the CHTH is eliminated or substituted

To stop a MS programme, when the CHTH is still in use, but the risk is reduced, the decision shall be based on both the MS findings and also the CHRA. Below are the criteria to be met, for the OHD, when determining when to stop the MS programme:

a) OHD is satisfied that there are no work-related findings based on the MS programme. The duration to consider stopping a MS programme, should be at least two (2) consecutive years of normal findings for all employees in the work unit; and

b) The OHD have to be sure that the control measures or exposure conditions will not result in a repeat case of the abnormal findings. Risk at the workplace is considered adequately controlled when all the conditions below are fulfilled (where applicable):

   i. the results of air monitoring at below half of 8 hours TWA, the ceiling limits (CL), MEL or STEL;

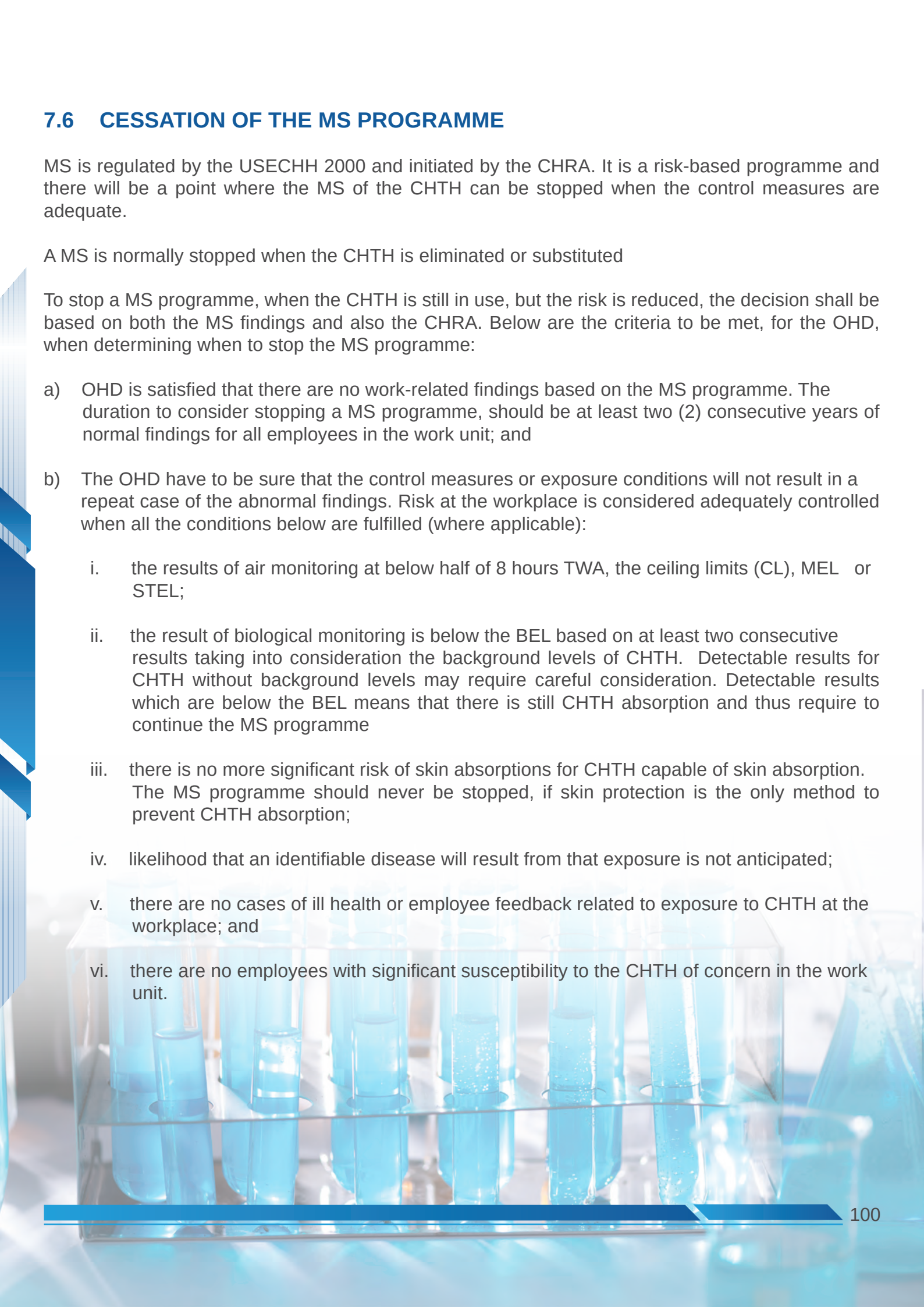
   ii. the result of biological monitoring is below the BEL based on at least two consecutive results taking into consideration the background levels of CHTH. Detectable results for CHTH without background levels may require careful consideration. Detectable results which are below the BEL means that there is still CHTH absorption and thus require to continue the MS programme

   iii. there is no more significant risk of skin absorptions for CHTH capable of skin absorption. The MS programme should never be stopped, if skin protection is the only method to prevent CHTH absorption;

   iv. likelihood that an identifiable disease will result from that exposure is not anticipated;

   v. there are no cases of ill health or employee feedback related to exposure to CHTH at the workplace; and

   vi. there are no employees with significant susceptibility to the CHTH of concern in the work unit.

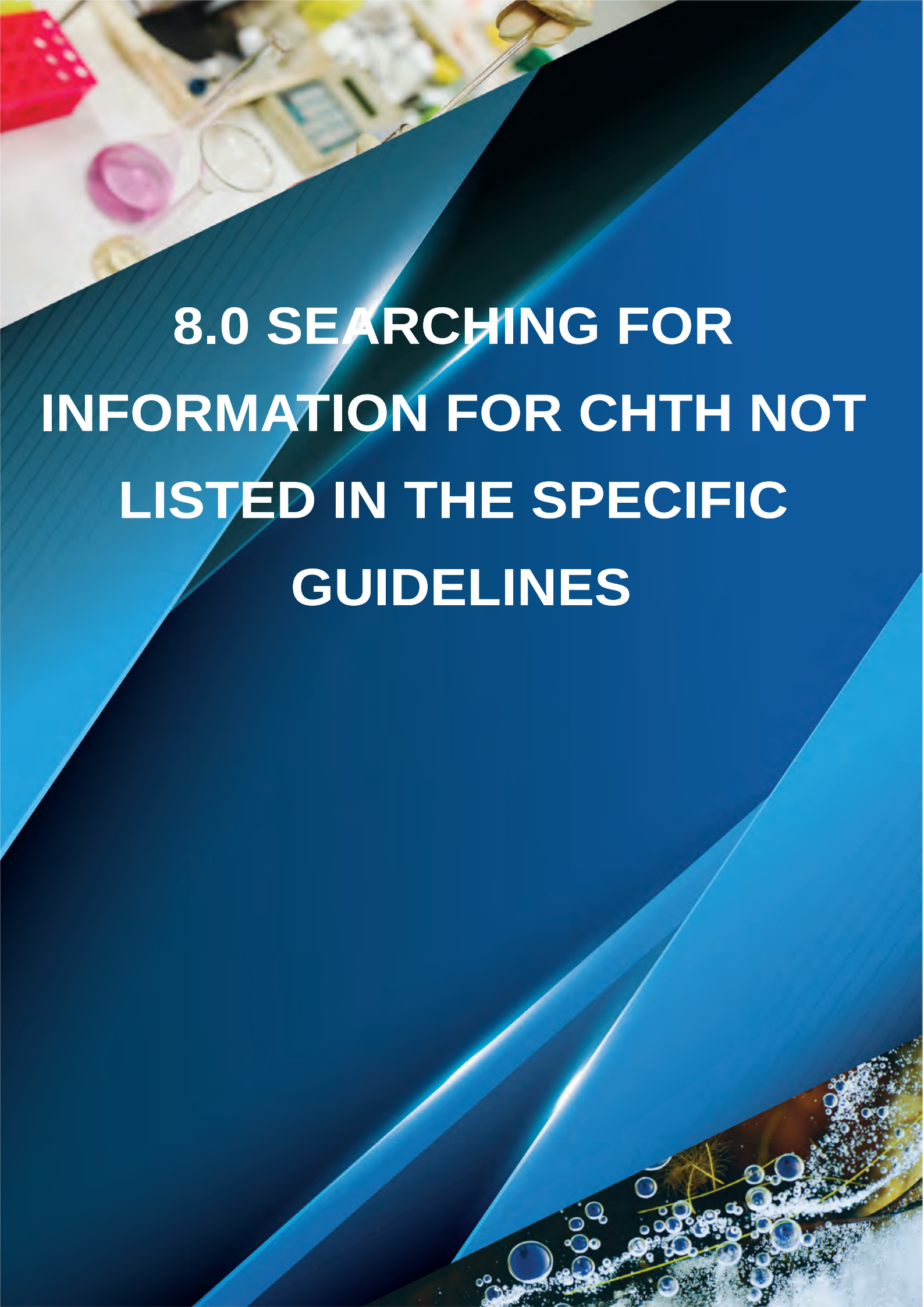


OHD may continue to conduct MS programme even though the above criteria have been met until he is satisfied that the employees are no longer at significant risk.

Exposure to carcinogen and respiratory sensitizers warrant for continuous MS programmes. Cessation of a MS programme where carcinogen and respiratory sensitizers are involved can be tricky, and decisions should only be made based on thorough, valid, and plausible logic and literature evidence.

OHD can refer to the maximum latent period of the CHTH to evaluate and decide when to cease the MS Programme. MS programme can be stopped once the maximum latent period is reached. Information on the maximum latent period can be found in ILO Diagnostic and Exposure Criteria for Occupational Diseases 2010 and European Commission Information Notices on Occupational Diseases: A Guide to Diagnosis 2009.

A written justification by the OHD for cessation of MS programme shall be submitted to the employer.



8.0 SEARCHING FOR INFORMATION FOR CHTH NOT LISTED IN THE SPECIFIC GUIDELINES

8.0 SEARCHING FOR INFORMATION FOR CHTH NOT LISTED IN THE SPECIFIC GUIDELINES

There are 43 CHTH listed in the Specific Guidelines on MS Programme. However, OHD need to perform their own research for CHTH which are not included in these Guidelines. The following criteria are used to assess unlisted CHTH that can be considered for MS.

a) Availability of well-established and approved methods.

b) Opportunities to detect early changes and prevent occupational disease through MS.

c) HEM is possible with a determined target organ, where the progression of health related to exposure, can be observed, and monitored.

d) Availability of chemical determinant threshold level is preferable.

The OHD must keep the references for DOSH audit purposes and for the recommendation of improvements.

The following information is required to carry out an MS programme:

a. The PEL is determined by referring to the Schedule 1 under USECHH 2000. However, MS programmes for CHTH can still be conducted without PEL provided that early health effects can be detected before end organ damage or poisoning occurs. E.g., Herbicides are an example where the irritation effects are significant, and the health effects monitoring are available.

b. The route of exposure.
   - This information helps the OHD to understand how the CHTH enter the body.

c. The toxicokinetics of the CHTH.
   - This information helps the OHD to understand the CHTH absorption, distribution, metabolism, and excretion.

d. The acute health effects.
   - This information helps the OHD to understand the condition of overexposure.

e. Chronic health effects and also the medical conditions caused by CHTH.

f. The target organ of the CHTH and the test/investigation available to monitor the health effects.

g. BEI determinants, if available.
   - The OHD must determine if the accredited laboratory is capable of performing the analysis.

The following are examples of common and reliable references found on the website:

1. Resource with complete guide

- Centers for Disease Control and Prevention, Occupational Health Guidelines for Chemical Hazards (Guide on exposure routes, symptoms and target organs) (Link: https://www.cdc.gov/niosh/docs/81-123/default.html.)

2. Guide on exposure routes, symptoms, and target organs

- Centre of Disease Control and Prevention, NIOSH Pocket Guide to Chemical Hazards (Link: https://www.cdc.gov/niosh/npg/)

3. Guide on toxicity and health effects of the chemical

- National Library of Medicine (NIH), TOXNET (Link: https://www.nlm.nih.gov/toxnet/index.html)
- International Chemical Safety Cards (ICSCs) (Link: https://www.ilo.org/dyn/icsc/showcard.listCards3)

- Agency for Toxic Substances and Disease Registry (ATSDR) (Link : https://wwwn.cdc.gov/TSP/ToxFAQs/ToxFAQsLanding.aspx?id=248&tid=45)
- National Library of Medicine (NIH), PubChem (Link: https://pubchem.ncbi.nlm.nih.gov/)
- World Health Organization, IARC monograph on the identification of carcinogenic hazards to health (Link: https://monographs.iarc.who.int/)

4. References for BM and BEM

- TLV/BEI Guideline, ACGIH (Link: https://www.acgih.org/science/tlv-bei-guidelines/)

The following are examples of CHTH not listed in the Specific Guidelines.

Example 1: Single chemical

CHTH: Sodium Hydroxide

PEL : 2 mg/m^3 ceiling limit (USECHH)

Toxicokinetic:

There is no quantitative data for the absorption of sodium hydroxide through the skin. Solutions which contain 50 % sodium hydroxide have been shown to be corrosive and lethal when applied dermally to mice. Because of its high-level alkalinity, sodium hydroxide in aqueous solution directly causes bond breakage in proteins (especially disulfide bridges). Hair and fingernails are found to be dissolved after 20 hours of direct contact with sodium hydroxide at pH values higher than 9.2. Sodium hydroxide has depilatory effects which have been described after accidental contact with solutions in the workplace. The breakage of bonds in proteins may lead to severe necrosis to the application site. The level of corrosion depends on the period of contact with the tissue, and on the concentration of sodium hydroxide.

Health effects:

Irritation of eyes, skin, mucous membrane; pneumonitis; eye, skin burns; temporary loss of hair.

Strong corrosive action on contacted tissues. INHALATION: dust may cause damage to the upper respiratory tract and lung itself, producing effects from mild nose irritation to pneumonitis. INGESTION: severe damage to mucous membranes; severe scar formation or perforation may occur. EYE CONTACT: produces severe damage.

There is reference for MS guidelines of sodium hydroxide at https://www.cdc.gov/niosh/docs/81-123/default.html.

The medical examination involves:

a) Detail history taking to detect upper airway irritation and inflammation.

b) Physical assessment for signs of chronic irritation and inflammation of the upper airway.

c) Spirometry

d) Chest X-ray

Example 2: Mixtures

Searching information for a mixture, also begins by reading the CHRA report. It contains the hazard classification of the mixture.

A mixture contains multiple chemicals. The health hazards of a mixture depend on the concentration of each chemical and its hazardous properties.

a). CHTH: Welding Fumes

Welding fumes generated by the welding process consist of mixtures of particulates and gases.

Welding fumes exposure depends mainly on:

1. The type of metals being welded and the welding rods.

2. Welding technique: examples are electric arc, MIG, TIG

3. Coatings on the surface of the metal being welded.

Common types of welding fumes are from mild steels and stainless steels.

Mild steels generate iron oxide fumes and manganese, while stainless steel can produce a significant amount of chromium and nickel.

All welding fumes generate irritant particulates of different sizes and are regulated under USECHH 2000 as Welding Fumes Non-Otherwise Classified (NOC).

Irritation to the airway systems produces several pathogeneses, especially inflammation. Welding fumes is known to cause occupational asthma. Methods for the MS programme shall include spirometry and chest X-ray when indicated.

IARC has classified all welding fumes as Group 1 carcinogen (IARC monograph volume 118) causing lung cancer.

Since the specific CHTHs of concern such as manganese, chromium and nickel are provided in these Guidelines, OHD should refer to respective chemicals for the MS programme.

b). CHTH: Paint Mixture

Based on the CHRA, the workers are exposed to chemical mixtures during preparation of the paints. The composition of the paint mixtures is from a thinner and a paint. The tasks that expose the workers to the mixture are weighing of the thinner and paint, pouring them into a mixing container, manual mixing by stirring using a stick, filling the mixture into the container of a sprayer and painting using a spray gun. Other activities that may expose workers to the mixture are the cleaning of related equipment and housekeeping.

Below are the steps to determine the necessary component of the medical surveillance program;

1. Determination of the hazards based on the new hazard classification (H-codes) of mixture from the CHRA report. The CHRA report may also recommend the chemicals which are significant in the mixture based on the consideration of additive effects in the exposure assessment.

   Example:

   Information from Form B and findings of the CHRA report.

   Mixture of epoxy, thinner and Drum Paint General, contains:

   n-butanol<10%

   CAS No: 71-36-3

   Diacetone Alcohol <3%

   CAS No: 123-42-2

   1-methoxy-2-propanol (20-<25)% CAS No: 107-98-2

   Solvent naphtha (petroleum), light aromatic (10-<20)%

   CAS No: 64742-95-6

   Xylene (30-40)%

CAS No: 1330-20-7

Methyl ethyl ketone (30-40)%

CAS No: 78-93-3

Alcohols (25-35)%

CAS No: 8-83-1

Mixture classification:

Acute Tox. 4 (Oral) H302

Acute Tox. 3 (Skin) H311

Acute Tox. 2 (Inh.) H330

Skin Corr. 1B H314

Eye Dam. 1 H318

Resp. Sens. 1 H334

Skin Sens. 1 H317

Repr. 1B H360F

STOT RE 2 H371 (central nervous system)

STOT SE 3 (respiratory) H335

Personal exposure monitoring showed these results:

Xylene at 60% of PEL

Methyl ethyl ketone at 50% of PEL

Mineral oil mist at 60% of PEL (this represents respiratory cancer hazard)

And the combined exposure index exceeding 1.

2. List down all the anticipated health effects, based on the target organs:

Target organs:

- *Central nervous system*
- *Respiratory system*
- *Liver and kidney (based on literature review of the individual substances)*

Health effects:

- *Symptoms and signs of over exposures are irritations to the eye and respiratory system and central nervous system depression (refer to specific guidelines on Xylene)*
- *chronic solvent-induced encephalopathy*
- *Respiratory sensitization*
- *Skin sensitization*
- *Other chronic lung disease*
- *Lung cancer (mineral oil mist)*
- *Kidney impairment*
- *Liver impairment*
- *Acute poisoning due to abnormal exposures*

3. Anticipate the minimum induction duration and the maximum latency duration of each chemical. Additives effect may cause shorter minimum induction period and longer maximum latency period.

   Minimum induction period: a few days for respiratory and skin sensitization, 10 years for toxic encephalopathy, one year for renal impairment.

   Maximum latency period: not applicable for toxic encephalopathy, respiratory and skin sensitization. 24 months for renal impairment.

   Reference: Guidance notes for diagnosis and prevention of the diseases in the ILO List of Occupational Diseases (revised 2010).

4. Determine the assessment tools for the target organs:

   - *Screening questionnaire for chronic encephalopathy*
   - *Chest x ray*
   - *Spirometry*
   - *Liver and kidney function tests*

5. Determine the biological monitoring methods availability for inclusion in the medical surveillance program.

   Available for xylene.

6. All of these decisions should be documented for a review, in order for improvements.

Planning for medical surveillance of a mixture, requires research based on the information explained in this guideline.

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9.0 REPORT AND RECORD KEEPING

9.1 REPORT

9.2 RECORD KEEPING

9.0 REPORT AND RECORD KEEPING

9.1 REPORT

The MS report is recorded in the USECHH forms.

The USECHH forms are arranged according to the process flow of the medical examination of an employee as presented in Table 26.

The objective of an MS report is to:

a) Provide a report of the results and recommendation to the employer in order to take the necessary steps to ensure that the health of the employees is not affected by CHTH in the future.

b) Provide a summary report to DOSH.

The report to the employer, consist of the USECHH forms and be arranged according to the following order for one work unit:

a) USECCH 4 (summary work unit report)

b) USECHH 5ii (summary of individual abnormal result with recommendation) (if applicable)

c) USECHH 5i (MRP) (if applicable)

d) USECHH 3 (fitness to work certification)

Table 26. USECHH Forms for Reporting and Record Keeping.

Updated according to OHD flow of work process	Name of form and the usage	Records kept by OHD	Records kept by Employer	Submission to DOSH
USECHH 1	Examination form: Contain the medical examination findings and the individual fitness to work and other recommendtions. The USECHH 1 form can be used for more than one CHTH.	Yes (7 years)	No (A copy to be kept by the employee)	No
USECHH 2	Summary records of individual employee: Contain the results of an employee throughout the MS programme. The serial data may show progression of health status.	Yes (7 years)	Yes (30 years)	No
USECHH 3	Certificate of Fitness: to certify whether the employee fit or not fit to work with the CHTH.	Yes (7 years)	Yes (30 years)	No
USECHH 4	Summary report for MS: Represent the collective results of a work unit that include recommendations to the company for that work unit.	Yes (7 years)	Yes (30 years)	Yes

USECHH 5ii	Summary of individual abnormal result with recommendation: A list of employees with work related abnormalities and individual recommendations.	Yes (7 years)	Yes (30 years)	Yes
USECHH 5i	MRP	Yes (7 years)	Yes (30 years)	Yes

9.2 RECORD KEEPING

USECHH 2000 requires MS and thereunder relevant records to be kept and maintained by the employer.

Table 26 illustrates the party responsible for the record keeping.

The purpose of proper record keeping in an MS programme includes:

a) To fulfil the requirements as stipulated under USECHH 2000.

b) To provide evidence for compensation and litigation purposes.

c) To monitor progression of symptoms and signs related to CHTH exposures and medical conditions.

d) To establish a database for further analysis for statistics and improvements.

MS programme needs serial records of the medical examination results, for the whole period of the employee's work at the work unit at risk. Abnormalities and progression of the abnormalities may be evidenced when comparing current findings and the previous findings as recorded in USECHH Form 2.

For example, worsening of a spirometry result can still be within the normal reference range, but the serial records may already show worsening over time. Without the comparison, a progressive medical condition related to the CHTH will go unnoticed.

Employer should ensure confidentiality of employees' MS records.

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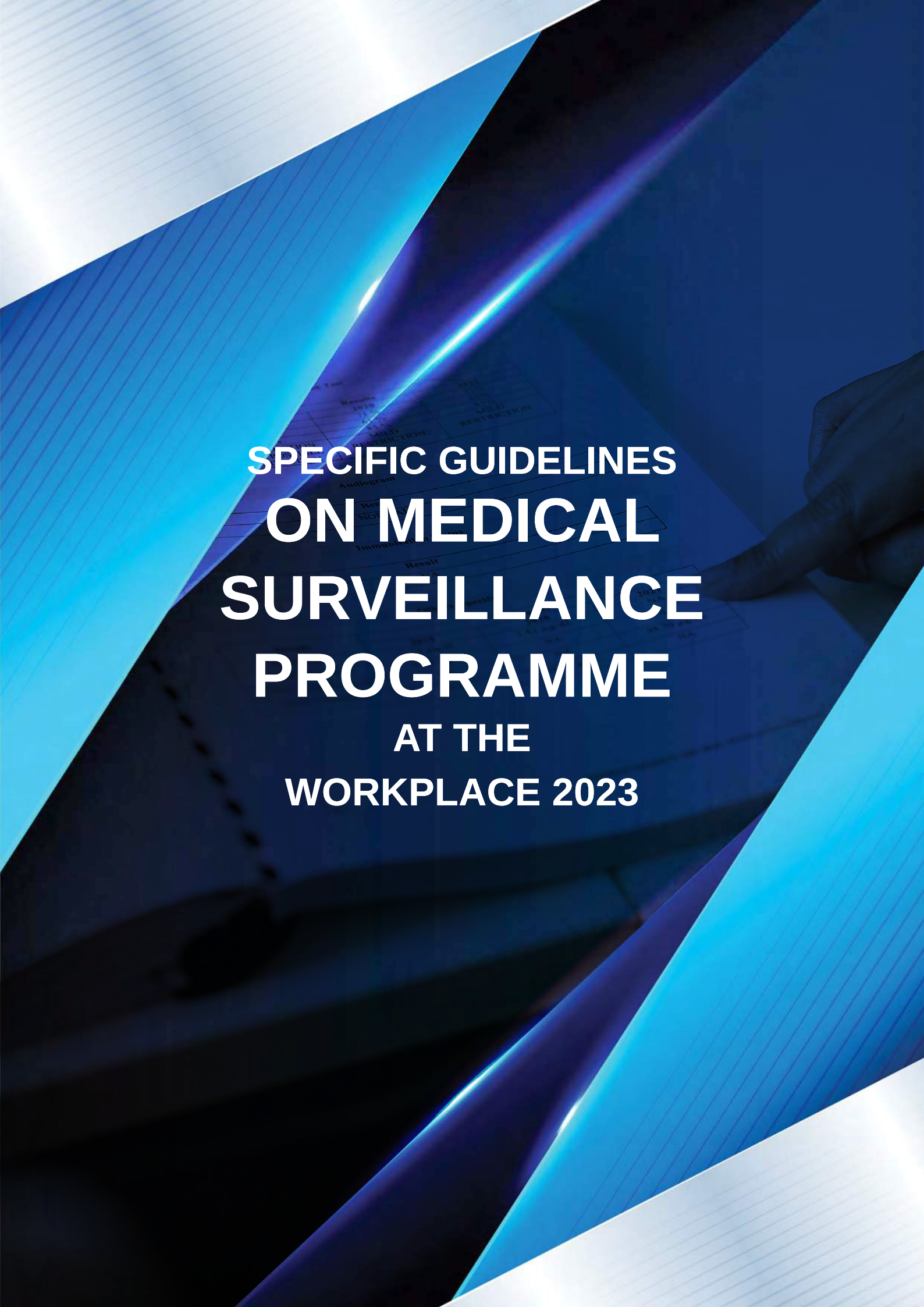

SPECIFIC GUIDELINES ON MEDICAL SURVEILLANCE PROGRAMME AT THE WORKPLACE 2023

ARSENIC

1.0 DESCRIPTION

1.1 Synonyms

Organic and Inorganic Arsenic

Arsen; Arsenic-75; Arsenicals; Arsenic black; Metallic arsenic; Arsenic, solid; Arsenic, solid; Grey Arsenic; Colloidal arsenic; Realgar, Ruby arsenic.

1.2 Occupational Exposure Limit (OEL)

USECHH 2000 (Eight-hour time weighted average limit)	
Arsenic, elemental, and inorganic compounds	0.01 mg/m^3 (TWA)

1.3 Physicochemical Properties

- Arsenic exists in organic and inorganic forms as well as arsine gas.
- Inorganic arsenic compounds exist as odourless, grey, and brittle crystals.
- Non-combustible but may emit irritating and toxic gases or vapours (arsine gas).

1.4 Material Use

Inorganic Arsenic

- Additive in alloy
- Components in electronic devices
- Veterinary medicine
- Agricultural use of insecticides, herbicides, larvicides and pesticides
- Pigment production
- Glass manufacturing as bronzing or decolorization agent
- Opal glass and enamels manufacturing
- Textile printing
- Tanning
- Taxidermy
- Smelting of non-ferrous metals like copper, zinc, and lead.
- Semiconductors

- Wood preservatives
- Production of arsenic compounds most notably arsenic trioxide (As2O3) used pigments, leather hide preservation, ceramic enamels, and anti-fouling paints.

2.0 TOXICITY

2.1 SOURCE OF POTENTIAL OCCUPATIONAL EXPOSURE

ARSENIC COMPOUNDS

Most commonly encountered arsenic compound in occupational settings is trivalent arsenic compound, As (III) and pentavalent arsenic compound, As (V) and occur in the form of mists, fumes, vapours and dusts.

- Manufacture of electrical devices
- Agricultural use: insecticide, herbicide, larvicide and pesticide
- Construction and mining with exposure to arsenic-containing soil
- Production of pigments, use of antifouling paints and textile painting
- Glass, opal, and enamel manufacturing
- Non-ferrous smelting of copper, zinc, and lead

2.2 ROUTE OF EXPOSURE

- Inhalation of dust or fumes (primary)
- Skin absorption
- Ingestion

2.3 TOXICOKINETIC

ELEMENTAL AND INORGANIC ARSENIC

ABSORPTION	• Approximately 60 to 90% of inorganic arsenic is absorbed through the gastrointestnal tract. • Poorly absorbed through intact human skin but able to bind to skin and hair. However, dermal absorption is possible for certain inorganic arsenic compounds (e.g., arsenic acid).

DISTRIBUTION	• Once absorbed into the body, arsenic is transported via bloodstream to other organs bound to sulfhydryl groups. • Target organs of inorganic arsenic are the liver, kidneys, lungs, skin and lymphatic system. • Arsenic is rapidly cleared from tissues except for skin, hair and nails. Most of the arsenic remaining in the body can be found in keratin-rich tissues after two to four weeks (hair, nails, skin, bones and teeth).
METABOLISM	• Arsenic undergoes metabolism in the liver via demethylation to less toxic compounds, monomethylarsenic acid (MMA^{V}), monomethylarsenous acid (MMA^{III}) and dimethylarsenic acid (DMA^{V}). This results in metabolites that are more readily excreted. • Exposure to high doses of inorganic arsenic compounds may result in ineffective methylation to less toxic compounds.
EXCRETION AND HALF-LIFE	• Arsenic is excreted in the urine primarily through the kidneys. Humans excrete a mixture of inorganic, monomethylated, and dimethylated (but not trimethylated) forms of arsenic. • Inorganic arsenic and its metabolites have elimination half-lives of approximately 2–4 days (Lauwerys and Hoet, 2001; NRC, 2001).

- Arsenic compounds bind to sulfhydryl enzymes and disrupts cellular metabolism in the liver, lungs, intestinal wall, and lymph.
- Arsenic generates reactive oxygen species (ROS) that disrupts cell antioxidants. Pentavalent arsenic is able to replace phosphate in synthesis of adenosine triphosphate (ATP) which may disrupt storage of intracellular energy.
- Trivalent arsenic compounds, As (III) inorganic and organic arsenic are more toxic than pentavalent arsenic compounds, As (V). Inorganic arsenic is considered more toxic than organic arsenic.

2.4 HAZARD CLASSIFICATION

INORGARNIC ARSENIC

CLASSIFICATION CODE	HAZARD CLASSIFICATION	H-CODE	SIGNAL
Acute Tox. 3 (Oral)	Acute toxicity category 3 - Oral	H301	DANGER
Acute Tox. 3 (inh)	Acute toxicity category 3 - Inhalation	H331	

Source: *ICOP on Chemicals Classification and Hazard Communication* (Amendment) 2019.

- Cancer Classification IARC
  - Group 1 (Carcinogenic to Human)
  - Chronic exposure to inorganic arsenic results in increased risk of skin, lung, and lymphatic cancer.

3.0 HEALTH EFFECTS MONITORING

3.1 ACUTE EFFECTS

Acute arsenical poisoning via ingestion and inhalation is a generally rare occupational occurrence. Symptoms may develop within ½ to 4 hours of ingestion including throat constriction, dysphagia, epigastric pain, vomiting and watery diarrhoea. Ingestion of inorganic arsenic at high concentration, shock may develop due to fluid loss. Death may be possible in 24 hours.

INORGARNIC ARSENIC

System/Organ	Acute Effects
Ear, Nose and Throat	• Irritation to the angles of the ear, nose and mouth • Throat constriction • Acute trivalent arsenical poisoning occures characterized as serve inflamation of mucous membranes due to increased permeability of the blood capillaries

Eye	• Irritation to the eyes with more sensitive areas being the: ◦ Conjunctiva ◦ Eyelids
Gastrointestinal	• Dysphagia • Epigastric pain • Vomiting • Diarrhea (possibly bloody) • Death may occure due to fluid loss
Hepatobiliary	• Liver damage
Respiratory	• Irritation to the respiratory system specifically the respirtory mucosa causing: ◦ Dyspnoea ◦ Pulmonary edema • Acute arsenical poisoning following inhalation is a rare occupational condition but is usually characterized as respiratory symptoms (cough, chest pain, dyspnea-giddiness, headache, extreme general weakness followed by GI symptoms))
Renal and Genitourinary	• Kidney damage
Skin	• Irritation to the skin with the moist and macerated areas of skin being more sensitive. • Dermatitis frequently observed on the wrist. • Keratoses frequently on the palm and soles. • Arsenic trioxide and pentoxide may cause skin sensitization and contact dermatitis.

3.2 CHRONIC EFFECTS

System/Organ	Acute Effects
Eye, Nose and Throat	• Ulcerations in the inner nose
Gastrointestinal	• Chronic arsenical posoning due to ingestion is rare in an occupational setting but may occur by swallowing sputum with inhaled organic arsenic as well as unhygienic eating habits. Symptoms include: ◦ Weight loss, nausea and diarrhoea which alternates with constipation

Hepatobiliary	• Chronic hepatitis and cirrhosis • Liver damage
Haematological	• Normochromic anemia • Neutropenia • Thrombocytopenia • Aplastic anemia • RBC basophilic stippling
Nervous System CNS and PNS	• “Gloves and stockings” distribution of paresthesias • Nerve damage • Chronic poisoning is separated into three phases: • Phase One: weakness, loss of appetite, nausea and occasional vomiting, heaviness in stomach and diarrhea • Phase Two: Conjuctivitis, catarrhal state of mucous membranes of nose, larynx and respiratory passages. Coryza, hoarseness and mild tracheobronchitis may develop. Lesions of nasal septum, skin, eczemoid (allergic in type) • Phase Three: Symptoms of sensory peripheral neuritis. Severe cases result in motor paralysis in which the toe extensors and peronei are usually affected
Renal and Genitourinary	• Kidney damage: ◦ Tubular and glomerular damage ◦ Oliguria ◦ Uremia
Respiratory	Causes skin cancer
Reproductive	Possible reproductive hazard
Skin	• Thickening of the skin with patches of darkening and loss of pigment • Pigmentation and eruption of the skin • Peripheral neuritis • Skin lesions ◦ Appear melanotic and keratotic and may sometimes resemble intradermal cancer of the squamous cell type without infillrative properties
Others	• Hair loss • Horizontal white lines seen on fingernails and toenails

4.0 MEDICAL SURVEILANCE PROGRAMME

ELEMENTAL AND INORGANIC ARSENIC

4.1 INDICATIONS

Any occupational exposure to arsenic and its compound >50% PEL and/or possibility of excessive skin absorption.

4.2 CLINICAL EXAMINATION AND RELEVANT INVESTIGATION

- Clinical examination and baseline data with particular emphasis on the nervous system, liver, skin, nasal septum, lungs and lymph nodes.
- History of smoking, medication use, alcohol consumption, previous occupation.
- Full blood Count including differential count.
- Renal function test.
- Liver function test.
- Urine Arsenic samples should be taken at the end of shift at the end of the work week. Ensure that employee avoids seafood for three (3) days prior to urine collection.
- Chest X-ray (if indicated).

4.3 PRE-PLACEMENT MEDICAL EXAMINATION

- Clinical examination and relevant investigations as listes in 4.2.
- Baseline urine arsenic
- Decision for fitness to work:
    - Employees with disease of the skin, renal, kidney and respiratory system should not work in areas where there is significant inorganic and organic arsenic.
    - Detect early skin changes (hyperpigmentation and thickening)

4.4 PERIODIC MEDICAL EXAMINATION

- Clinical examination and relevant investigations as listed in 4.2.
- Frequency of periodic medical examination - annually.

4.5 BIOLOGICAL MONITORING

DETERMINANT	SAMPLING TIME	BEL	NOTATION
Inorganic Arsenic and methylated metabolite in urine (exclude gallium arsenide and arsine)	End of shift at end of work week	35 µg As/L	B

Source: TLVs & BEIs ACGIH, 2022.

Note:

- Need to confirm with the laboratory, whether they are testing for total arsenic or inorganic arsenic. Total arsenic has significant background levels and cofounders such as organic arsenic from seafoods. If the laboratory is testing total arsenic, then please ensure that the employee avoids seafood at least 3 days or 1 week as recommended by OSHA USA.
- Arsenic and its metabolites accumulate over the work week. Urine samples should be taken at the end of shift at the end of the work to obtain sample representative of exposure over the previous days.
- Arsenic has a short half-life therefore blood As is less useful than urine levels. Urinalysis is currently the most reliable procedure for monitoring employees exposed to arsenic.
- Unexposed individuals normally show levels above 0.05 mg/L (background level).

- **Laboratory Method**
  - Sampling procedures

Arsenic (Total Inorganic) in Urine	
Container	Plastic container (acid washed or free from trace metals) Note: *Please request container from the laboratory *Own container can be used (provide at least one unit container to the lab for blank test)
Transport	Urine specimens should be refrigerated
Stability	• Urine specimens are stable at room temperature for 5 days. • Urine specimens are stable for 28 days when refrigerated or frozen.
Preservation	• No specific preservative required. • Avoid exposure to gadolinium-based contrast media and seafood con sumption 4 hours prior to sample collection.
Volume	• Requested volume: 30 mL • Minimum volume: 10mL

◦ Analytical equipment/procedure

CHEMICAL	ANALYTICAL EQUIPMENT PROCEDURE
Total Inorganic Arsenic (Inorganic Arsenic and methylated metabolite in urine, excluding gallium arsenide and arsine).	Inductively Coupled Plasma/Mass Spectrometry (ICP/MS).

5.0 MEDICAL REMOVAL PROTECTION

• Indications for removal

Temporary MRP due to medical determination	
Cases of definite or suspected poisoning and excessive absorption	All cases
Evidence of cancer	All cases
Persistent liver abnormalities	• One or more abnormal result in the liver function on at least 2 occasions • Test being carried out preferably not more than one month apart
Temporary MRP due to elevated urine arsenic	
Urine arsenic levels • Repeat test must be done	>35 µg/L
Temporary MRP due to pregnancy	
Pregnant and breast-feeding employee	All individual/cases

• All employee undergoing MRP should have repeat urine arsenic examination at **3-monthly intervals or earlier**.
• They should not return to arsenic work until the urinary arsenic level falls below BEL and all other biochemical results have returned to normal and any related signs and symptoms have disappeared.
• Cases with abnormal liver function tests should be investigated to exclude effects due to arsenic.
• Cases with anemia, proteinuria or haematuria should be investigated to exclude effects due to arsine.

6.0 RETURN TO WORK

- Return to Work is based on:
    i. BEL
        - Return to work when the results are below the BEL.
    ii. Other biochemical results
        - Abnormal biochemical results have returned to normal.
    iii. Medical condition
        - Liver function returns to normal.
        - Associated signs, symptoms and effects of disease no longer detected.
    iv. Workplace management
        - Ensure workplace hygiene is safe and healthy and does not place the employee at risk of material impairment to healtch from exposure to arsenic.

7.0 NOTIFICATION TO DOSH

DOSH shall be notified within 7 days of confirmed occupational disease/poisoning as listed in NADOPOD 2004 using the relevant notification forms.

8.0 TREATMENT AND MANAGEMENT

- Seek medical attention for acute exposure upon contact to eyes and/or skin, inhalation and/or ingestion.
- Upon contact with skin, clothing should be removed and washed immediately with soap and water.
- If inhaled, the employee should be removed from exposure and given rescue breathing if breathing has stopped and CPR if heart has stopped.
- If ingested, provide large quantities of water to induce vomitting. Do not induce vomiting on unconscious individuals.

9.0 PREVENTIVE MEASURES

- Employees should maintain hygiene and sanitation.
- Early detection of signs of absorption, skin contact irritation and sensitivity.
- Using appropriate protective equipment uncluding protective gloves and clothing to prevent skin contact.
- When working with powder or dust, wear dust-proof chemical goggles and face sheild or full face-piece respiratory protection.
- If skin comes into contact with arsenic, wash skin immediately with soap.

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ARSINE GAS

1.0 DESCRIPTON

1.1 SYNONYMS

Arsenic hydride; arsenic trihydride; arsenous hydride; hydrogen arsenide (OSHA).

1.2 OCCUPATIONAL EXPOSURE LIMIT (OEL)

USECHH 2000 (Eight-hour time weighted average limit)	
Arsine	0.05 ppm (TWA) 0.16 mg/m^3 (TWA)

1.3 PHYSICOCHEMICAL PROTERTIES

- Arsine gas exists as colourless, compressed liquefied gas with a distinct odor. An extremely toxic gas which may cause death if inhaled in sufficient quantities.

1.4 MATERIAL USE

- Semiconductor and metals refining industries.

2.0 TOXICITY

2.1 SOURCES OF PONTENTIAL OCCUPATIONAL EXPESURE

- Semiconductor and metals refining industries.

2.2 ROUTE OF EXPOSURE

- Inhalation of arsine gas following release into the air.

2.3 TOXICOKINETIC

ABSORPTION	• Absorbed readily absorbed into blood circulation via respiratory tract • Rapidly dissolved in body fluid
DISTRIBUTION	Following inhalation of arsine gas into the respiratory system, the gas enters red blood cells and causes haemolysis.

Metabolism	Oxidized to trivalent arsenic, As (III) then to pentavalent arsenic, As (V).
Excretion and Half-Life	• Arsine is rapidly excreted in the form of metabolites via urine. • Half-life of the oxidised form (As3+) after ingestion is 7 hours (Baselt, 1988).

- Arsine toxicity is different from arsenic toxicity. Arsine causes massive hemolysis and results in anemia, jaundice, and hemoglobinuric renal failure. The intensity and length of arsine exposure, and the premorbid condition of the person exposed, will contribute to the time of onset and the severity of illness. For example, exposure of a person with underlying coronary artery disease is likely to result in greater morbidity than exposure of a healthy person at the same dose.

Classification Code	Hazard Classification	H-Code
Acute Tox. 2 (inh)	Acute toxicity category 2-inhalation	H330
STOT RE 2	Specific Target Organ Toxicity - Repeated exposure category 2	H373[(a)]

*(a) - State the target organ
*(b) - State the category (compressed gas, liquefied gas, dissolved gas or refrigerated liquefied gas).
*(c) - For gases under pressure, state the relevant H-code based on its hazard category Source: ICOP on Chemicals Classification and Hazard Communication (Amendment) 2019.

- Potential Teratogenic Agent (ATSDR 2015)
- **Cancer Classification IARC**

  Not listed

- **Cancer Classification NIOSH**

  Ca - Potential Occupational Carcinogen

3.0 HEALTH EFFECTS MONITORING

3.1 SIGNS AND SYMPTOMS

The following is a more comprehensive list of signs and symptoms that may be encountered in a person exposed to arsine. Symptoms are not listed in order of presentation or specificity. Also, partial presentations (an absence of some of the following signs/symptoms) do not necessarily imply less severe disease.

- **General**
  - Weakness
  - Malaise
  - Fatigue
  - Fever
- **Gastrointestinal signs and symptoms**
  - Nausea
  - Vomiting
  - Abdominal pain
  - Liver injury
- **Central nervous system signs and symptoms**
  - Headache
  - Lethargy
  - Convulsions
  - Coma
  - Peripheral neuropathy (1–3 weeks after acute exposure)
  - Neuropsychological symptoms (several days after exposure): memory loss,
  - restlessness, confusion.
- **Cardiovascular signs**
  - Tachycardia
  - Hypotension
  - ECG changes (repolarization, S-T segment, and T-wave changes).
- **Respiratory signs and symptoms**
  - Tachypnea
  - Dyspnea
  - Pulmonary edema
- **Genitourinary signs**
  - Hemoglobinuria
  - Oligouria
  - Renal failure
- **Laboratory findings**
  - Anemia
  - Elevated bilirubin (indirect)
  - Hyperkalemia
  - Damaged red blood cells (basophilic stippling, anisocytosis, Heinz-Ehrlich bodies).

- Low haptoglobin
- Elevated plasma-free haemoglobin levels
- Elevated urinary haemoglobin
- Elevated BUN and creatinine
- Abnormal liver function tests
- Thrombocytopenia
- Elevated blood or spot urine arsenic level.

Note: The actual clinical manifestations of arsine exposure may be more variable than the syndrome described above.

4.0 MEDICAL SURVEILLANCE PROGRAMME

4.1 INDICATIONS

Occupational exposure to arsine gas at >50% PEL and/or exceeding the MEL.

4.2 CLINICAL EXAMINATION AND RELEVANT INVESTIGATION

- Clinical examination and baseline data with particular emphasis on the liver, renal and genitourinary system, and haematological systems.
- Liver function test (Serum bilirubin, alkaline phosphatase, serum transaminase e.g., SGOT, SGPT, gamma-glutamyl transpeptidase).
- Urine examination for protein and blood
- Renal function test
- Estimation of urinary arsenic content in an early morning urine specimen (with creatinine correction). Ensure that the employee avoid seafood for 3 days prior to urine collection as it may contain arsenic.
- Haemoglobin estimation and peripheral blood film examination to look for basophilic stippling.

Note: Fish and shellfish contain very large amounts of organically bound arsenic which are readily absorbed from the GIT and rapidly excreted in the urine

4.3 PRE-PLACEMENT MEDICAL EXAMINATION

- Clinical examination and relevant investigations as listed in 4.2.
- Baseline of urine arsenic.
- Decision for fitness to work:
  - Employees with cardiac or renal disease and those with hypersensitivity to haemolytic agents should not work in areas where there is significant exposure to Arsine gas.

4.4 PERIODIC MEDICAL EXAMINATION

- Clinical examination and relevant investigations as listed in 4.2.

- Spot urine arsenic post acute exposure incidence
- Frequency of periodic medical examination – annually.

4.5 BIOLOGICAL MONITORING

DETERMINANT	SAMPLING TIME	BEL	NOTATION
Arsenic, Elemental and Soluble Inorganic Compounds - Inorganic arsenic plus methylated metabolites in urine	End of shift at end of work week	> 35 µg As/L	B

Source: TLVs & BEIs ACGIH, 2022.

- Laboratory Method
  - o Sampling procedures

Arsenic (Total Inorganic) in Urine	
Container	• Plastic container (acid washed or free from trace metals) or • Plastic container (free from preservative) Note: *Request container from the laboratory *Own container can be used (provide at least one unit container to the lab for blank test).
Transport	Urine specimens should be refrigerated
Stability	• Urine specimens are stable at room temperature for 5 days. • Urine specimens are stable for 28 days when refrigerated or frozen.
Preservation	• No specific preservative required. • Avoid exposure to gadolinium-based contrast media and seafood consumption 48 hours prior to sample collection
Volume	• Requested volume: 30 mL • Minimum volume: 10mL

o Analytical equipment/procedures

Chemical	Analytical equipment/procedure
Total Inorganic Arsenic	Inductively Coupled Plasma/Mass Spectrometry (ICP/MS)

5.0 MEDICAL REMOVAL PROTECTION

- Indications for removal

Temporary MRP due to medical determination	
Cases of definite or suspected poisoning and excessive absorption	All cases especially with haematuria
Temporary MRP due to elevated urine arsenic	
Urine arsenic levels ● Repeat test must be done	> 35 µg As/L
Temporary MRP due to pregnancy	
Pregnant and breast-feeding employee	All individual/cases

- All employee undergoing MRP should have repeat urine arsenic examination at **3-monthly intervals or earlier.**

- They should not return to arsenic work until the urinary arsenic level falls below BEL and all other biochemical results have returned to normal and any related signs and symptoms have disappeared.

- Cases with anaemia, proteinuria or haematuria should be investigated to exclude effects due to arsine.

- All cases recommended for suspension and suspected cases of arsenic/arsine poisoning/excessive absorption must be notified to DG (DOSH).

6.0 RETURN TO WORK

- Return to Work is based on:
    i. BEL
        - Return to work when the results are below the BEL.
    ii. Other biochemical results normalise.
    iii. Medical condition
        - Medical condition is no longer detected.
    iv. Workplace management
        - Ensure workplace hygiene is safe and healthy, and
        - Does not place the employee at increased risk of material impairment to health from exposure to arsine gas.

7.0 NOTIFICATION TO DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed in NADOPOD 2004 using the relevant notification forms.

8.0 TREATMENT AND MANAGEMENT

- Immediate removal of employees exposed to large amounts of arsine gas release (CDC) and perform artificial respiration.

- Transfer to medical facility.

9.0 PREVENTIVE MEASURES

- Employees should maintain hygiene and sanitation.
- Early detection of indication of absorption, skin contact irritation and sensitivity.
- Using appropriate protective equipment including protective gloves and clothing to prevent skin contact.
- When working with powder or dust, wear dust-proof chemical goggles and face shield or full face-piece respiratory protection.
- If skin comes into contact with arsenic, wash skin immediately with soap.

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ASBESTOS

1.0 DESCRIPTION

1.1 SYNONYMS

Asbestos fiber, Ascarite, White asbestos or serpentine (chrysotile), blue asbestos or Riebeckite asbestos (crocidolite), brown asbestos or mysorite (amosite), silicic acid or calcium magnesium salt 8:4 (tremolite), stralite (actinolite), ferro anthophyllite or azolane asbestos (anthophyllite).

1.2 OCCUPATIONAL EXPOSURE LIMITS (OEL)

USECHH 2000 (Eight-hour time weighted average limit)	
Asbestos, all forms (except crocidolite)	0.1 f/ml (TWA)

1.3 PHYSICOCHEMICAL PROPERTIES

- Different colours depending on asbestos type: white or greenish (chrysotile), blue (crocidolite), gray or green (amosite).
- May exist in fibrous or non-fibrous form.
- Odourless, non-flammable and non-volatile

1.4 MATERIAL USE

As thermal and electric insulation such as cement pipe and sheets, flooring, gaskets, friction materials, roof coating, plastics, paints, paper, floor tiles, textiles, fiber jointing and millboard.

2.0 TOXICITY

2.1 SOURCES OF POTENTIAL OCCUPATIONAL EXPOSURE

- Construction and shipyard industries (during the removal of asbestos materials due to renovation, repairs, or demolition).
- General industry in manufacture of asbestos products (such as textiles, friction products, insulation, and other building materials).
- Automotive brake and clutch repair work.
- Open-pit mining operations (drilling and blasting).
- Renovation/demolition work, e.g., old buildings and power stations where asbestos is present.
- Material may have been used as roof tiles, fire-proof doors/partitions, rubbish chutes in high rise buildings.
- Ship breaking and repairing of old ships where asbestos is used for insulation of boilers, pipes and for partitions.

- Insulation work, e.g., replacement and removal of asbestos insulation of pipes, furnaces, ovens, boilers.
- Handling of asbestos products, e.g., fireproof cloth and gaskets.
- Repair and replacement of asbestos brake linings by car and bus mechanics.

2.2 ROUTE OF EXPOSURE

- Inhalation (primary)
- Ingestion
- Skin and/or eye contact

2.3 TOXICOKINETICS

Absorption	• Short asbestos fibers are deposited in the upper respiratory tract where they are cleared by mucociliary action. • Longer fibers are carried into the alveolar regions where they are retained in the lungs for longer periods. • Mucocililary action in the respiratory tract will cause some inhaled fibers to be swallowed.
Distribution	• Only a small proportion of fibers reach the blood. • Distributed with the blood flow into the tissue of various organs (including the brain). • Asbestos targets the respiratory system, eyes, lungs (carcinogenic target organ).
Metabolism	• Retained asbestos in the lungs are cleared via leaching, dissolution, and breakage at a slow rate. • A certain amount of fibers is decomposed by the macrophages in a process that can take a very long time and is influenced by the type, and physicochemical properties of asbestos.
Excretion	• Most inhaled asbestos fibers are expelled but some can lodge and remain lifelong in the lungs. • Ingested asbestos will eventually be excreted in the faeces. • Tiny quantities of fibers are eliminated with the urine. • Half-life depends on the type of asbestos but generally persist for many years in the lungs (U.S. Department of Health and Human Services, 2014).

- Inhalation and cumulative deposition of fine asbestos dust in the lung triggers autoimmune responses.
- This leads to the formation of collagenous connective tissue fibers with alterations of the normal lung tissue structure.

2.4 HAZARD CLASSIFICATION

Classification code	Hazard Classification	H-Code	Signal
Crocidolite, Chrysotile, Anthophyllite, Amosite			
Carc. 1A	Carcinogenicity category 1A	H350	Danger
STOT RE 1	Specific target organ toxicity – repeated exposure category 1	H372**	

Source: European Union, Commission Regulation (EC) 1272/2008.

Cancer Classification IARC

Group 1 (Carcinogenic to Humans)

- Lung Cancer (cigarette smoking is an important synergistic factor and the risk may be increased by more than 50 times when compared to an unexposed non-smoker).
- Mesothelioma (mainly pleural and peritoneal; latency period: more than 30 years which may be low or intermittent).
- Gastro-intestinal cancers and laryngeal cancers (association evidence).

3.0 HEALTH EFFECTS MONITORING

- Asbestosis: latency period of 15 or more years after initial exposure usually related to high cumulative exposure

- Pleural plaques/calcification: latency period of 10 to 20 years after initial exposure which may be low or intermittent.

3.1 ACUTE EFFECTS

- Asbestos is not known to be acutely toxic.
- Skin, eye, and respiratory tract irritation may arise from acute exposure of Airborne substances that may contain asbestos.
- Acute high-level exposure may cause pleural disorders, mesothelioma, or lung cancer after a long latency period of at least 15 to 30 years.

3.2 CHRONIC EFFECTS: MAIN TARGET ORGAN IS THE RESPIRATORY SYSTEM

SYSTEM/ORGAN	Acute Effects
Gastrointestinal	Gastrointestinal cancer
Reproductive	Possesses reproductive toxic effect and may cause ovarian cancer.
Respiratory	• Asbestosis • Lung fibrosis with loss of function in respiration (interstitial fibrosis with dyspnea). • Progressive stiffening of tissue. • Pleural thickening • Pleural hyalinosis • Pleural plaques Both leading to reduced respiratory function • Mesothelioma and lung cancer • Lung cancer • Cancer of the larynx • Pleura mesothelioma • Benign pleural effusion • Chronic bronchitis • Bronchogenic cancer (cigarette smoking may increase risk) • Cancer of larynx
Others	• Asbestosis resulting in finger clubbing

4.0 MEDICAL SURVEILLANCE PROGRAMME

4.1 INDICATIONS

- Based on CHRA manual 3rd edition, the indication of the Medical Surveillance is when the exposure exceeds 50% of the PEL. However, if there are possibilities of high exposures, medical surveillance is still recommended.

4.2 CLINICAL EXAMINATION AND RELEVANT INVESTIGATIONS

- Clinical examination and baseline data with particular emphasis on the respiratory system (medical, occupational & smoking history, exertional dyspnoea and basal crepitations) and the gastrointestinal system.
- Lung function test
- Chest X-ray
- Specialist referral is indicated for confirmation of diagnosis. Examples of investigation that may be needed:
  - Sputum examinations for asbestos bodies, abnormal cells
  - Carbon monoxide transfer factor (DLCO)

4.3 PRE-PLACEMENT MEDICAL EXAMINATION

- Clinical Examination and Relevant Investigation as listed in 4.2.
- Decision for fitness to work:
  - Employees who are found to have abnormal lung function and abnormal chest X-ray are advised not to work in the area exposed to asbestos fibers.
  - Smokers are advised to stop smoking.

4.4 PERIODIC MEDICAL EXAMINATION

- Clinical Examination and Relevant Investigation as listed in 4.2.
- Chest X-ray (if clinically indicated or once in every 3 years)
- Frequency of periodic medical examination - annually.

Note:
It is not yet established whether the disease can be diagnosed at a stage when progression would cease if further exposure to asbestos is avoided.

4.5 BIOLOGICAL MONITORING

- No biological exposure indices determinant based on ACGIH 2022 TLVs and BEIs.

5.0 MEDICAL REMOVAL PROTECTION

- Indications for removal:
  - Clinical findings
    - Abnormal clinical findings
  - Target organ function
    - Abnormal chest X-ray findings
    - Deterioration in lung function
- Duration of temporary MRP
  - MRP based on medical condition.
    - The determination of the temporary MRP duration should consider the following aspects:
      - expected duration of diagnostic procedures
      - duration of recovery.

6.0 RETURN TO WORK

- Indications for permanent MRP
  - All symptomatic cases of definite or suspected asbestosis.
  - All cases with evidence of lung cancer and mesothelioma.
  - Employees with deteriorating lung function or deteriorating chest X-ray findings.
  - Note:
    - Suspended asbestosis cases should be followed up annually or more frequently to exclude complications.
- Return to work based on:

i. Medical condition
  - No longer detected of a medical condition.

ii. Workplace management
  - Ensure workplace hygiene is safe and healthy and does not place the employee at increased risk of material impairment to health from exposure to asbestos.

7.0. NOTIFICATION TO DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed in NADOPOD 2004 using the relevant notification forms.

8.0 TREATMENT AND MANAGEMENT

- There is no definitive treatment for asbestosis.
- All cases of suspected bronchogenic cancer or mesothelioma should be referred to a specialist for further management in a chest hospital/ clinic.
- Symptomatic asbestosis cases may require treatment when indicated.

9.0 PREVENTIVE MEASURES

- Good workplace and personal hygiene and safety.
- Approved personal protective equipment.
- Young persons under 18 years of age should not be exposed to asbestos.
- Employees should be advised to stop smoking as smoking has a synergistic effect on likelihood of lung cancer if there is asbestos exposure.

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AURAMINE

1.0 DESCRIPTION

1.1 SYNONYMS

Auramine O base, Auramine base, Benzenamine, 4,4’ Carbonimidoylbis (N,N’ dimethyl-), 4,4'-(imidocarbonyl) bis (N,N-dimethyl-aniline, 4,4’-Dimethylaminobenzophenonimide, Glauramine; 4,4-Imidocarbonyl)bis (N,N-dimethylaniline), Tetramethyldiaminodiphenylacetimine

1.2 OCCUPATIONAL EXPOSURE LIMIT (OEL)

USECHH 2000 (Eight-hour time weighted average limit)	
Auramine	Not listed

Note:

- Not listed in USECHH 2000 or ACGIH TLV-TWA 2022
- No numerical OELs have been established.
- No safe level of exposure for potential carcinogen.

1.3 PHYSICOCHEMICAL PROPERTIES

- Yellow crystalline powder or flaky material.

1.4 MATERIAL USE

- Used industrially as a dye or dye intermediate for colouring textiles, paper, and leather.
- Used as an antiseptic (in ear and nose surgery and gonorrhoea) and fungicide.
- Used as dye for acid fast bacilli in laboratories.

2.0 TOXICITY

2.1 SOURCES OF POTENTIAL OCCUPATIONAL EXPOSURE

- Auramine plants (Manufacture of auramine).

- Dye industry (Paper dyeing and flexographic printing).
- Auramine O is a hydrochloride obtained by combining 4,4'-carbonimidoylbis (N,N-dimethylaniline) with one molar equivalent of hydrogen chloride. A Fluorescent stain for demonstrating acid fast organisms in a method similar to the Ziehl Neelsen. It also can be used to make a fluorescent Schiff reagent. It has a role as a fluorochrome and a histological dye. It contains an auramine O(1+).

2.2 ROUTE OF EXPOSURE

- Skin absorption (primary)
- Inhalation of vapours
- Ingestion

2.3 TOXICOKINETICS

Limited information on toxicokinetics in human and animal study.

2.4 HAZARD CLASSIFICATION

Classification Code	Hazard Classification	H-Code	Signal
Carc. 2	Carcinogenicity category 2	H351	Warning
Acute Tox. 4*	Acute toxicity category 4	H302	
Eye Irrit. 2	Serious eye damage or eye irritation category 2	H319	

Source: European Union, Commission Regulation (EC) 1272/2008

- **Cancer Classification IARC**
  - Auramine production
    - Group 1 (Bladder cancer)

  - Auramine
    - Limited evidence in humans for auramine.

3.0 HEALTH EFFECTS MONITORING

3.1 ACUTE EFFECTS

System/organ	Acute Effects
Eye	Irritation and possible damage to eyes
Gastrointestinal	Exposure to high doses ● Nausea ● Vomiting
Hematological	Methemoglobinemia
Skin	Dermal absorption ● Dermatitis ● Burns
Others	● May cause tumours. ● May be mutagenic.

3.2 CHRONIC EFFECTS

Auramine has been shown to cause bladder cancer in employees of auramine production.

System/organ	Acute Effects
Renal and Genitourinary	Auramine manufacturing which involved exposure to other chemicals has been casually associated with increase in bladder cancer.
Hepatobiliary	Oral administration in animals: ● Liver tumours
Reproductive	Considered to be potentially genotoxic for human but limited data is available for reproductive toxicity.
Skin	Subcutaneous injection in rats: ● Local sarcomas

4.0 MEDICAL SURVEILLANCE PROGRAMME

4.1 INDICATIONS

- Any occupation where employees are liable to be exposed significantly to Auramine and Auramine products and/or possibility of skin absorption.

4.2 CLINICAL EXAMINATION AND RELEVANT INVESTIGATION

- Clinical examination with particular attention to the skin and eye, respiratory, renal, and genitourinary system.
- Kidney function test
- Urine cytology
- Cystoscopy (if clinically indicated)
- Full blood counts

4.3 PRE-PLACEMENT MEDICAL EXAMINATION

- Clinical examination and relevant investigation as listed in 4.2.

4.4 PERIODIC MEDICAL EXAMINATION

- Clinical examination and relevant investigation as listed in 4.2.
- Frequency of periodic medical examination – annual.

4.5 BIOLOGICAL MONITORING

- No biological exposure indices determinant based on ACGIH 2022 TLVs and BEIs.

5.0 MEDICAL REMOVAL PROTECTION

- Indications for removal:
  - Clinical findings
    - Evidence of health effects that is suspected to be caused by CHTH or may be worsened by CHTH.
  - Target organ function
    - Examination of cells shed from bladder.
    - Abnormal urinalysis.
  - Pregnancy and breastfeeding

- Temporary MRP
  - MRP based on medical condition.
    - The determination of the temporary MRP duration should consider the following aspects:
      - expected duration of diagnostic procedures
      - duration of recovery.
  - All cases recommended for MRP, suspected cases of poisoning and excessive absorption must be notified to the DG of DOSH.
  - All employees with abnormal results must have repeat tests and be referred to urologist.
  - All employees undergoing MRP should have a repeat urine examination (and relevant biochemical test) at monthly intervals.

6.0 RETURN TO WORK

- Return to Work is based on:

i. Biochemical results
  - Abnormal results have returned to normal.

ii. Workplace management
  - Ensure workplace hygiene is safe and healthy and does not place the employee and increased risk of material impairment to health from exposure to auramine.

7.0 NOTIFICATION TO DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed in NADOPOD 2004 using the relevant notification forms.

8.0 TREATMENT AND MANAGEMENT

- All cases of acute poisoning must be immediately removed from exposure and referred for hospital treatment.
- Treatment based on specific medical conditions arising from exposure to the chemical.

9.0 PREVENTIVE MEASURES

- Adequate ventilation.
- Appropriate signage.
- Approved personal protective equipment.
- Chemical goggles and good personal hygiene.

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BENZENE

1.0 DESCRIPTION

1.1 SYNONYMS

Benzol, Benzole, Cyclohexatriene, Pyrobenzole, Benzine, Phenyl hydride, Pyrobenzol, Phene, Mineral naphtha.

1.2 OCCUPATIONAL EXPOSURE LIMIT (OEL)

USECHH 2000	
Benzene	
Eight-hour time weighted average limit	15 min – Short-term exposure limit
0.5 ppm (1.6 mg/m^3)	2.5 ppm

1.3 PHYSICOCHEMICAL PROPERTIES

- Colourless liquid with sweet odour.
- Highly flammable.
- Volatile.

1.4 MATERIAL USE

As an important industrial solvent and component of many unrefined petroleum products.

2.0 TOXICITY

2.1 SOURCES OF POTENTIAL OCCUPATIONAL EXPOSURE

- Benzene production (petrochemicals, petroleum refining, coke, and coal chemical manufacturing).
- Industries that use benzene to produce other chemicals such as styrene.
- Rubber tire manufacturing.
- Storage or transport of benzene and petroleum products containing benzene.
- Manufacture of detergents, explosives, pharmaceuticals, and dye stuff.
- Free release of benzene from crude oil or poorly refined mineral oil.
- Possible contaminant from some solvent mixtures.

2.2 ROUTE OF EXPOSURE

- Inhalation (primary)
- Skin absorption
- Skin and/or eye contact
- Ingestion

2.3 TOXICOKINETICS

Absorption	• The lung rapidly absorbs approximately 50% of benzene in air. • The gastrointestinal tract absorbs approximately 90% of ingested benzene.
Distribution	• Absorbed benzene is rapidly distributed throughout the body and tend to accumulate in the fatty tissues due to its lipophilicity. • Target organs of benzene are the eyes, skin, respiratory system, blood, central nervous system, and bone marrow. • May also cross the placental barrier.
Metabolism	• The liver is an important organ in benzene metabolism in production and detoxification of several reactive metabolites. • The major product of benzene metabolism is phenol. Alternatively, at low exposure levels, benzene is rapidly metabolized and excreted predominantly as conjugated urinary metabolites such as s-phenylmercapturic acid (SPMA) and t't-muconic acid (TTMA).
Excretion and Half- Life	• At higher exposure levels, a large portion of absorbed benzene is excreted as a parent compound in exhaled air due to the saturation of the metabolic pathway. • At lower exposure levels, benzene is rapidly metabolized to conjugated metabolites and excreted in the urine. • Benzene has an approximate half-life of 8 hours in the blood. • An inhaled dose of benzene was estimated to be excreted in urine as tt-MA with an elimination half life of 5.0 ± 2.3 hours and S-PMA with a half life of 9.1 ± 3.7 hours (Boogaard & Sittert, 1995).

2.4 HAZARD CLASSIFICATION

Classification Code	Hazard Classification	H-Code	Signal
Flam Liq. 2	Flammable liquid category 2	H225	Danger
Carc 1A	Carcinogen category 1A	H350	
Muta. 1B	Germ cell mutagenicity category 1A	H340	
STOT RE 1	Specific Target Organ Toxicity-Repeated Exposure Category 1	H372 (CNS, hematopoietic system)	
Asp. Haz.	Aspiration hazard category 1A	H304	
Eye Irrit. 2	Serious eye damage or eye irritation category 2	H319	
Skin Irrit.2	Skin corrosion or irritation category 2	H315	

Source: ICOP on Chemicals Classification and Hazard Communication (Amendment) 2019.

- Reproductive toxin: possible risk of gene damage/impaired fertility.

- Cancer Classification IARC
  Group 1 (Carcinogenic to Humans)

3.0 HEALTH EFFECTS MONITORING

3.1 ACUTE EFFECTS

- Slight poisoning is characterized by:
  - Vertigo
  - Dazed feeling
  - Headache
  - Retching

- Very rapid recovery generally takes place after rapid cessation of exposure.
- Alcoholic use may aggravate the health effects.

System/organ	Acute Effects
Cardiovascular	• At high levels may cause cardiac dysrhythmia
Ear, Nose and Throat	• Irritation to the nose and throat
Eyes	• Irritation to the eyes
Gastrointestinal	• Irritation of mouth and stomach
Haematological	• Blood effects
Nervous System CNS and PNS	• Nerve effects: ○ Exaggerated feeling of well-being and excitement ○ Headache ○ Dizziness ○ Slurred speech • Euphorigenic action: ○ Feeling of drunkenness ○ At high levels may cause unconsciousness
Respiratory	• Irritation to the lungs • Lung congestion upon high level of exposure • At high levels may cause: ○ Bronchitis ○ Pneumonia ○ Slowed breathing (which can be fatal)
Skin	• Irritation to the skin (redness and blistering). • Moderate burning sensation but rapidly reversible tissue damage.
Others	Death has occurred at 20,000 ppm for 5-10 minutes or 7500 ppm for 30 minutes.

3.2 CHRONIC EFFECTS

SYSTEM/ORGAN	Chronic Effects
Cardiovascular	• Heart damage

Gastrointestinal	• Loss of appetite • Nausea • Weight loss

Hematological	• Blood effects
	  ○ Leucopenia (decreased leukocyte counts)
	  ○ Thrombocytopenia (decreased platelet counts)
	  ○ Anaemia
	  ○ Pancytopenia
	  ○ Aplastic anaemia
	  ○ Myelodysplastic syndrome
	• Leukemia
	  ○ Acute myeloid leukemia (AML)
	  ○ Acute non-lymphocytic leukemia (ANLL)
	  ○ Chronic lymphocytic leukemia
	• Lymphoma
	  ○ Non-Hodgkin's lymphoma (NHL)

Hepatobiliary	• Liver damage
Musculoskeletal	• Muscle weakness
Nervous System CNS and PNS	• Headache • Nervousness • Fatigue • Dizziness • Irritability • Temporary paralysis has been reported.
Renal and Genitourinary	• Urothelial cancer
Reproductive	• Breast cancer • Fetotoxic effects
Skin	• Degrease of skin • Dry dermatitis

4.0 MEDICAL SURVEILLANCE PROGRAMME

4.1 INDICATIONS

Any occupational exposure to benzene exceeding 50% of the PEL and/or significant skin absorption or evidence of health effects.

4.2 CLINICAL EXAMINATION AND RELEVANT INVESTIGATION

- Past, present, and anticipated future exposure to benzene
- Clinical examination with emphasis on haematological systems including blood forming organs, nervous system, skin and eyes and respiratory system.
- Haematological and central nervous system disorder.
- Current usage of medication with potential hematotoxic side-effects.
- Haemoglobin level.
- Full blood picture/ peripheral blood film to look for blast cells.
- Urinary SPMA estimation in an end-of-the-shift urine sample taken mid-week (creatinine-corrected).
- Liver function test.
- Renal function test.
- Chest X-ray.

4.3 PRE-PLACEMENT MEDICAL EXAMINATION

- Clinical examination and relevant investigation as listed in 4.2. (conducted within three months of employment).
- Baseline urine SPMA.
- Decision for fitness to work.
    - Employees with a history of myelodysplastic syndrome, young persons under 18 years of age, pregnant/nursing mothers and persons diagnosed with liver disease are unfit for exposure to benzene.

4.4 PERIODIC MEDICAL EXAMINATION

- Clinical examination and relevant investigation as listed in 4.2.
- Frequency of periodic medical examination
  - Annual
  - Any suspected abnormal findings, laboratory investigation shall be repeated immediately. If the results are still abnormal, MRP shall be carried out. Re-examination shall be carried out within 1 month.

4.5 BIOLOGICAL MONITORING

Determinant	Sampling time	BEL	Note	Notation
S-Phenylmercapturic acid in urine	End of shift	25 µg/g creatinine	• Highly specific marker for recent benzene exposure (not for mid- or long-term exposure) • Reflects the internal dose with almost similar accuracies.	B

Note :

- Inhalation of tobacco smoke increases background levels, and this should be considered in the interpretation of the results.
- Urinary phenol is not used because it is not a specific biomarker for exposure below 5 ppm.
- t,t-Muconic acid is not used because it is not useful for low level benzene monitoring exposure at exposures <0.25 ppm. It can also be interfered by sorbitol from the diet.

- **Laboratory Method**
  - Method reference: NMAM 8326
  - Sampling procedures

S-PMA in Urine	
Container	Urine container or polyethylene bottles (without preservative). Note: • Request container from the laboratory. • Own container can be used (provide at least one unit container to the lab for blank test).
Transportation	Send refrigerated or frozen with ice or dry ice

Stability	● Specimens are stable when frozen (at least 8 weeks). ● Specimens for phenol test are stable in room conditions for 4 days, refrigerated for 7 days and frozen for 1 month.
Preservation	No specific preservative is required.
Volume	● Requested volume: 30 mL ● Minimum volume: 10 mL

Note :

- The creatinine in the urine should be measured within 24 hours of sample collection.

○ Analytical Equipment/procedure

Determinant	Analytical equipment/procedure
Urine SPMA	HPLC-tandem mass spectrometry
Creatinine	Colorimetry

Medical Removal Protection

- Indications for removal
  - o Clinical findings
    - ▪ Evidence of health effects such as signs of:
      - Anemia
      - Thrombocytopenia
      - Lymphadenopathy
  - o Target organ function
    - ▪ Hemoglobin and Full Blood Count
      - Either a decline from an absolute normal or an individual's baseline to a subnormal value or a rise to a supra-normal value, are indicative of potential toxicity, particularly if all blood parameters decline, for example:
        - o Anemia
        - o Macrocytosis
        - o Neutropenia
        - o Thrombocytopenia
        - o Thrombocytosis
        - o Detection of blast cell
  - o BEL
    - ▪ Exceed BEL
  - o Pregnancy and breastfeeding

- All employee undergoing MRP should have repeat urine SPMA at monthly intervals.

- All cases recommended for MRP, and suspected cases of benzene poisoning/excessive absorption or cancer must be notified to the DG of DOSH.

6.0 RETURN TO WORK

- Monthly assessment until fit to return to work.
- Return to work is based on:
    - i. BEL
        - Return to work when the results are below the BEL.
    - ii. Other Biomarker levels
        - Haematological results are normal.
    - iii. Medical condition
        - Symptoms and signs have resolved.
    - iv. Workplace management
        - Ensure workplace hygiene is safe and healthy and does not place the employee at increased risk of material impairment to health from exposure to benzene.

7.0 NOTIFICATION TO DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed in NADOPOD 2004 using the relevant notification forms.

8.0 TREATMENT AND MANAGEMENT

- All cases of acute poisoning must be immediately removed from exposure, provide appropriate first aid measures, and refer for hospital treatment.
- Treatment based on specific medical conditions arising from exposure to the chemical.
- No antidote for benzene poisoning.

9.0 PREVENTIVE MEASURES

- Good workplace and personal hygiene and safety.
- Approved personal protective equipment.
- Young persons under 18 years of age and pregnant/nursing mothers should not be exposed to benzene.
- Employees with liver disease and/or anaemia should not work in areas where there is significant benzene exposure.
- Employees should not smoke as smoke from one cigarette contains 60-80mg of benzene: a typical smoker inhales 1-2 mg of benzene daily. This may confound low-level benzene exposures.
- Benzene is prohibited for cleaning & degreasing purposes in Malaysia.

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BENZIDINE

1.0 DESCRIPTION

1.1 SYNONYMS

4,4-Diaminobiphenyl, p-Diaminodiphenyl, 4,4-Biailine, 4,4-Diaminodiphenyl, biphenyl-4,4-diamine, 4,4-Biphenylenediamine, 4,4-Diphenylenediamine, p,p-Diaminobiphenyl, p,p-Bianiline, 4-(4-aminophenyk)aniline, p,p-Dianiline, Benzidin, Fast Corinth Base B, p,p-Bianiline

1.2 OCCUPATIONAL EXPOSURE LIMIT (OEL)

	USECHH 2000 (Eight-hour time weighted average limit)
Benzidine	Not listed

1.3 PHYSICOCHEMICAL PROPERTIES

- Aromatic amines that exist as a crystalline grayish-yellow, white, or reddish-gray powder at room temperature.
- Darkens to brownish-red when exposed to oxygen and light.
- Combustible and produces toxic fumes of nitrogen oxides when burned or decomposes.
- High solubility in less-polar solvents like diethyl ether and ethanol, very soluble in hot water but only slightly soluble in cold water.
- It is a manufactured substance and does not exist naturally.

1.4 MATERIAL USE

- Reagent in dye manufacturing (azo dye stuffs)
- Hardener in Rubber industry (now discontinued)
- In chemical and biological analysis (now discontinued)
- Plastic films manufacturing
- Detection of H_2O_2 in milk
- Security paper production

2.0 TOXICITY

2.1 SOURCE OF POTENTIAL OCCUPATIONAL EXPOSURE

- Dye production specifically azo dyes in leather, textile, and paper industries.
- Wastewater from tannery dyeing.
- Reagent for detection of blood.
- Rubber compounding agent.

2.2 ROUTE OF EXPOSURE

- Inhalation
- Dermal absorption
- Ingestion

2.3 TOXICOKINETIC

Absorption	• May be absorbed within a few hours into the lungs via inhalatio or into the intestine via ingestion. • Readily absorbed through the skin.
Distribution	• The target organ or points of attack are the skin, bladder, kidney, and liver.
Metabolism	• Metabolized primarily in the liver to more reactive N-hydroxyarylamides and N-hydroxylamine which contributes to the carcinogenicity of benzidine. • Undergoes metabolic activation via N-oxidation to form electrophilic compounds which binds covalently to DNA. • Causes oxidative stress by generating reactive oxygen species (ROS). • May also damage DNA and biomolecules because of lipid peroxidation and ROS generation. • Benzidine is metabolized via N-acetylation, N-oxidation and N-glucuronidation to form primary metabolites N-acetylbenzidine, N,N'-diacetylbenzidine, 3-hydroxybenzidine and 3,3'-dihidroxybenzidine.
Excretion and Half- Life	• Benzidine is excreted via urine primarily as free benzidine and metabolites. • The half-life of benzidine and its metabolites vary.

- Undergoes metabolic activation via N-oxidation to form electrophilic compounds which bind covalently to DNA.
- Causes oxidative stress by generating reactive oxygen species (ROS).
- May also damage DNA and biomolecules because of lipid peroxidation and ROS generation.

2.4 HAZARD CLASSIFICATION

	Classification Code	Hazard Classification	H-code
Benzidine	Carc. 1A	Carcinogenicity Category 1A	H350
	Acute Tox. 4 *	Acute toxicity, oral Category 4	H302
Benzidine based azo dyes	Carc 1B	Carcinogenicity Category 1B	H350

Source: European Union, Commission Regulation (EU) 2018/669

Information on the hazard classification of Benzidine is not available in the ICOP on Chemicals Classification and Hazard Communication (Amendment) 2019.

- Cancer Classification IARC
  Group 1 (Carcinogenic to humans, bladder cancer)

3.0 HEALTH EFFECTS MONITORING

3.1 ACUTE EFFECTS

SYSTEM/ORGAN	Chronic Effects
Eye	• Irritation to the eyes.
Gastrointestinal	Animal studies indicate an increase in spleen weight.
Hepatobiliary	Animal studies indicate swelling and decrease in weight of the liver
Renal and Genitourinary	Animal studies indicate swelling and decreases in weight of the kidney and blood in the urine.
Respiratory	• Corrosive to the respiratory tract • Irritation of the lungs • Coughing • Shortness of breath • Upon high exposure causes pulmonary edema: ○May be delayed. Require immediate medical attention. ○Worsened by physical exertion

Skin	Corrosive to the skin

3.2 CHRONIC EFFECTS

System/organ	Chronic Effects
Hematological	Blood effects
Hepatobiliary	Liver damage
Renal and Genitourinary	• Increase in urination. • Urinary tract tumors with blood. • Benzidine based dyes has caused bladder cancer. • Kidney damage.
Skin	• Skin allergy. • Skin sensitization (DFG MAK)

4.0 MEDICAL SURVEILLANCE PROGRAMME

4.1 INDICATION

Significant airborne exposures or significant skin absorption and/ or evidence of health effects.

4.2 CLINICAL EXAMINATION AND RELEVANT INVESTIGATION

- Clinical examination and baseline data with particular attention to the genitourinary, liver, kidney, and skin.
- Liver function test
- Kidney function test
- Pulmonary function test
- Total blood count
- Urine cytology examination
- Blood and abnormal cells

4.3 PRE-PLACEMENT MEDICAL EXAMINATION

- Clinical examination and relevant investigation as listed in 4.2.
- Decision for fitness to work
    - Employees with disease of the liver and kidney should not work in areas where there is significant benzidine exposure.

4.4 PERIODIC MEDICAL EXAMINATION

- Clinical examination and relevant investigation as listed in 4.2.
- Frequency of periodic medical examination - annual

4.5 BIOLOGICAL MONITORING

- No biological exposure indices determinant based on ACGIH 2022 TLVs and BEIs.

5.0 MEDICAL REMOVAL PROTECTION

- Indications for removal
    - Clinical findings
        - Evidence of health effects that is suspected to be caused by CHTH or may be worsened by CHTH.
    - Target organ function abnormalities.
    - Pregnancy and breastfeeding.
- Temporary MRP
    - MRP based on medical condition.
        - The determination of the temporary MRP duration should consider the following aspects:
            - Expected duration of diagnostic procedures.
            - Duration of recovery.
- All employee undergoing MRP should have repeat urine/blood test.
- They should not return to work until the blood/urine level has normalized, urine cytology is normal, and any related signs and symptoms have disappeared.

6.0 RETURN TO WORK

- Return to Work is based on:
    i. Urine cytology has normalised.
    ii. Medical condition
        - Medical condition is no longer detected.
    iii. Workplace management
        - Ensure workplace hygiene is safe and healthy and does not place the employee an increased risk of material impairment to health from exposure to benzidine.

7.0 NOTIFICATION TO DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed in NADOPOD 2004 using the relevant notification forms.

8.0 TREATMENT AND MANAGEMENT

- All cases of poisoning must be immediately removed from exposure and referred for hospital treatment.
- Seek medical attention immediately if exposed to eyes, skin, inhaled or ingested.
- Contaminated clothing should be removed and washed immediately with soap and water.
- If inhaled, remove employee from exposure and begin rescue breathing if required.
- Immediate transfer to medical facility.
- Shower and change of clothing when exiting area of work with benzidine.
- Protective clothing and equipment removed in impervious containers at the end of work shif- for decontamination and disposal.

9.0 PREVENTIVE MEASURES

- Substitute other less toxic dyes for benzidine.
- Engineering controls
    - No open vessel operation
    - Closed process systems
    - Liquid metering systems
    - Walk-in hoods
    - Specific local exhaust ventilation
    - Suitable collectors to prevent ambient air contamination.
- Personal protective equipment
    - Full body protective clothing
    - Butyl rubber gloves
- Good housekeeping and occupational hygiene practices.
- Establish restricted areas, communicate with employees on the health effects or provide health hazard alerts.
- Provide washroom/shower facilities.
- Prohibit use for manufacture and use for all purposes including any manufacturing process except for research and analytical purposes.
- Appropriate signage.

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DICHLOROBENZIDINE

1.0 DESCRIPTION

1.1 SYNONYMS

3,3'-Dichlorobenzidine, 4,4'-Diamino-3,3'-dichlorobiphenyl, Dichlorobenzidine base, o,o'-Dichlorobenzidine; 3,3'-Dichlorobiphenyl-4,4'-diamine, 3,3'-Dichloro-4,4'-biphenyldiamine, 3,3'-Dichloro-4,4'-diaminobiphenyl.

1.2 OCCUPATIONAL EXPOSURE LIMIT (OEL)

USECHH 2000 (Eight-hour time weighted average limit)	
Dichlorobenzidine	Not listed

Note:
- No numerical OEL have been established.
- No safe level of exposure for potential carcinogen.

1.3 PHYSICOCHEMICAL PROPERTIES

Gray to purple crystalline solid.

1.4 MATERIAL USE

Used in manufacture of pigments and chemical intermediate for polymers and solid urethane plastic production.

2.0 TOXICITY

2.1 SOURCES OF POTENTIAL OCCUPATIONAL EXPOSURE

- Pigments manufacturing and processing plants (paperboard-printing).
- Rubber, plastics, and polymers manufacture industry.

2.2 ROUTE OF EXPOSURE

- Inhalation of airborne dusts (primary)
- Ingestion
- Skin and eye absorption

2.3 TOXICOKINETIC

There is a lack of information on the toxicokinetic profile of dichlorobenzidine. Most of the available information is on urinary elimination of the compound following occupational exposure.

Absorption	In animal studies, dichlorobenzidine is rapidly absorbed into the body and distributed to major organs.
Distribution	Adducts of 3,3'-dichlorobenzidine and N-acetyl-3,3'-dichlorobenzidine to hemoglobin were detected in rats treated with 3,3'-dichlorobenzidine.
Metabolism	Mono- and di-N-acetylated metabolites of 3,3'-dichlorobenzidine have been identified in urine.
Excretion	• Metabolic transformed dichlorobenzidine will be excreted in urine and faeces. • Unchanged dichlorobenzidine occurs as minor product in urine excretion.

2.4 HAZARD CLASSIFICATION

	Classification Code	Hazard Classification	H-code
3,3'-dichlorobenzidine and its salts	Carc. 1B	Carcinogen category 1B	H350
	Acute Tox. 4 *	Acute toxicity, dermal Category 4	H312
	Skin Sens. 1	Sensitization, skin Category 1	H317

Source: European Union, Commission Regulation (EU) 2018/669.

- Cancer Classification IARC
  Group 2B (Possibly carcinogenic to humans)

3.0 HEALTH EFFECTS MONITORING

3.1 ACUTE EFFECTS

Insufficient information on the acute effects of dichlorobenzidine in humans

SYSTEM/ORGAN	Chronic Effects
Gastrointestinal	• GI disturbances
Renal and Genitourinary	• Frequent urination • Dysuria • Hematuria
Nervous System: CNS and PNS	• Headache • Dizziness
Respiratory	• Respiratory irritation. • Upper respiratory irritation
Skin	• Skin allergic sensitization • Caustic burns

3.2 CHRONIC EFFECTS

SYSTEM/ORGAN	Chronic Effects
Gastrointestinal	GI disturbances
Hematological	Animal studies indicate exposure causes leukemia.
Hepatobiliary	Animal studies indicate exposure causes liver injury and liver cancer.
Nervous System: CNS and PNS	CNS effects • Headache • Dizziness
Reproductive	• Animal studies indicate potential adverse effects to developing embryo. • Animal studies indicate cancer formation in mammary gland.
Reproductive	Respiratory effects
Skin	• Dermatitis • Animal studies indicate exposure causes skin cancer

4.0 MEDICAL SURVEILLANCE PROGRAMME

4.1 INDICATION

Any occupation where employees are exposed to significant exposure to dichlorobenzidine and/ or possibility of absorption.

4.2 CLINICAL EXAMINATION AND RELEVANT INVESTIGATION

General examination and baseline information with emphasis on the liver, skin, respiratory and urinary system.

- Full blood picture
- Kidney function test
- Urinalysis and cytology
- Liver function test
- Pulmonary function test (FEV1, FVC & FEV1/FVC)
- Skin examination

4.3 PRE-PLACEMENT MEDICAL EXAMINATION

- Clinical examination and relevant investigation as listed in 4.2.
- Decision for fitness to work
    - Employees with disease of the liver and kidney should not work in areas where there is significant benzidine exposure.

4.4 PERIODIC MEDICAL EXAMINATION

- Clinical examination and relevant investigation as listed in 4.2.
- Frequency of periodic medical examination - annual

4.5 BIOLOGICAL MONITORING

- No biological exposure indices determinant based on ACGIH 2022 TLVs and BEIs.

5.0 MEDICAL REMOVAL PROTECTION

- Indications for removal:
    - Clinical findings
        - Evidence of health effects that is suspected to be caused by CHTH or may be worsened by CHTH.
    - Target organ function abnormalities
    - Pregnancy and breastfeeding

.

- Temporary MRP
    - MRP is based on medical condition.
        - The determination of the temporary MRP duration should consider the following aspects:
            - expected duration of diagnostic procedures.
            - duration of recovery.

- All cases recommended for MRP, suspected cases of poisoning and excessive absorption must be notified to the DG of DOSH.
- All employees undergoing MRP should have repeat urine and blood biochemical test at monthly intervals.

6.0 RETURN TO WORK

- Return to Work is based on:
    I. Urine and blood biochemical test
        - All abnormal biochemical results have returned to normal.
    ii. Medical condition
        - No longer detected of a medical condition.
    Iii. Workplace management
        - Ensure workplace hygiene is safe and healthy and does not place the employee at increased risk of material impairment to health from exposure to dichlorobenzidine.

7.0 NOTIFICATION TO DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed in NADO-POD 2004 using the relevant notification forms.

8.0 TREATMENT AND MANAGEMENT

- All cases of poisoning must be immediately removed from exposure, provide appropriate first aid mesaures, and referfor hospital treatment.
- Treatment is based on specific medical conditions arising from exposure to the chemical.

9.0 PREVENTIVE MEASURES

- Good workplace and personal hygiene and safety.
- Aprroved personal protective equiptment.

10.0 REFERENCES

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BERYLLIUM AND ITS COMPOUNDS

1.0 DESCRIPTION

1.1 SYNONYMS

Beryllium-9; Beryllium dust; Beryllium metal powder

1.2 OCCUPATIONAL EXPOSURE LIMIT (OEL)

USECHH 2000	
Beryllium and compounds, as Be	
8-hour time weighted average limit	**15 min – Short-term exposure limit**
0.002 mg/m³	0.01 mg/m³

1.3 PHYSICOCHEMICAL PROPERTIES

- Exist as shiny grey metal or powder, or fine granules resembling powdered aluminium.
- Combustible and emits irritating or toxic fumes or gases.
- Finely dispersed particles form explosive mixtures in the air.
- Slightly soluble in water.

1.4 MATERIAL USE

- Moderator in nuclear reactors.
- Beryllium oxide used in ceramics.
- Gyroscopes or wear resistant devices as Beryllium bronze.
- Beryllium alloys used in springs, spot welding electrodes and non-sparking tools.
- Component in X-ray tubes.

2.0 TOXICITY

2.1 SOURCES OF POTENTIAL OCCUPATIONAL EXPOSURE

Industrially used as a pure metal, beryllium oxide and commonly as an alloy with copper, aluminium, magnesium, or nickel (OSHA US).

- Mining of beryllium ore.
- Manufacturing of electrical components, chemicals, ceramics, nuclear reactors.
- Aerospace industry and missile use.

- Component in X-ray tubes.
- Beryllium alloy making and fabricating:
  - Foundry employees
  - Furnace tenders
  - Machine operators
  - Machinists
  - Metal fabricators
  - Welders
  - Dental technicians
- Manufacturing of phosphor.
- Jewellery production.
- Trace amounts of beryllium (<0.1% by weight) can be found in fly ash and numerous abrasive blasting materials like slags, garnet, silica sand and crushed glass.

Soluble beryllium compounds are rarely used industrially and found in primary extraction and concentration facilities and laboratories but in small quantities. (Strupp, 2011)

2.2 ROUTE OF EXPOSURE

- Inhalation of fume or dust.

2.3 TOXICOKINETIC

Absorption	• Absorbed through the lungs. • Limited information on the rate of absorption. • Evidence suggests beryllium may penetrate the placental barrier and be eliminated in breast milk.
Distribution	• Once absorbed, beryllium is distributed to all tissues. • Main deposition occurs in the liver and is transported slowly to the bones. • The target organ or points of attack are the skin, eyes, respiratory system, lungs, liver, spleen, and heart.
Metabolism	• Beryllium and its compounds do not undergo biotransformation.
Excretion and Half- Life	• Half the beryllium that is inhaled is excreted within two weeks. Any beryllium retained in the lungs is cleared slowly and forms granulomata. • The estimated half-life in animals ranges from days to years, with more soluble compounds having shorter half-lives. The estimated half-life in bones has been estimated at 450 days (ATSDR., n.d) • Beryllium is excreted in the urine.

- Beryllium combines with protein causing proliferation of specific CD4 lymphocyte subsequently releasing lymphokines. This process induces granuloma formation.

- Sensitization to beryllium induces disease of the lungs known as beryllicosis. Sensitization of beryllium in the lungs must first occur before a person develops granulomas characteristic of chronic beryllium disease. The outcome of beryllium sensitization varies among individuals with the proportion of beryllium sensitization cases progressing to chronic beryllium disease unknown.

- Chronic beryllium diseases (CBD) occur due to the cell death which releases DNA and cytokines to the lungs. This initiates an inflammatory response in the lungs specifically IL-1R-dependent expression of KC and recruitment of neutrophils.

2.4 HAZARD CLASSIFICATION

Classification code	Hazard classification	H-Code	Signal
Carc. 1B	Carcinogenicity category 1B	H350i	Danger
Acute Tox. 2 (inh)	Acute toxicity category 2 (inhalation)	H330	
Acute Tox. 3 (oral)	Acute toxicity category 3 (oral)	H301	
STOT RE 1	Specific Target Organ Toxicity- repeated exposure category 1	H372 (resp. system)	
Eye Irrit. 2	Serious eye damage or eye irritation category 2	H319	
STOT SE 3	Specific target organ toxicity- single exposure category 3	H335	
Skin Irrit. 2	Skin corrosion or irritation category 2	H315	
Skin Sens. 1	Skin sensitization category 1	H317	

Source: ICOP on Chemicals Classification and Hazard Communication (Amendment) 2019.

- Cancer Classification IARC

  Group 1 (Carcinogenic to Humans)

3.0 HEALTH EFFECTS MONITORING

3.1 ACUTE EFFECTS

Soluble and Insoluble Beryllium Compounds

System/Organ	Acute Effects
Ear, Nose & Throat	Nasopharyngitis: swollen and edematous mucous membranes; bleeding points and ulceration.
Eyes	● Eye irritation ● Conjunctivitis ● Itching and burning ● Allergic reaction possible with subsequent exposure.
Respiratory	● Irritation of the airways and lungs causing: o nasal discharge o chest tightness o cough o shortness of breath o fever o may be fatal. o lung scarring possible. o symptoms may be delayed for days. ● High level of exposure may cause pneumonitis and pulmonary edema.
Skin	● Skin irritation. ● Granulomatous lesions on areas of broken skin. ● Skin sensitization ● Acid salts of beryllium causes contact dermatitis (delayed onset of about 2 weeks following first exposure).
Others	Moderate weight loss

3.2 CHRONIC EFFECTS

System/Organ	Chronic Effects
Cardiovascular	Right heart failure may occur manifested as a non-productive cough leading to vomiting following meals.
Gastrointestinal	Intestinal lesions
Haematological	Chronic beryllium disease (CBD) can be categorized as moderately severe disabling form characterized by: ● oxygen desaturation. ● increase in hematocrit. ● Hypercalciuria.
Hepatobiliary	CBD can be categorized as moderately severe disabling form characterized by the negative effects to liver and spleen.
Musculoskeletal	Damage to bones CBD can be categorized as moderately severe disabling form characterized by bone and joint pain.
Renal and Genitourinary	Kidney stones
Respiratory	● Lung cancer ● Permanent scar tissue in the lungs or other body organs ● Damage to lungs ● Sensitization to beryllium may cause berylliosis. ● CBD can be categorized as: ○ Asymptomatic non disabling disease ○ Mildly disabling form characterized as non-productive cough and dyspnea after exertion, joint pain, and weakness. ○ Moderately severe disabling form characterized by: - distressing cough and shortness of breath. - Negative effects to liver and spleen. - Spontaneous pneumothorax may occur. - Weight loss. - Bone and joint pain. - Oxygen desaturation. - Increase in hematocrit. - Disturbed liver function. - Hypercalciuria. - Spontaneous skin lesions.

Skin	Allergic skin rash. CBD can be categorized as moderately severe disabling form characterized by spontaneous skin lesions.
Others	CBD can be categorized as severely disabling disease characterized by all the previous signs and symptoms with the addition of severe physical wasting and negative nitrogen balance.

4.0 MEDICAL SURVEILLANCE PROGRAMME

4.1 INDICATIONS

Any occupational exposure to beryllium exceeding 50% of the PEL and/or significant skin absorption or evidence of health effects.

4.2 CLINICAL EXAMINATION AND RELEVANT INVESTIGATION

- Clinical examination with emphasis on the respiratory system, skin (with particular attention to atopy and allergic skin and respiratory disease), renal and genitourinary system.
- Pre-employment history, medical history targeted at identifying symptoms.
- Pulmonary function tests
- Chest X-ray
- Full blood count
- Measurement of body weight
- Liver function test
- Kidney function test
- The Beryllium Lymphocyte Proliferation Test (BeLPT), if available.

4.3 PRE-PLACEMENT MEDICAL EXAMINATION

- Clinical examination and relevant investigation as listed in 4.2.
- Decision for fitness to work
  - Atopic subjects and persons with respiratory diseases are considered by some as especially vulnerable.

4.4 PERIODIC MEDICAL EXAMINATION

- Pulmonary function test
- Beryllium lymphocyte proliferation test (BeLPT)
  - BeLPT is a test that determines if the immune system reacts to beryllium as a foreign substance - this reaction results in an abnormal BeLPT. In individuals who are not sensitized to beryllium and do not have chronic beryllium disease (CBD), the immune system does not respond to beryllium in any manner, and they have normal BeLPT results. Individuals must be sensitized to beryllium in order to develop CBD, although not every beryllium-sensitized person will develop CBD.

- The BeLPT is generally conducted using blood but has also been performed using bronchoalveolar lavage (BAL) fluid. The blood test involves drawing blood from a venepuncture and collecting it in tubes for shipment to a lab. The lab separates the immune cells from the blood and mixes the cells with beryllium. The BAL test is performed by inserting a flexible tube into the bronchial tube and inserting saline into the lung. The fluid is collected and sent to a lab, which analyzes it in a similar way to a blood sample. If an employee is sensitized to beryllium, the immune cells will react by multiplying. This is referred to as an abnormal response. Cells from non-sensitized employees do not react and multiply.
- Results from the BeLPT are given as either abnormal, borderline, or normal. If the results are abnormal or borderline, the employer must offer a second test to confirm the results within 30 days.
- What do the results mean? Individuals with either two abnormal BeLPT test results, an abnormal and a borderline test result, or three borderline test results are considered to be "confirmed positive" and should be encouraged to undergo further evaluation to determine if they have CBD. An individual may also be considered "confirmed positive" based on the result of a more reliable and accurate test indicating the person has been identified as having beryllium sensitization.

- Periodic examination every 1 to 3 years to identify the progression of BeS to CBD (Balmes et al. 2014) which include:
  - Review of symptoms and physical examination
  - Pulmonary function test
  - Chest computed tomography scan (upon evidence of declining pulmonary function
  - Bronchoscopy (depending on case)

- Frequency of periodic medical examination - annual

4.5 BIOLOGICAL MONITORING

- No biological exposure indices determinant based on ACGIH 2022 TLVs and BEIs.

5.0 MEDICAL REMOVAL PROTECTION

- Indications for removal
  - Clinical findings

    - Evidence of health effects that is suspected to be caused by CHTH or may be worsened by CHTH.
  - Target organ function

    - Chest X-ray
      - Diffuse, bilateral granulomatosis.
      - Early stages: only enlarged lymph nodes.

    - Positive BeLPT test: Confirmed positive means the person tested has had two abnormal BeLPT test results, an abnormal and a borderline test result, or three borderline test results, obtained from tests conducted within a three-year period.

- Respiratory function
    - ▪ Impaired due to reduced diffusion capacity (can be detected in early stages of disease)

○ Pregnancy and breastfeeding

- All positive BeLPT test should be permanently removed.
- All employee undergoing MRP should have repeat tests
- Positive BeLPT test should be repeated as soon as possible (within 30 days) and to be confirmed positive.
- All cases recommended for suspension and suspected cases of poisoning/excessive absorption must be notified to the DG (DOSH).

6.0 RETURN TO WORK

- Return to work is based on:

i. Abnormal test has returned to normal.

ii. Medical condition

○ No longer detected of a medical condition.

iii. Workplace management

○ Ensure workplace hygiene is safe and healthy and does not place the employee at increased risk of material impairment to health from exposure to beryllium.

7.0 NOTIFICATION TO DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed in NADOPOD 2004 using the relevant notification forms.

8.0 TREATMENT AND MANAGEMENT

- In acute berylliosis, contact with beryllium must be discontinued. Since mild symptoms precede a severe attack, the patient must be admitted to the hospital.

- Seek medical attention immediately if a chemical gets into eyes, comes into contact with skin, is inhaled or ingested. Remove contaminated clothing and wash immediately with soap and water. Begin rescue breathing if required. Induce vomiting by giving large quantities of water (do not induce vomiting for an unconscious person).

- Affected employees must be transferred immediately to a medical facility.

9.0 PREVENTIVE MEASURES

- Good workplace hygiene and safe work practice is important. Personal hygiene should also be stressed.
- Work areas should be monitored to limit and control levels of exposure by recommended use of personnel samplers.
- Provide respiratory protective devices of the appropriate class for employees that are exposed to beryllium above the acceptable levels. Protective clothing should also be provided for these employees, which should be reissued clean on a daily basis. The clothing worn during work with beryllium must be changed. Use of chemical goggles and rubber gloves.
- Adequate ventilation, mechanical filter respirator. Pressurized suit in particularly hazardous places.
- Appropriate signage.

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BIS(CHLOROMETHYL) ETHER

1.0 DESCRIPTION

Bis(chloromethyl) ether is an organic compound with the chemical formula (CH2Cl)2O. It is a colourless liquid with an unpleasant suffocating odour, and it is one of the chloroalkyl ethers. Bis(chloromethyl) ether was once produced on a large scale but was found to be highly carcinogenic and thus such production has ceased.

1.1 SYNONYMS

BCME; Bis-CME; Chloromethyl ether; Dichlorodimethyl ether; Dichloromethyl ether; Oxybis(chloromethane).

1.2 OCCUPATIONAL EXPOSURE LIMITS (OEL)

USECHH 2000 (Eight-hour time weighted average limit)	
Bis(chloromethyl) ether	0.001 ppm 0.005 mg/m^3

1.3 PHYSICOCHEMICAL PROPERTIES

- The chemical formula for BCME is C2H4Cl2O, and its molecular weight is 114.97 g/mol.
- BCME occurs as a colourless liquid
- BCME has a suffocating odour; the odour threshold has not been established.
- The vapour pressure for BCME is 30 mm Hg at 22 °C, and its log octanol/water partition coefficient (log K ow) is -0.38.
- BCME is a chloroalkyl ether compound that exists at room temperature as a colourless volatile liquid with a suffocating odour (chloroform-like odour).
- Dangerous fire risk - flash point below 0°F.
- Vapours much denser than air. Insoluble in water and denser than water.

1.4 MATERIAL USE

- Used as crosslinking agent in the manufacture of ion-exchange resins and in the textile industry.
- Used as an alkylating agent in the manufacture of polymers.
- Used as a solvent for polymerization reactions.
- Used in the preparation of ion exchange resins.
- Used as an intermediate for organic synthesis.
- Used as a research chemical and lab reagent.

2.0 TOXICITY

2.1 SOURCES OF POTENTIAL OCCUPATIONAL EXPOSURE

- Laboratory
- Chemical plant

2.2 ROUTE OF EXPOSURE

- Inhalation of vapour (primary)
- Skin absorption

2.3 TOXICOKINETICS

- No information was located on the toxicokinetic of BCME in humans.
- It is expected that BCME is rapidly degraded in the aqueous environment of tissues, forming formaldehyde and HCl.

Classification code	Hazard classification	H-Code	Signal
Flam. Liq. 2	Flammable liquids category 2	H225	Danger
Carc. 1A	Carcinogenicity category 1A	H350	
Acute Tox. 2 (inh)	Acute toxicity category 2 - inhalation	H330	
Acute Tox. 3 (dermal)	Acute toxicity category 3 - dermal	H311	
Acute Tox. 4 (oral)	Acute toxicity category 4 - oral	H302	

Source: ICOP on Chemicals Classification and Hazard Communication (Amendment) 2019.

- Cancer Classification IARC
    - Group 1 (Lung cancer)

3.0 HEALTH EFFECTS MONITORING

3.1 ACUTE EFFECTS

System/Organ	Acute Effects
Ear, Nose and Throat	Sore Throat
Eye	Eye irritation
Gastrointestinal	Nausea
Musculoskeletal	Weakness
Nervous System – CNS	● Fatigue ● Irritability
Respiratory	● Respirotary tract irritation ● Pulmonary edema
Skin	Skin irritation
Others	● Fever ● Loss of appetite

3.2 CHRONIC EFFECTS

System/Organ	Chronic Effects
Cardiovascular	Chest pains
Eye	Severe corneal necrosis (to animal)
Respiratory	● Chronic bronchitis ● Carcinogenic to humans and causing lung cancer. ● Lungs may be affected by repeated or prolonged exposure.
Others	● Loss of weight ● Oat cell & small cell carcinoma ● May cause genetic damage in humans.

4.0 MEDICAL SURVEILLANCE PROGRAMME

4.1 INDICATIONS

- Any occupation where employees are exposed to any detectable airborne levels (highly carcinogenic) and which are liable to be absorbed or where there is significant risk of ingesting it.

4.2 CLINICAL EXAMINATION AND RELEVANT INVESTIGATIONS

- Clinical examination with emphasis on respiratory system.
- Chest X-ray.
- Sputum cytology.

4.3 PRE-PLACEMENT MEDICAL EXAMINATION

- Clinical examination and relevant investigations as listed.

4.4 PERIODIC MEDICAL EXAMINATION

- Clinical Examination and Relevant Investigations as listed.
- Frequency of periodic medical examination - annually

4.5 BIOLOGICAL MONITORING

- No biological exposure indices determinant based on ACGIH 2022 TLVs and BEIs.

5.0 MEDICAL REMOVAL PROTECTION

- Indications for removal
    - Clinical findings
        - Evidence of health effects that is suspected to be caused by CHTH or may be worsened
    - Target organ function abnormalities
    - Abnormal sputum cytology
- Temporary MRP
    - The determination of the temporary MRP duration should consider the following aspects:
        - expected duration of diagnostic procedures.
        - duration of recovery.
- All cases recommended for MRP, suspected cases of poisoning and excessive absorption must be notified to the DG of DOSH.

6.0 RETURN TO WORK

- When to return to work based on:

  i. Medical condition

  - No abnormality is detected in history, physical examination and investigations after tests are repeated.
  - The results of chest X-ray/sputum cytology are within normal limits after tests are repeated.

  ii. Workplace management

  - Ensure workplace hygiene is safe and healthy and does not place the employee at increased risk of material impairment to health from exposure to BCME.

7.0 NOTIFICATION TO DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed in NADOPOD 2004 using the relevant notification forms.

8.0 TREATMENT AND MANAGEMENT

- All cases of acute poisoning must be immediately removed from exposure, provide appropriate first aid measures, and refer for hospital treatment.
- Treatment based on specific medical conditions arising from exposure to the chemical.
- Eyes and skin that become exposed to BCME should be flushed for at least 15 minutes and washed thoroughly followed by immediate medical attention.

9.0 PREVENTIVE MEASURES

- Use of engineering controls.
- Good work practises.
- Wear personal protective equipment including respirators.

10.0 REFERENCES

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CADMIUM

1.0 DESCRIPTION

1.1 SYNONYMS

- Elemental cadmium; Colloidal cadmium.

1.2 OCCUPATIONAL EXPOSURE LIMITS (OEL)

- Elemental cadmium; Colloidal cadmium.

USECHH 2000 (Eight-hour time weighted average limit)	
Cadmium, elemental and compound, as Cd	0.025 mg/m³ (TWA)

1.3 PHYSICOCHEMICAL PROPERTIES

- Cadmium is bluish-white metal,
- Soft, silvery, ductile metal, chemically similar to zinc.
- Insoluble in water but acids.

1.4 MATERIAL USE

- Used as electrodes for nickel-cadmium batteries.
- Used as a protective coating for iron, steel, and copper; it is generally applied by electroplating, but hot dipping and spraying are possible.
- Cadmium may be alloyed with copper, nickel, gold, silver, bismuth, and aluminium to form easily fusible compounds. These alloys may be used as coatings for other materials, welding electrodes, solders, etc.
- Used as stabilizer for polyvinyl chloride plastics.

2.0 TOXICITY

2.1 SOURCES OF POTENTIAL OCCUPATIONAL EXPOSURE

- Cadmium production and refining.
- Cadmium-related product manufacture plants (Nickel-cadmium battery, cadmium pigments & alloy production).
- Construction & base metal industry.
- Plastics industry, especially compounding of polyvinyl chloride (PVC); used as thermal stabilizer.

2.2 ROUTE OF EXPOSURE

- Inhalation of cadmium dust & fume (primary)
- Ingestion

2.3 TOXICOKINETICS

CADMIUM	
Absorption	• Cadmium is efficiently absorbed from the lungs (25-60%) and GIT (5-10%). • A small amount of the cadmium in food and water (about 1–10%) will enter the body through the digestive tract. If the body does not have enough iron or other nutrients in the diet, it will likely take up more cadmium from the food than usual. • Absorption rate is dependent on the solubility and particle size of cadmium compounds.
Distribution	• Cadmium is bound to red blood cells or proteins and widely distributed in the bloodstream. • Eventually it is accumulated (50-70% of body burden) in the kidney and liver and can remain there for many years.
Metabolism	• There is little or no metabolism of cadmium. • Cadmium is scavenged by metallothionein and cleared in the kidney.

CADMIUM	
Excretion & Half-life	• Most of the oral dose will be excreted in feces. Following inhalation, excretion via the urine and feces are approximately equal. • The estimated half-lives of cadmium in the blood, kidneys and liver are 2–3 months, 10–30 years and 5–10 years, respectively.

2.4 HAZARD CLASSIFICATION

Classification Code	Hazard Classification	H-Code	Signal
Carc. 1B	Carcinogenicity category 1B	H350	Danger
Muta. 2	Germ cell mutagenicity category 2	H341	
Repr. 2	Reproductive toxicity category 2	H361fd	
Acute Tox 2 (inh)	Acute toxicity, category 2 - inhalation	H330	
STOT RE 1	Specific target organ toxicity, repeated expo-	H372[(a)]	

*(a) - State the target organ

Source: ICOP on Chemicals Classification and Hazard Communication (Amendment) 2019.

- **Cancer Classification IARC**

  - Group 1
  - Elevated risk of prostate, kidney, and bladder cancers.

Note:

Cigarette smoking adds to cadmium burden. Each cigarette contains about 1 - 2 mcg cadmium (Cd) of which approximately 25 -50% is retained in the lungs. The average normal gastrointestinal absorption in man ranges from 3 - 7% of ingested cadmium. This increases to as high as 20% with nutritional deficiencies of calcium, iron, or protein.

3.0 HEALTH EFFECTS MONITORING

● Acute Poisoning

- Metal fume fever with fl u-like symptoms of weakness, fever, headache, chills, sweating and muscular pain.
- Delayed pulmonary oedema following fume inhalation; onset within 8 to 24 hours and peaking by 3 days; mortality 15%. If death does not occur, symptoms may resolve within a week.
- Acute renal failure after inhalation of high concentration of fumes.
- Gastrointestinal tract irritation following accidental ingestion.

● Chronic Poisoning

- Renal dysfunction (tubular and/or glomerular damage with low molecular weight proteinuria, e.g., beta2 microglobulinuria, glucosuria, amino aciduria, albuminuria, and reduced creatinine clearance) with latency of about 10 years.
- Emphysema.
- Bone pain; osteomalacia, osteoporosis and fractures.
- Anaemia, teeth discoloration and anosmia.

3.1 ACUTE EFFECTS

System/Organ	Acute Effects
Cardiovascular	Higher doses may affect the cardiovascular system.
Gastrointestinal	Lower ingestion doses may lead to GIT irritation, vomiting, abdominal pain and diarrhea
Hepatobiliary	Higher doses of exposure may affect the renal and lead to renal failure and death.
Nervous System	Higher doses may affect the nervous system.
Respiratory	● Chemical pneumonitis following fume inhalation; onset within 8 to 24 hours; mortality 15%. ● Acute and sometimes fatal cadmium poisoning cases may occur via inhalation route. ● Inhalation of cadmium or cadmium oxide fumes can cause metal fume fever which may progress to pulmonary edema or pneumonia. ● Higher doses may affect the respiratory system and lead to pulmonary edema, bronchitis, chemical pneumonitis, respiratory failure, and death.

3.2 CHRONIC EFFECTS

System/Organ	Chronic Effects
Hepatobiliary	● Liver damage (focal hepatic necrosis) is only observed at high concentrations of cadmium. ● Elevation of hepatic enzymes. ● Cadmium and cadmium compounds increase the risk of liver cancers.
Musculoskeletal	● Osteomalacia and osteoporosis. ● Increased incidence of fractures. ● Hyperphosphaturia. ● Decreased 1-hydroxylation of 25-hydroxy vitamin D in tubular cells.
Renal and Genitourinary	● Chronic oral exposure leads to renal failure, characterized by proteinuria. ● Proximal renal tubular dysfunction. ● Renal calculi. ● The accumulation of cadmium in the kidney affects renal vitamin D metabolism, leading to osteomalacia and osteoporosis (itai-itai disease).
Reproductive	● Cadmium and cadmium compounds increase the risk of prostate cancers. ● There are possible developmental effects of cadmium in humans. ● Birth weights of new-born may be lower but no congenital abnormalities have been reported.
Respiratory	● Chronic inhalation impairs lung function by causing bronchitis, obstructive lung disease and in some cases interstitial fibrosis ● Cadmium and cadmium compounds increase the risk of lung cancers

4.0 MEDICAL SURVEILLANCE PROGRAMME

4.1 INDICATIONS

- Any occupation where employees are liable to be exposed to cadmium above 50% of PEL and/or possibility of absorption.

4.2 CLINICAL EXAMINATION AND RELEVANT INVESTIGATIONS

- Clinical examination with emphasis on hepatobiliary system, renal system, reproductive system, and respiratory system.
- Complete physical examination:
    - Respiratory (chest X-ray & lung function tests: FEV1, FVC & FEV1/FVC).
    - Olfactory sense.
    - Skeletal system.
    - Renal system.
- Tests:
    - Haemoglobin, creatinine level.
    - Renal function test.
    - Liver function test.
    - Urine analysis.
    - Cadmium in blood and urine.

4.3 PRE-PLACEMENT MEDICAL EXAMINATION (WITHIN 3 MONTHS OF EXPOSURE)

- Clinical examination and relevant investigations as listed in 4.2.
- Baseline samples should be taken for cadmium in blood and urine.
- Decision for fitness to work and frequency of periodic medical examination:
    - Employees with diseases of hepatobiliary system, renal system, reproductive system, and respiratory system should not work in areas where there is significant cadmium exposure.

4.4 PERIODIC MEDICAL EXAMINATION

- Clinical examination and relevant investigations as listed in 4.2.
- Cadmium in blood is recommended for periodic testing to determine recent exposures. If the Levels exceeds the BEL, a urine sample for cadmium level should be taken to determine whether the issue is long standing.
- Frequency of periodic medical examination: annually.

4.5 BIOLOGICAL MONITORING

Determinants	Sampling time	BEL	Notation
Cadmium in urine	Not critical	5 µg/g creatinine	B
Cadmium in blood	Not critical	5 µg/L	B

***Total cadmium**

Note:

For cadmium, an employee's exposure can be assessed by either blood or urine samples; but the sample type dictates the information gained about that exposure. Blood cadmium levels reflect more recent exposures (previous months) whereas urine levels reflect current lifetime exposure.

If using biological monitoring to assess the current adequacy of control in the workplace, then blood cadmium is recommended as it is a better reflection of current body burden and will respond more quickly to any improvements or interventions. If a blood sample indicates that excessive exposure has occurred, then a urine sample can be useful in determining whether this is a short-term issue or has likely been ongoing for an extended period of time. A urine sample can also be measured for retinol binding protein to determine if there are any early health effects as a result of the exposure.

- **Laboratory Method**
  - Method reference: NMAM 8310
  - Sampling procedure

Cadmium in Urine	
Container	Polyethylene or trace metal-free plastic container Note: *Request container from the laboratory. *Own container can be used (provide at least one unit container to the lab for blank tests).
Transportation	Urine specimens should be refrigerated.
Stability	● Urine specimens are stable at room temperature for 5 days. ● Urine specimens are stable for 14 days when refrigerated. ● Urine specimens are stable for 30 days when frozen.
Sample Volume	Requested volume: 30 mL Minimum volume: 10 mL

Cadmium in Blood	
Container	EDTA tube Note: *Request container from the laboratory *Own container can be used (provide at least one unit container to the lab for blank test).
Transportation	Blood specimens should be refrigerated.
Stability	● Blood specimens are stable for 30 days at room condition or when refrigerated. ● Frozen specimens are stable at least for 3 months.
Sample Volume	Requested volume: 7 mL Minimum volume: 2 mL

Note: The creatinine in the urine should be measured within 24 hours of sample collection.

○ Analytical equipment/procedure

Cadmium

- Atomic absorption spectrometry (AAS) with electrothermal atomization.
- Inductively coupled plasma mass spectrometry (ICP-MS).

Creatinine

- Colorimetry

5.0 MEDICAL REMOVAL PROTECTION

● Indications for removal

○ Clinical findings

- Evidence of health effects that is suspected to be caused by CHTH or may be worsened by CHTH.

○ Target organ function

- Persistent renal and lung abnormalities:
- one or more abnormal results on at least 2 occasions, the tests being carried out preferably not more than one month apart.

○ BEL

- Exceed BEL

● Pregnancy and breastfeeding

- All cases recommended for MRP, suspected cases of poisoning and excessive absorption must be notified to the DG of DOSH.

Note:

- Inform all women that they are to inform their supervisors as soon as they are found to be pregnant.
- Follow up - do biological monitoring every 3 months with **6 monthly medical examinations**
- Abnormal results - repeat blood Cd, urine Cd levels three monthly until results below BEL, up to 18 months.

6.0 RETURN TO WORK

When to return to work based on:

- Clinical findings
  - i. Blood and urine Cd level as below, and
  - ii. No other medical conditions.

Blood Cd	≤5 µg/l whole blood
Urine Cd	≤5 µg/g creatinine

Note:

If an employee is unable to return to the former position by the end of the 18-month period, the employer must provide the employee with a medical examination to obtain a final medical determination regarding whether the employee can return to the former position or needs permanent MRP from excess cadmium exposure.

7.0 NOTIFICATION TO DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed in NADOPOD 2004 using the relevant notification forms.

8.0 TREATMENT AND MANAGEMENT

- All cases of acute poisoning must be immediately removed from exposure, provide appropriate first aid measures, and refer for hospital treatment.
- Treatment based on specific medical conditions arising from exposure to the chemical.

9.0 PREVENTIVE MEASURES

- Good workplace and personal hygiene and safety.
- Approved personal protective equipment.

10.0 REFERENCES

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CARBON DISULPHIDE

1.1 SYNONYMS

- Carbon disulfide (CS2) is a colourless liquid with an ether-like odour and with high vapor pressure, with high vaporization rate.

1.2 OCCUPATIONAL EXPOSURE LIMITS (OEL)

USECHH 2000 (Eight-hour time weighted average limit)	
Carbon disulphide	10 ppm, 31 mg/m^3 (TWA)

1.3 PHYSICOCHEMICAL PROPERTIES

- Colourless to faint yellow liquid.
- Pure liquid has a pleasant smell, commercial and reagent grade have a rotten egg odour.
- Highly flammable, volatile, and irritating to the eye and skin.

1.4 MATERIAL USE

- Used predominantly in the manufacture of rayon, cellophane, and carbon tetrachloride.
- Used in perfume production and certain varnishes.
- Also used to produce rubber chemicals and pesticides.

2.0 TOXICITY

2.1 SOURCES OF POTENTIAL OCCUPATIONAL EXPOSURE

- Technological processes in the rayon viscose industry.
- Exposure to CS2 emulsion during fumigation for agriculture soil treatment.
- During production of rubber chemicals and pesticides

2.2 ROUTE OF EXPOSURE

- Inhalation (primary).
- Dermal absorption.
- Ingestion of contaminated food and drinking water.

2.3 TOXICOKINETICS

Absorption	• Readily absorbed through the lungs, gastrointestinal tract and to a lesser extent through the skin. • CS2 will achieve an equilibrium in inhaled and exhaled air during the first hour of exposure. • In the state of equilibrium, retention is about 40-50%.
Distribution	• After absorption, CS2 is transported by the blood, being distributed between blood erythrocytes and plasma in the ratio 2:1. • It disappears relatively quickly from the blood and is distributed to various tissues and organs.
Metabolism	• The remaining 70-90% of absorbed CS2 undergoes biotransformation. • The metabolites produced, 2-Thioxothiazolidine-4-carboxylic acid (TTCA) together with less than 1% of unchanged CS2, are excreted in the urine, saliva, and faeces.
Excretion	• Around 10-30% of absorbed CS2 is exhaled. • The elimination of CS2 via exhaled air takes place in the respiratory tract, more slowly from the blood and very slowly from tissues and organs.

2.4 HAZARD CLASSIFICATION

Classification Code	Hazard Classification	H-Code	Signal
Flammable liquids category 2	Flam. Liq. 2	H225	Danger
Repr. 2	Reproductive toxicity category 2	H361fd	
STOT RE 1	Specific target organ toxicity - repeated exposure category 1	H372 (CNS, cardiovascular, kidney)	

Eye Irrit. 2	Serious eye damage or eye irritation category 2	H319	
Skin Irrit. 2	Skin corrosion or irritation category 2	H315	

Source: ICOP on Chemicals Classification and Hazard Communication (Amendment) 2019.

- **Cancer Classification IARC**

  - Not listed

3.0 HEALTH EFFECTS MONITORING

3.1 ACUTE EFFECTS

System/Organ	Acute Effects
Eyes	● Ocular exposure causes immediate severe irritation. ● Nystagmus and diplopia have been reported.
Gastrointestinal	● Ingestion causes irritation to mucous membranes with nausea and vomiting.
Nervous System CNS and PNS	● Systemic effects include headache, nausea, vomiting, nervousness, and tremor. ● In severe cases there may be drowsiness, coma, and convulsions. ● Acute exposure may cause psychosis.
Respiratory	● Irritation to the respiratory tract with coughing, wheezing and dyspnoea. ● Respiratory failure and death from respiratory paralysis.
Skin	● Dermal exposure causes irritation, erythema, and skin peeling. ● Concentrated solutions can cause severe burns.

3.2 CHRONIC EFFECTS

System/Organ	Chronic Effects
Cardiovascular	● Peripheral nervous system effects may disturb heart function. ● Vascular changes of cerebrovascular and cardiovascular system ● Increased incidence of coronary heart disease.
Nervous System CNS and PNS	● Peripheral nervous system is first affected. Manifestations include ○ distal-symmetrical disturbances to the sensitivity of the arms and legs, ○ reduced reflexes, ○ disturbance to heart function, and ○ signs of paralysis. ● CNS damage is possible with behavioral and neurophysiological changes.
Reproductive	● Decreased sperm count and menstrual disturbances.

4.0 MEDICAL SURVEILLANCE PROGRAMME

4.1 INDICATIONS

● Any occupation where employees are liable to be exposed to carbon disulphide above 50% of PEL and/or possibility of absorption.

4.2 CLINICAL EXAMINATION AND RELEVANT INVESTIGATIONS

● Clinical examination with emphasis on the central nervous system and the peripheral nervous system.
● Cardiovascular system
● Liver function tests, blood urea nitrogen, LDL and HDL profile.
● Electrocardiogram.
● Ophthalmic examination including visual acuity and colour vision test.

4.3 PRE-PLACEMENT MEDICAL EXAMINATION

● Clinical Examination and Relevant Investigations as listed in 4.2.

- Decision for fitness to work:
    - Employees with disease of the central nervous system and cardiovascular system should not work in areas where there is significant carbon disulphide exposure.

4.4 PERIODIC MEDICAL EXAMINATION

- Clinical Examination and Relevant Investigations as listed in 4.2.
- Frequency of periodic medical examination – annual
- Referral for Electromyogram to detect abnormalities in the peripheral nerve conduction if clinically indicated.

4.5 BIOLOGICAL MONITORING

Determinants	Sampling time	BEL	Notation
2-Thioxothiazolidine-4-carboxylic acid (TTCA) in urine	End of shift	0.5 mg/g creatinine	B, Ns

- Laboratory Method
    - No method reference currently established for carbon disulfide.
    - Sampling procedures:

TTCA in Urine	
Container	Plastic or polyethylene container (preservative-free). Note: *Request container from the laboratory. *Own container can be used (provide at least one unit container to the lab for blank tests).
Transportation	Specimens should be refrigerated.
Stability	● Specimens are stable at room temperature for 1 day. ● Specimens are stable for 7 days when refrigerated. ● Frozen specimens are stable for 1 month.
Preservation	No specific preservative mentioned.
Volume	Requested volume: 30 mL Minimum volume: 10 mL Sample must be creatinine corrected.

Note:

The creatinine in the urine should be measured within 24 hours of sample collection.

- Analytical equipment/procedure

  TTCA

  - High performance liquid chromatography-tandem mass spectrometry (LC-MS/MS).

  Creatinine

  - Colorimtetry

5.0 MEDICAL REMOVAL PROTECTION

Indications for removal

- Clinical findings
  - Evidence of health effects that is suspected to be caused by CHTH or may be worsened by CHTH.
- Target organ function abnormality
- Exceed BEL
- Pregnancy and breastfeeding

Temporary MRP

- MRP is based on medical conditions.
  - The determination of the temporary MRP duration should consider the following aspects:
  - expected duration of diagnostic procedures
  - duration of recovery

- MRP based on BEL.
  - The determination of the temporary MRP duration should consider the following aspects:
    >Availability of the repeat BM sample results.

All cases recommended for MRP, suspected cases of poisoning and excessive absorption must be notified to the DG of DOSH.

6.0 RETURN TO WORK

Return to work is based on:

i. BEL

- Return to work when the results are below the BEL:
  - Urinary TTCA level

Categories	Level
Urinary TTCA	<0.5 mg/g creatinine

ii. Medical Condition

- No longer detected as having a medical condition.

iii. Workplace management

- Ensure workplace hygiene is safe and healthy and does not place the employee at increased risk of material impairment to health from exposure to carbon disulphide.

7.0 NOTIFICATION TO DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed in NADOPOD 2004 using the relevant notification forms.

8.0 TREATMENT AND MANAGEMENT

- All cases of acute poisoning must be immediately removed from exposure, provide appropriate first aid measures, and refer for hospital treatment.
- Treatment based on specific medical conditions arising from exposure to the chemical.

9.0 PREVENTIVE MEASURES

- Good workplace and personal hygiene and safety.
- Approved personal protective equipment.

10.0 REFERENCES

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CARBON TETRACHLORIDE

1.0 DESCRIPTION

1.1 SYNONYMS

- Tetrachloromethane, perchloromethane, methane tetrachloride, Nectorina, benzinoform, carbon chloride, Carbona, carbon tet.

1.2 OCCUPATIONAL EXPOSURE LIMITS (OEL)

USECHH 2000	
Carbon tetrachloride	
8-hour time weighted average limit	**15 min – Short-term exposure limit**
5 ppm (31 mg/m3)	10 ppm (63 mg/m^3)

1.3 PHYSICOCHEMICAL PROPERTIES

- Colourless, clear, non-flammable volatile liquid with a sweet odour.

1.4 MATERIAL USE

- Used in the synthesis of chlorinated organic compounds such as chlorofluorocarbon refrigerants and propellants.
- Used as a solvent in the production of semiconductors in the processing of fats, oils, lacquers, varnishes, rubber, waxes, and resins (in degreasing operation) and in laboratory applications.
- Also used as an azeotropic drying agent for spark plugs; a dry-cleaning agent; a fire extinguishing agent; a fumigant, and an anthelmintic agent.

2.0 TOXICITY

2.1 SOURCES OF POTENTIAL OCCUPATIONAL EXPOSURE

- Chemical industry and laboratories

2.2 ROUTE OF EXPOSURE

- Inhalation of vapour (primary)
- Ingestion

- Skin and eye contact
- Dermal absorption

2.3 TOXICOKINETICS

Carbon Tetrachloride	
Absorption	• Carbon tetrachloride is readily absorbed after ingestion, inhalation, and dermal absorption.
Distribution	• Distributed to all organs with concentration primarily in the liver, brain, kidney, muscle, fat, and blood.
Metabolism	• Absorbed carbon tetrachloride is metabolised by cytochrome P-450 enzymes to become highly reactive trichloromethyl and trichloromethylperoxy radicals and decompose into phosgene. • Most of the carbon tetrachloride is eliminated from the body unchanged, but some may change to other chemicals (for example, chloroform, hexachloroethane, and carbon dioxide). Chloroform and hexachloroethane may themselves cause harmful effects. • Alcohol misuse has been found to exaggerate the toxic effects of carbon tetrachloride.
Excretion	• Most of the absorbed carbon tetrachloride will be eliminated through expired air, faeces and to a lesser extent in the urine. • However, the remainder of chemical that is distributed to adipose tissue may take up to a few weeks to fully be eliminated.

2.4 HAZARD CLASSIFICATION

Information on hazard classification for Carbon Tetrachloride is currently unavailable in the ICOP on Chemicals Classification and Hazard Communication (Amendment) 2019.

Classification Code	Hazard Classification	H-Code	Signal
Carc. 2	Carcinogenicity Category 2	H351	Danger
Acute Tox. 3 (inh)	Acute toxicity, category 3 - inhalation	H331	
Acute Tox. 3 (dermal)	Acute toxicity, category 3 - dermal	H311	
Acute Tox. 3 (oral)	Acute toxicity, category 3 - oral	H301	
STOT RE 1	Specific target organ toxicity, repeated exposure category 1	H372**	

Source: The European Union, Commission Regulations (EU) 2018/669.

Note:

Possible risk of gene damage/impaired fertility.

- **Cancer Classification IARC**
  - Group 2B
  - Generally, evidence in human studies is inadequate.

3.0 HEALTH EFFECTS MONITORING

The PEL for carbon tetrachloride is at 5 ppm 8 hours TWA. The ACGIH TLV is at 5 ppm with the basis of liver impairment. The estimated No-observed-effect level (NOEL) is 10 ppm for 3 hours (Hathaway et al, 1991).

Acute exposure to carbon tetrachloride via any route of exposure can cause gastrointestinal and neurological effects in the first 24 hours, such as nausea, vomiting, diarrhoea, headache, dizziness, depression of conscious level and dyspnoea. The liver and kidney are the major target organs for toxicity following acute inhalation or ingestion exposure to carbon tetrachloride [2, 3]. Liver damage can occur after 24 hours and in serious cases this can result in painful swollen liver, haemorrhage, hepatic coma, and death [1, 2]. Kidney damage with an impairment in function normally occurs 2-3 weeks after exposure [2], but in severe cases this can occur within 1-6 days in association with liver failure [1]. Adverse effects on the liver can be markedly increased by the co-ingestion of alcohol [4], due to hepatic enzyme induction which results in increased production of toxic metabolites.

The toxic effects following acute exposure to carbon tetrachloride by inhalation at 10 to 80 ppm showed no adverse effects. Exposure at 100 ppm, after 3-4 hours, caused nausea, gastrointestinal irritation, headache, dizziness and depression and dyspnoea. Exposure >200 ppm caused drowsiness, nausea, vomiting, tachycardia, tachypnoea, liver, and kidney toxicity.

Generally, exposure between 5 ppm and 10 ppm, showed no toxic effects that can be derived from the history of workplace exposures. The earliest symptoms of significant acute exposure are the Nervous system effects, eye irritations and the GIT irritations.

3.1 ACUTE EFFECTS

System/Organ	Acute Effects
Nervous System	• CNS – nausea, dizzy, unconsciousness and coma, optic neuritis, neurosis, Headache, Nervousness. • Skin absorption may cause polyneuritis.
Gastrointestinal	• Nausea • Vomiting • Haematemesis • Abnormal cramps and diarrhoea
Ear, Nose and Throat	• Nose and throat irritation
Eye	• Eye irritation • Burning sensations to eye • Lacrimation
Cardiovascular	• Cardiac muscle depression • Ventricular fibrillation and sudden death
Hepatobiliary	• Liver failure/necrosis – hepatomegaly & jaundice
Renal and Genitourinary	• Destruction of renal tubules with nephritic and nephrotic symptoms - oliguria, proteinuria, haematuria.
Respiratory	• Dyspnoea and cyanosis • Pulmonary oedema
Skin	• Dermatitis

3.2 CHRONIC EFFECTS

System/Organ	Acute Effects
Eyes	● Restriction of visual fields. ● Diminished visual acuity.
Gastrointestinal	● Anorexia ● Nausea ● Vomiting ● Abdominal pain
Hepatobiliary	● Loss of weight ● Jaundice ● Liver toxicity
Musculoskeletal	● Fatigue
Nervous System	● CNS – Headache, dizziness depression, apathy, and mental confusion
Renal and Genitourinary	● Evidence of renal damage ● Kidney toxicity
Skin	● Dermatitis ● Repeated skin contact may cause skin inflammations.

4.0 MEDICAL SURVEILLANCE PROGRAMME

4.1 INDICATIONS

● Any occupational exposure to carbon tetrachloride exceeding 50% of the PEL and/or there is significant risk of absorbing it and/ or evidence of health effects.

4.2 CLINICAL EXAMINATION AND RELEVANT INVESTIGATIONS

● Medical examination with emphasis on:
    ○ Hepatobiliary system: liver necrosis, steatosis, and fibrosis.
    ○ Renal and Genitourinary system.
    ○ Nervous system: central nervous system depression and polyneuritis.
    ○ Look out for signs of skin defatting and rash.
    ○ Look out for eye irritations.

● Lung function test.
● Liver function test (including prothrombin time).
● Renal function test.

4.3 PRE-PLACEMENT MEDICAL EXAMINATION

- Clinical examination and relevant investigation as listed in 4.2.
- Decision for fitness to work.
    - Employees with disease of liver, kidney and central nervous system, employees with history of solvent abuse or who are alcoholics should not work in areas where there is significant carbon tetrachloride exposure.

4.4 PERIODIC MEDICAL EXAMINATION

- Clinical examination and relevant investigation as listed in 4.2.
- Frequency of periodic medical examination: annually

4.5 BIOLOGICAL MONITORING

Determinants	Sampling time	BEL	Notation
Blood carbon tetrachloride	End of shift or after long term exposure	3.5 µg/L biological tolerance value (BAT) by Deutsche Forschungsgemeinschaft (DFG).	-

Source: Deutsche Forschungsgemeinschaft (DFG) MAK and BAT Values

Note:

No ACGIH determinant or BEI value for blood or urine carbon tetrachloride.

- Laboratory Method
    - Sampling procedure

Blood Carbon Tetrachloride	
Container	Gray top tube (Sodium Fluoride/Potassium Oxalate)
Transportation	Specimens should be refrigerated.
Stability	● Specimens are stable at room condition for 5 days. ● Specimens are stable for 10 days when refrigerated. ● Specimens are stable for 14 days when frozen.

Blood Carbon Tetrachloride	
Preservation	Tube should be filled to prevent loss of volatile compounds into headspace. Ensure the container remains tightly sealed.
Volume	Requested volume: 7 ml Minimum volume: 2 ml

○ Analytical equipment/procedure

Carbon tetrachloride

- Headspace Gas chromatograph with electron-capture detection (ECD), or mass-spectrometry detection (MSD).

5.0 MEDICAL REMOVAL PROTECTION

Indications for removal

- Clinical findings
  - ○ Evidence of health effects that is suspected to be caused by CHTH or may be worsened by CHTH.
  - ○ Target organ function abnormalities.
  - ○ Exceed BEL.
  - ○ Pregnancy and breastfeeding.
  - ○ Others:
    - Medical conditions of the liver, kidney and central nervous system, solvent abuse or being alcoholics.

- Temporary MRP
  - ○ MRP is based on medical condition.
    - The determination of the temporary MRP duration should consider the following aspects:
      - ▪ expected duration of diagnostic procedures
      - ▪ duration of recovery.

  - ○ MRP based on BEL.
    - The determination of the temporary MRP duration should consider the following aspects:
      - ▪ Availability of the repeat BM sample results

6.0 RETURN TO WORK

- Return to work based on:

  i. BEL
     - Return to work when the results are below the BEL.

  ii. Medical condition
     - No longer detected of having a medical condition.

  iii. Workplace management
     - Ensure workplace hygiene is safe and healthy and does not place the employee at increased risk of material impairment to health from exposure to carbon tetrachloride.

- The repeat BM sample should be done as soon as possible.

7.0 NOTIFICATION TO DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed in NADOPOD 2004 using the relevant notification forms.

8.0 TREATMENT AND MANAGEMENT

- All cases of acute poisoning must be immediately removed from exposure, provide appropriate first aid measures, and refer for hospital treatment.
- Treatment based on specific medical conditions arising from exposure to the chemical.

9.0 PREVENTIVE MEASURES:

- Good workplace and personal hygiene and safety.
- Approved personal protective equipment.
- Substituting to a less toxic chemical is the best. (Carbon tetrachloride prohibited in cleaning and degreasing purposes).
- Avoid alcohol as alcohol abuse increases risk of toxicity.

10.0 REFERENCES

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CHROMIC ACID

1.0 DESCRIPTION

The long-term health hazards are due to Chromium (VI).

1.1 SYNONYMS

Chromic acid (H2CrO4)

1.2 OCCUPATIONAL EXPOSURE LIMITS (OEL)

USECHH 2000 (Eight-hour time weighted average limit)	
Water soluble Cr VI compound, as NOC	0.05 mg/m³ (TWA)

1.3 PHYSICOCHEMICAL PROPERTIES

- Chromic acid is a dark purplish-red odourless flakes or crystalline powder.

1.4 MATERIAL USE

- Used in chromium plating; medicine; ceramic glazes and paints
- Used in antioxidants, batteries, cement, pigment (yellow), refractories, steel alloys, welding, and wood preservatives.

2.0 TOXICITY

2.1 SOURCES OF POTENTIAL OCCUPATIONAL EXPOSURE

- Manufacturing
- Paint industry

2.2 ROUTE OF EXPOSURE

- Inhalation (primary)
- Ingestion
- Skin and/or eye contact

2.3 TOXICOKINETICS

Chromic acid	
Absorption	• Chromium (VI) is more efficiently absorbed through the skin. • Transfer rates of chromium (VI) across forearm skin in volunteers exposed to sodium chromate (0.01, 0.1 and 0.2 M) were 1, 6 and 10 µg chromium (VI) cm^{-2} h^{-1}.
Distribution	• Once deposited in the lungs, chromium (VI) compounds are generally transferred to the systemic circulation. • Chromium compounds are widely distributed in the body, with a greater distribution reported there is greater tendency of chromium (VI) to cross plasma membranes.
Metabolism	• Chromium (VI) is unstable in the body and is reduced to chromium (V), chromium (IV), and ultimately reduce to chromium (III) by endogenous substances such as ascorbate and glutathione and it is believed that the toxicity of chromium may result from damage to cellular components during this process (e.g., through the generation of free radicals).
Excrete & Half-life	• In humans, absorbed chromium is excreted primarily via urine. • The half-life for elimination of chromium when given as potassium chromate (0.05 mg chromium (VI) kg^{-1} in drinking water) is estima -ed to be approximately 35-40 hours.

2.4 HAZARD CLASSIFICATION

- Chromium (VI) compounds

Classification code	Hazard classification	H-Code	Signal
Carc. 1B	Carcinogenicity category 1B	H350i	Danger
Skin Sens.1	Sensitization, skin category 1	H317	

Source: The European Union, Commission Regulations (EU) 2018/669

- Chromium (VI) trioxide

Classification code	Hazard classification	H-Code	Signal
Carc. 1B	Carcinogenicity category 1B	H350i	Danger
Skin Sens.1	Sensitization, skin category 1	H317	
Muta. 1B	Germ cell mutagenicity category 1B	H340	
Repr. 2	Reproductive toxicity category 2	H361f	
Acute Tox. 2 (inh)	Acute toxicity categor 2 (inhalation)	H330	
Acute Tox. 3 (dermal)	Acute toxicity category 3 (dermal)	H311	
Acute Tox. 3 (oral)	Acute toxicity category 3 (oral)	H301	
STOT RE 1	Specific target organ toxicity – single exposure category 1	H372 (resp. system)	
Skin Corr. 1A	Skin corrosion or irritation category 1A	H314	
Eye Dam. 1	Serious eye damage or eye irritation category 1	H318	
Resp. Sens. 1	Respiratory sensitization category 1	H334	
Skin Sens. 1	Skin sensitization category 1	H317	

Source: ICOP on Chemicals Classification and Hazard Communication (Amendment) 2019.

- Cancer Classification IARC
  - Group 1

3.0 HEALTH EFFECTS MONITORING

The main target organ is the nasopharynx and the respiratory system

3.1 ACUTE EFFECTS

System/Organ	Acute Effects
Ear, Nose & Throat	• Perforation of nasal system • Nasal polyps • Epistaxis • Sinusitis • Laryngitis • Anosmia • May be poisonous. Dust may cause severe irritation to the nose, throat and lungs, causing coughing; shortness of breath. • May be poisonous if swallowed. May cause severe burns of the mouth, throat.
Eyes	• Conjunctivitis • May cause severe irritation, burns, pain, and possible blindness.
Gastrointestinal	• Anorexia • Hypertrophic gastritis • Duodenal ulcer • Colitis • High exposure may cause nausea, salivation, vomiting, cramps, diarrhoea. • Watery or bloody diarrhoea if swallowed. • May cause severe burns to the stomach if swallowed.
Hepatobiliary	• Damage to liver if swallowed
Nervous system - CNS	• May cause flu-like symptoms including chills, muscle ache, headache, fever. • If swallowed, may cause collapse and convulsions.
Renal and Genitourinary	• Acute renal tubular necrosis • Damage to kidneys if swallowed.

Respiratory	• Irritate respiratory tract. • Chest pain • Dyspnoea • High exposure may cause pulmonary edema.
Skin	• Sensitising dermatitis • Chrome hole – skin ulcers • May cause severe irritation and thermal and acid burns, especially if skin is wet.

3.2 CHRONIC EFFECTS

System/Organ	Chronic Effects
Ear, Nose & Throat	• Nasal septum perforation. Usually, symptomless. • Injury to the nasal septum (may cause a hole in the nose). • Discoloration of teeth.
Hepatobiliary	• Liver damage
Renal and Genitourinary	• Kidney damage
Reproductive	• Birth defects • Miscarriage
Respiratory	• Bronchitis • Chemical pneumonitis • Chromatosis (pneumoconiosis) • Bronchogenic carcinoma of lung • Lung cancer • Lung allergy
Skin	• Chrome ulcers – deep ulcers where chromate is deposited on the skin and not washed off. • Skin allergy and ulcers

4.0 MEDICAL SURVEILLANCE PROGRAMME

4.1 INDICATIONS

- Any work where employees are exposed to levels of airborne levels, which are liable to be in excess of half the -in-air standard, and/ or where there is significant risk of ingesting it.

4.2 CLINICAL EXAMINATION AND RELEVANT INVESTIGATIONS

- Clinical examination with emphasis on respiratory system and skin.
- Detecting pre-existing allergies.
- Pulmonary function tests (FEV1, FVC & FEV1/FVC ratio).
- Chest X-ray

4.3 PRE-PLACEMENT MEDICAL EXAMINATION

- Clinical examination and relevant investigations as listed in 4.2.
- Decision for fitness to work:
    - Employees with disease of skin and respiratory system and blood should not work in areas where there is significant chromic acid exposure.

4.4 PERIODIC MEDICAL EXAMINATION

- Clinical examination and relevant investigations as listed in 4.2.
- Frequency of periodic medical examination - annual

4.5 BIOLOGICAL MONITORING

Determinants	Sampling time	BEL	Notation
Total chromium in urine	End of shift at end of workweek	0.7 µg/L	Pop

Notes:

Determinant being tested is total chromium in urine, Total chromium in urine consists of organic chromium (III) and inorganic chromium (VI). Interpretation without an unexposed baseline will be inaccurate in assessing the chromium (VI).

- Laboratory Method
  - Method reference: NMAM 8310
  - Sampling procedure

Total Chromium in Urine	
Container	Polyethylene or plastic container Note: *Request container from the laboratory *Own container can be used (provide at least one unit container to the lab for blank test)
Transportation	Specimens should be refrigerated
Stability	• Urine specimens are stable at room temperature for 5 days. • Urine specimens are stable for 1 month when refrigerated and frozen.
Preservation	No specific preservative mentioned
Sample volume	Requested volume: 30 mL Minimum volume: 10 mL

  - Analytical equtipment/procedure

    Chromium:
    - Atomic absorption spectrometry.
    - Inductively Coupled Plasma Mass Spectrometry (ICP-MS).

5.0 MEDICAL REMOVAL PROTECTION

- Indications for removal:
  - Clinical findings
    - Evidence of health effects that is suspected to be caused by CHTH or may be worsened by CHTH.
  - Target organ function abnormalities
  - BEL
    - Exceed BEL
  - Pregnancy and breastfeeding.
  - Others
    - Medical conditions of the skin and respiratory system and the blood.

- Temporary MRP
  - MRP based on medical condition.
    - The determination of the temporary MRP duration should consider the following aspects:
      - expected duration of diagnostic procedures
      - duration of recovery.
  - MRP based on BEL.
    - The determination of the temporary MRP duration should consider the following aspects:
      - Availability of the repeat BM sample results.

- All cases recommended for MRP, suspected cases of poisoning and excessive absorption must be notified to the DG of DOSH.

6.0 RETURN TO WORK

i. BEL
   - Return to work when the results are below the BEL.

ii. Medical condition
   - No longer detected of having a medical condition and no possibility of worsening when further exposed.

iii. Workplace management
   - Ensure workplace hygiene is safe and healthy and does not place the employee at increased risk of material impairment to health from exposure to chromic acid.

- The repeat BM sample should be done as soon as possible.

7.0 NOTIFICATIONS TO DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed in NADOPOD 2004 using the relevant notification forms. must be notified to the DG of DOSH.

8.0 TREATMENT AND MANAGEMENT

- First Aid: All cases of poisoning must be immediately removed from exposure and must be referred for hospital treatment.
- Irrigate eyes with water.
- Wash contaminated areas of the body with soap and water.
- Dermatitis: Antihistamines, cortisone locally.
- Skin ulcers: Apply 10% edathamil calcium disodium in lanolin base to ulcer, bandage for 24 hours, curette base and repeated as necessary. Edathamil calcium disodium has been suggested. Symptomatic and supportive.

9.0 PREVENTIVE MEASURES

- Adequate ventilation and regular monitoring of the work environment.
- Use of mechanical filter respirator, chemical goggles, rubber gloves, aprons, boots.
- No eating or smoking in the work area.
- Apply vaseline or paraffin to the nose before going to work.
- Appropriate signage.

10.0 REFERENCES

ACGIH. 2022. TLVs® and BEIs® Threshold Limit Values for Chemical Substances and Physical Agents & Biological Exposure Indices. Ohio: American Conference of Governmental Industrial Hygienists.

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DIANISIDINE

1.0 DESCRIPTION

1.1 SYNONYMS

Dianisidine, 3,3'-Dianisidine, 3,3'-Dimethoxybenzidine.

1.2 OCCUPATIONAL EXPOSURE LIMIT (OEL)

USECHH 2000 (Eight-hour time weighted average limit)	
Dianisidine	Not listed

Note:

- Not listed in USECHH 2000 or ACGIH 2022.
- No numerical OELs have been established.
- No safe level of exposure for potential carcinogen.

1.3 PHYSICOCHEMICAL PROPERTIES

- Colourless crystal but change to purple colour when standing in room conditions.

1.4 MATERIAL USE

- Used as chemical intermediate in dyes and pigments production.

2.0 TOXICITY

2.1 SOURCES OF POTENTIAL OCCUPATIONAL EXPOSURE

- Dye manufacturing and processing plants.

2.2 ROUTE OF EXPOSURE

- Ingestion (primary).
- Inhalation.
- Skin and eye absorption.

2.3 TOXICOKINETICS

- **There is a lack of information on the toxicokinetic profile of dianisidine.**

Absorption and Distribution	Dianisidine is readily absorbed through skin, lung and intestine.
Metabolism	Most of them will be metabolized with only a small quantity remaining unchanged
Excretion	Unchanged dianisidine is eliminated through faeces. Limited studies on the estimated half life of dianisidine.

2.4 HAZARD CLASSIFICATION

Classification code	Hazard classification	H-Code
Carc. 1B	May cause cancer [Danger Carcinogenicity]	H350
Acute Tox. 4*	Harmful if swallowed [Warning Acute toxicity, oral]	H302

Source: European Union, Commission Regulations (EC) 1272/2008.

- **Cancer Classification IARC**
  - Group 2B
  - Possibly carcinogenic to humans (inadequate evidence in human).

3.0 HEALTH EFFECTS MONITORING

3.1 ACUTE EFFECTS

- Limited information on the toxicity to humans.
- Exposure to dianisidine may cause skin, eye, and respiratory tract irritation.
- May cause methemoglobinemia.

3.2 CHRONIC EFFECTS

- No information is available on the chronic and carcinogenic effects in humans.
- Prolonged skin contact may cause skin rashes, redness, and itching.
- Associated with bladder cancer but inadequate evidence in humans.
- No information is available on the reproductive toxicity and mutagenicity.

4.0 MEDICAL SURVEILLANCE PROGRAMME

4.1 INDICATIONS

Any occupation where employees are liable to significant airborne exposures and/or possibility of skin absorption.

4.2 CLINICAL EXAMINATION AND RELEVANT INVESTIGATION

- Clinical examination with emphasis on the renal and haematological system
- Urine cytology
- Renal function test
- Examination of the breast, skin, respiratory, gastrointestinal, urinary, and reproductive system.

4.3 PRE-PLACEMENT MEDICAL EXAMINATION

- Clinical examination and relevant investigation as listed in 4.2.

4.4 PERIODIC MEDICAL EXAMINATION

- Clinical examination and relevant investigation as listed in 4.2.
- Frequency of periodic medical examination - annual

4.5 BIOLOGICAL MONITORING

Determinants	Sampling time	BEL/Reference
Blood methaemoglobin	During of end of shift	1.5% of total haemoglobin.

Note:
Methaemoglobin level may not be specific for dianisidine biological monitoring.

5.0 MEDICAL REMOVAL PROTECTION

- Indications for removal
  - Clinical findings
    - Evidence of health effects that is suspected to be caused by CHTH or may be worsened by CHTH.
  - Target organ function abnormalities.
  - BEL
    - Exceed BEL

- Temporary MRP
  - MRP based on medical condition.
    - The determination of the temporary MRP duration should consider the following aspects:
      - expected duration of diagnostic procedures.
      - duration of recovery.
  - MRP based on BEL.
    - The determination of the temporary MRP duration should consider the following aspects:
      - Availability of the repeat BM sample results.

- The repeat BM sample should be done as soon as possible.
- All cases recommended for MRP, suspected cases of poisoning and excessive absorption must be notified to the DG of DOSH.
- All employees undergoing MRP must have repeat urine and blood biochemical test at monthly interval.

6.0 RETURN TO WORK

i. BEL
  - Return to work when the results are below the BEL.

ii. Medical condition
  - No longer detected of having a medical condition and no possibility of worsening when further exposed.

iii. Workplace management
  - Ensure workplace hygiene is safe and healthy and does not place the employee at increased risk of material impairment to health from exposure to dianisidine.

7.0 NOTIFICATION TO DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed in NADOPOD 2004 using the relevant notification forms.

8.0 TREATMENT AND MANAGEMENT

- All cases of acute poisoning must be immediately removed from exposure, provide appropriate first aid measures, and refer for hospital treatment.
- Treatment based on specific medical conditions arising from exposure to the chemical.

9.0 PREVENTIVE MEASURES

- Good workplace and personal hygiene and safety.
- Approved personal protective equipment.

10 REFERENCES

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DISULPHUR DICHLORIDE

1.0 DESCRIPTION

Disulfur dichloride is the inorganic compound of sulfur and chlorine with the formula S_2Cl_2.

1.1 SYNONYMS

Sulphur chloride, sulfur monochloride, chlorosulfane, sulfur monochloride (di-); sulfur subchloride & Thiosulfurous dichloride.

1.2 OCCUPATIONAL EXPOSURE LIMIT (OEL)

USECHH 2000 (Eight-hour time weighted average limit)	
Disulphur Dichloride	Not listed

Note:

- Not listed in USECHH 2000 or ACGIH 2022.
- No numerical OELs have been established.
- No safe level of exposure for potential carcinogen.

1.3 PHYSICOCHEMICAL PROPERTIES

- Highly corrosive fuming and oily liquid.
- Light amber to yellowish red colour with strong, nauseating, and irritating odour.

1.4 MATERIAL USE

- Used as intermediate and chlorinating agent in organic chemicals, sulphur dyes, insecticides, and synthetic rubber production.
- Disulfur dichloride is a chemical compound. It has been used to introduce C-S bonds. It is also used to prepare the sulfur mustard "gas" by reaction with ethylene.
- Used in gold extraction.
- Used as military poisons.

2.0 TOXICITY

2.1 SOURCES OF POTENTIAL OCCUPATIONAL EXPOSURE

- Chemical industry (usage in chlorination, catalyzation and manufacture).
- Food processing factory (purification of sugar juice & vegetable oil processing).
- Synthetic rubber plants (vulcanization of rubber).

2.2 ROUTE OF EXPOSURE

- Inhalation of fumes (primary).
- Skin/eye contact (primary).
- Ingestion.

2.3 TOXICOKINETICS

Limited information on the toxicokinetics of disulphur dichloride available.

2.4 HAZARD CLASSIFICATION

Classification code	Hazard classification	H-Code	Signal
Acute Tox. 3 (oral)	Acute toxicity category 3 - oral	H301	Danger
Acute Tox. 4 (inh)	Acute toxicity category 4 - inhalation	H332	
Skin Corr. 1A	Skin corrosion category 1A	H314	
Eye Dam. 1	Serious eye damage category 1	H318	

Source: ICOP on Chemicals Classification and Hazard Communication (Amendment) 2019.

- Cancer Classification IARC
  - Not listed

3.0 HEALTH EFFECTS MONITORING

3.1 ACUTE EFFECTS

System/Organ	Acute Effects
Eyes	• Fumes can cause severe irritation to eyes. • Contact with disulphur dichloride can cause permanent eye damage.
Nervous System - CNS	• Exposure can cause headache, nausea, and dizziness.
Respiratory	• Fumes can cause severe irritation to the mucous membrane of the upper respiratory tract. • Higher exposures can cause pulmonary edema and shortness of breath which may lead to death.
Skin	• Fumes can cause severe irritation to skin. • Contact with disulphur dichloride can cause severe irritation and burns.

3.2 CHRONIC EFFECTS

System/Organ	Acute Effects
Renal and Genitourinary	• Bladder tumours.
Respiratory	• May cause lung damage; bronchitis may develop.
Skin	• Repeated exposure can cause drying and cracking of the skin.

4.0 MEDICAL SURVEILLANCE PROGRAMME

4.1 INDICATIONS

- Any occupation where employees are liable to be exposed to disulphur dichloride above 50% PEL.

4.2 CLINICAL EXAMINATION AND RELEVANT INVESTIGATIONS

- Clinical examination with emphasis on skin, eyes, and respiratory system.
- Pulmonary function tests.

4.3 PRE-PLACEMENT MEDICAL EXAMINATION

- Clinical Examination and Relevant Investigations as listed in 4.2.

4.4 PERIODIC MEDICAL EXAMINATION:

- Clinical Examination and Relevant Investigations as listed in 4.2.
- Frequency of periodic medical examination – annually but more frequent if high

4.5 BIOLOGICAL MONITORING

No biological exposure indices determinant based on ACGIH 2022 TLVs and BEIs.

5.0 MEDICAL REMOVAL PROTECTION

- Indications for removal
  - Clinical findings
    - Evidence of health effects that is suspected to be caused by CHTH or may be worsened by CHTH.
  - Target organ function abnormalities.

- Temporary MRP
  - MRP based on medical condition.
    - The determination of the temporary MRP duration should consider the following aspects:
      - expected duration of diagnostic procedures.
      - duration of recovery.

- All cases recommended for MRP, suspected cases of poisoning and excessive absorption must be notified to the DG of DOSH.

6.0 RETURN TO WORK

i. Medical condition
   - No longer detected of having a medical condition and no possibility of worsening when further exposed.

ii. Workplace management
   - Ensure workplace hygiene is safe and healthy and does not place the employee at increased risk of material impairment to health from exposure to disulphur dichloride.

7.0 NOTIFICATION TO DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed in NADOPOD 2004 using the relevant notification forms.

8.0 TREATMENT AND MANAGEMENT

- All cases of acute poisoning must be immediately removed from exposure, provide appropri ate first aid measures, and refer for hospital treatment.
- Treatment based on specific medical conditions arising from exposure to the chemical.

9.0 PREVENTIVE MEASURES

- Contact lenses should not be worn when working with this chemical.
- Good workplace and personal hygiene and safety.
- Approved personal protective equipment.

10.0 REFERENCES

Department of Occupational Safety and Health. 2001. Guidelines on Medical Surveillance. Putrajaya: Department Occupational Safety and Health Malaysia.

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UKM Pakarunding. 2014. DOSH Chemical Review 2014. Volume II.

N-HEXANE

1.0 DESCRIPTION

n-Hexane is an organic solvent normally used in laboratories. Hexane is a mixture containing n-Hexane and other alkanes mixtures and used as solvents in the industries.

1.1 SYNONYMS

Hexane, Exxsol hexane, Genesolv 404 azeotrope, Gettysolve-B, Hexyl hydride, Skellysolve B.

1.2 OCCUPATIONAL EXPOSURE LIMITS (OEL)

Basis of the PEL is central nervous system (CNS) impairment, peripheral neuropathy, and eye irritation.

USECHH 2000 (Eight-hour time weighted average limit)	
n-Hexane	50 ppm 176 mg/m³

1.3 PHYSICOCHEMICAL PROPERTIES

- Clear colorless volatile liquid with gasoline-like odor.

1.4 MATERIAL USE

- Used as emulsifier, solvent, laboratory reagents and chemical intermediate for plastics and resins production.

2.0 TOXICITY

2.1 SOURCES OF POTENTIAL OCCUPATIONAL EXPOSURE

- Rubber and synthetic plastic industry.
- Refinery industry.
- Construction and footwear assembly factory (usage as solvent).
- Chemical laboratories.
- In the palm oil mill refineries, it is used in the laboratories for oil extraction analysis.

2.2 ROUTE OF EXPOSURE

- Inhalation (primary).
- Skin absorption.
- Ingestion.

2.3 TOXICOKINETICS

n-Hexane	
Absorption	• Following inhalation exposure, n-Hexane is absorbed into the circulation and transported to the liver.
Distribution	• In the liver, n-Hexane is metabolized and distributed in the blood to various organs and tissues, including the liver, kidney, and brain.
Metabolism	• n-Hexane is metabolized to various metabolites (2,5-hexanedione & 4,5-dihydroxy-2-hexanone).
Excretion	• n-Hexane and its metabolites (2,5-hexanedione & 4,5-dihydroxy-2-hexa - none) are excreted in the urine. • The average half life of n-Hexane in the blood is estimated to be about 1.5 to 2 hours. (EPA., 2005).

2.4 HAZARD CLASSIFICATION

Classification code	Hazard classification	H-Code	Signal
Flam. Liq. 2	Flammable liquids category 2	H350i	Danger
Repr. 2	Reproductive toxicity category 2	H317	
Asp. Haz.	Aspiration hazard category 1	H340	
STOT RE 2	Specific target organ toxicity, repeated exposure category 2	H361f	
Skin Irrit. 2	Skin irritation category 2	H330	
STOT SE 3	Specific target organ toxicity, single exposure; Narcotic effects category 3	H311	

*(a) State the target organ.
Source: ICOP on Chemicals Classification and Hazard Communication (Amendment) 2019.

- Cancer Classification IARC
  - Not listed

3.0 HEALTH EFFECTS MONITORING

The primary target organs are the central and peripheral nervous system. It may cause organic solvent neurotoxicity. The peripheral neurotoxicity occurs at the level of exposure several times of the PEL and may cause paralysis of the lower limbs.

3.1 ACUTE EFFECTS

System/Organ	Acute Effects
Ear, Nose and Throat	Inhaled hexane **irritates the nose.**
Eyes	• Inhaled hexane **irritates the eyes.** • Conjunctivitis
Gastrointestinal	Nausea
Nervous System	CNS: • Dizziness • Narcosis • Ataxia • High level of exposure regardless of route causing neurological symptoms such as **lightheadedness, giddiness, headaches, incoordination, euphoria, and nausea.** PNS: • High level of exposure regardless of route can affects nervous system and cause **peripheral neuropathy**
Respiratory	• Inhaled hexane **irritates the respiratory tract.** • Aspiration into the lungs causes pneumonitis, chemical-induced pneumonia, and pulmonary edema. • At very high concentration, n-hexane **acts as asphyxiant** and displaces oxygen from the breathable air. Extreme high levels can lead to **unconsciousness** and **death.**
Skin	• Skin contact may cause **irritation, redness, swelling, blisters**, and **pain.** • Defatting dermatitis
Others	Anorexia

3.2 CHRONIC EFFECTS

System/Organ	Chronic Effects
Nervous System	CNS: ● Weakness ● Loss of sensation at extremities ● High or repeated exposure can damage the nervous system **(neuropathy)**, causing **numbness, tingling, and/or muscle weakness in the hands, feet, arms, and legs.**
Skin	● Repeated skin contact can cause **irritation, dryness, and cracking**, and can lead to **rash.**
Others	● Limited information on teratogenic and carcinogenic effects of n-Hexane ● Testicular and spermatotoxic effects were found in some animal studies with continuous exposure and very high dosages which were simultaneously neurotoxic. ● No damage to developing embryos or fetus was found. ● n-Hexane did not show genotoxic effects in in-vitro and in-vivo studies.

4.0 MEDICAL SURVEILLANCE PROGRAMME

4.1 INDICATIONS

- Any occupation where employees are liable to be exposed to n-hexane above 50% of PEL and/or possibility of routine skin absorption.

4.2 CLINICAL EXAMINATION AND RELEVANT INVESTIGATIONS

- A complete history and physical examination. The purpose is to detect pre-existing conditions that might place the exposed employee at increased risk, and to establish a baseline for future health monitoring.

- Clinical examination and baseline with particular attention to:
    - Skin: n-Hexane is a defatting agent and can cause dermatitis on prolonged exposure. Persons with pre-existing skin disorders may be more susceptible to the effect of this agent.
    - Respiratory system: In person with impaired pulmonary function, especially those with obstructive airway diseases, the breathing of n-hexane might cause exacerbation of symptoms due to its irritant properties.
    - Neurological system. It affects both the central nervous system and the peripheral nervous system. Use of the Swedish Q16 questionnaire is recommended to detect early changes of the CNS. Early changes can be easily missed without the use of specific questionnaires such as the Q16 Swedish Questionnaire.
    - Kidneys: The importance of this organ in the elimination of toxic substances justifies special consideration in those with impaired renal function.
    - Liver: The importance of this organ in the biotransformation and detoxification of foreign substances should be considered before exposing a person with impaired liver function.

- **Tests**
    - Urinary 2,5-hexanedione as biological exposure determinant. Samples should be taken end of shift.
    - Pre-placement spirometry.
    - Pre-placement liver function test.
    - Pre-placement renal function test.

4.3 PRE-PLACEMENT MEDICAL EXAMINATION

- Clinical Examination and Relevant Investigations as listed in 4.2.

- Decision for fitness to work and frequency of periodic medical examination:
  - Employees with disease of skin, respiratory system and nervous system should not work in areas where there is significant n-hexane.

4.4 PERIODIC MEDICAL EXAMINATION

- Clinical Examination and Relevant Investigations as listed in 4.2.
- Spirometry, LFT and RFT, may be repeated when clinically indicated.
- BM: Urine hexanedione.
- Frequency of periodic medical examination - annually. The frequency should be shortened when abnormality detected.

4.5 BIOLOGICAL MONITORING

Determinants	Sampling time	BEL	Notation
2,5-hexanedione in urine	End of shift	0.5 mg/L	-

Note:
ACGIH suggested monitoring free urinary 2,5-hexanedione (without hydrolysis) instead of total 2,5-hexanedione (acid hydrolysis). However, urine 2,5-hexanedione is not specific to n-Hexane exposure. It is also a metabolite of Hexanone.

- Laboratory Method
  - Sampling procedure

2,5-Hexanedione in Urine	
Container	Plastic urine container (free of preservative) Note: *Request container from the laboratory *Own container can be used (provide at least one unit container to the lab for blank tests).
Transportation	Specimens should be refrigerated.
Stability	Urine specimens are stable for 30 days when frozen.
Preservation	No specific preservative is mentioned.
Sample volume	Requested volume: 30 mL Minimum volume: 10 mL

- Analytical equipment/procedure

  2,5-hexanedione
  - Gas chromatography with mass spectrometry (GC-MS) or flame ionization detector (GC-FID).

5.0 MEDICAL REMOVAL PROTECTION

- **Indications for removal**

  - Clinical findings
    - Evidence of health effects that is suspected to be caused by CHTH or may be worsened by CHTH.
  - Target organ function abnormalities.
  - BEL
    - Exceed BEL
  - Pregnancy and breastfeeding.
  - Others:
    - Medical conditions of the skin, respiratory system, and nervous system.

- All cases recommended for MRP, suspected cases of poisoning and excessive absorption must be notified to the DG of DOSH.

6.0 RETURN TO WORK

- Duration of temporary MRP based on BM, is the duration of the repeat BM sample to be available. Return to work when the results are below the BEL.

- Central nervous system neurotoxicity requires 3 monthly follow up until conditions are normalized.

- Permanent or severe medical conditions should be permanently removed.

- Workplace management:

  - Ensure workplace hygiene is safe and healthy and does not place the employee at increased risk of material impairment to health from exposure to n-Hexane.

7.0 NOTIFICATION TO DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed in NADOPOD 2004 using the relevant notification forms.

8.0 TREATMENT AND MANAGEMENT

- Irrigate eyes with water.
- Wash contaminated areas of the body with soap and water.
- Symptomatic and supportive.
- Appropriate signage.

9.0 PREVENTIVE MEASURES

- Good workplace and personal hygiene and safety.
- Approved personal protective equipment.

10.0 REFERENCES

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HEXAVALENT CHROMIUM COMPOUND

1.0 DESCRIPTION

Hexavalent chromium (chromium(VI), Cr(VI), chromium 6) is chromium in any chemical compound that contains the element in the +6 oxidation state (thus hexavalent). Virtually all chromium ore is processed via hexavalent chromium, specifically the salt sodium dichromate. Hexavalent chromium is key to all materials made from chromium.

Chromium can exist in several different forms (valencies) – the metal, chromium (III) compounds and chromium (VI) compounds. Chromium (III) exists naturally, the other twotypes are produced by industrial processes. Chromium (III) and chromium (VI) are also known
as trivalent chromium and hexavalent chromium respectively.

1.1 SYNONYMS

- Chromium (VI)

1.2 OCCUPATIONAL EXPOSURE LIMITS (OEL)

USECHH 2000 (Eight-hour time weighted average limit)	
Chromium and inorganic compounds, as Cr	0.5 mg/m³
Water soluble Cr VI compound, as NOC	0.05 mg/m³

1.3 PHYSICOCHEMICAL PROPERTIES

- Second most stable oxidation state of chromium.
- Rarely occurring naturally, most chromium (VI) compounds are manufactured (products or by-products).
- Chromium (VI) compounds are typically classified as water soluble (e.g., sodium chromate and potassium chromate) or water insoluble (e.g., barium chromate and lead chromate).
- Mostly chromium (VI) compounds are lemon-yellow to orange to dark red solids (except chromyl chloride in dark red liquid form).

1.4 MATERIAL USE

- Added to alloy steel to increase hardenability and corrosion resistance.
- Used as pigments in dyes, paints, inks, and plastics.
- Used as an anti-corrosive agent added to paints, primers, and other surface coatings.
- Used to electroplate chromium onto metal parts to provide a decorative or protective coating.

- Galvanizing and electroplating
- By-product of welding – airborne contaminants (including fumes and gases) from welding and flame cutting arise from a variety of sources: the metal in the filler rod or constituents of various types of steel (chromium).

2.0 TOXICITY

2.1 SOURCES OF POTENTIAL OCCUPATIONAL EXPOSURE

- Welding and other types of “hot work” on stainless steel and other alloy steels containing chromium metal.
- Operating chrome plating baths.
- Use of pigments, spray paints and coatings.

2.2 ROUTE OF EXPOSURE

- Inhalation (primary)
- Ingestion
- Skin/eye contact

2.3 TOXICOKINETICS

Hexavalent Chromium Compound	
Absorption	• Chromium (VI) is more efficiently absorbed through the skin. • Although the extent of uptake is difficult to quantify, absorption by inhalation exposure appears to occur rapidly for water-soluble chromium (VI) compounds. • Transfer rates of chromium (VI) across forearm skin in volunteers exposed to sodium chromate (0.01, 0.1 and 0.2 M) were 1, 6 and 10 µg chromium (VI) cm^{-2} h^{-1}.
Distribution	• Once deposited in the lungs, chromium (VI) compounds are generally transferred to the systemic circulation. • Chromium compounds are widely distributed in the body, with a greater distribution reported there is greater tendency of chromium (VI) to cross plasma membranes. • Bone is also a site of distribution, which may contribute to the long-term retention kinetics of chromium. • Absorbed chromium can be transferred to fetuses through the placenta and to infants via breast milk.
Metabolism	• Chromium (VI) is unstable in the body and is reduced to chromium (V), chromium (IV), and ultimately reduce to

	chromium (III) by endogenous substances such as ascorbate and glutathione and it is believed that the toxicity of chromium may result from damage to cellular components during this process (e.g., through the generation of free radicals).
Excretion & Half-life	• In humans, absorbed chromium is excreted primarily via urine. • The half-life for elimination of chromium when given as potassium chromate (0.05 mg chromium (VI) kg^{-1} in drinking water) is estimated to be approximately 35-40 hours. • Chromium can also be eliminated in hair, nails, and breast milk.

2.4 HAZARD CLASSIFICATION

- Chromium (VI) compounds

Classification Code	Hazard classification	H-Code	Signal
Carc. 1B	Carcinogenicity category 1B	H350i	Danger
Skin Sens. 1	Sensitization, skin category 1	H317	

Source: The European Union, Commission Regulations (EU) 2018/669.

- Chromium (VI) trioxide

Classification Code	Hazard classification	H-Code	Signal
Ox. Sol. 1	Oxidizing solids category 1	H271	Danger
Carc. 1A	Carcinogenicity category 1A	H350	
Muta. 1B	Germ cell mutagenicity category 1B	H340	
Repr. 2	Reproductive toxicity category 2	H361f	
Acute Tox. 2 (inh)	Acute toxicity category 2 (inhalation)	H330	
Acute Tox. 3 (dermal)	Acute toxicity category 3 (dermal)	H311	
Acute Tox. 3 (oral)	Acute toxicity category 3 (oral)	H301	
STOT RE 1	Specific target organ toxicity – single exposure category 1	H372 (resp. system)	
Skin Corr. 1A	Skin corrosion or irritation category 1A	H314	
Eye Dam. 1	Serious eye damage or eye irritation category 1	H318	
Resp. Sens. 1	Respiratory sensitization category 1	H334	
Skin Sens. 1	Skin sensitization category 1	H317	

Source: ICOP on Chemicals Classification and Hazard Communication (Amendment) 2019.

- **Cancer Classification IARC**
  - Group 1

3.0 HEALTH EFFECTS MONITORING

3.1 ACUTE EFFECTS

System/Organ	Acute Effects
Cardiovascular	• Large amounts of exposure can lead to severe cardiovascular damage and potentially death.
Gastrointestinal	• Large amounts of exposure can lead to severe gastrointestinal damage and potentially death.
Hepatobiliary	• Large amounts of exposure can lead to severe hepatic damage and potentially death.
Renal and Genitourinary	• Large amounts of exposure can lead to severe renal damage and potentially death.
Respiratory	• May cause occupational asthma in sensitized individuals. • Large amounts of exposure can lead to severe respiratory damage and potentially death.
Skin	• Dermal ulcers • Dermatitis

3.2 CHRONIC EFFECTS

System/Organ	Chronic Effects
Ear, Nose and Throat	• Occupational exposure to some inhaled mists may cause **nasal septal ulceration and perforation.** • Occupational exposure **increased the risk of rare sinonasal cancer.**
Gastrointestinal	• Occupational exposure to some inhaled mists may cause **gastrointestinal effects.**
Haematological	• Occupational exposure to some inhaled mists may cause **haematological effects.**
Hepatobiliary	• Occupational exposure to some inhaled mists may cause **hepatic effects.**
Renal and Genitourinary	• Occupational exposure to some inhaled mists may cause **renal effects.** • There is some limited evidence to suggest that chromium (VI) compounds **may be toxic to the male reproductive system.**

Respiratory	• Occupational exposure to some inhaled mists may cause **respiratory irritation and inflammation, dyspnoea,** and **cyanosis.** • Occupational exposure to hexavalent chromium **increased the risk of lung cancers.**
Skin	• Dermal contact in chromium-sensitised individuals can lead to **allergic dermatitis** and chronic dermal exposure can result in **deeply penetrating skin ulcers** if left untreated.
Others	• Chromium (VI) compounds have mutagenic potential and are carcinogenic to humans.

4.0 MEDICAL SURVEILLANCE PROGRAMME

4.1 INDICATIONS

- Employers must provide medical surveillance for the employees who are:
  - Exposed or may be exposed to Chromium (VI) at concentrations at or above 50% of the PEL and/or exceeds the MEL.
  - Significant skin exposures.
  - Experiencing signs and symptoms of adverse health effects associated with Chromium (VI) exposures (e.g., blistering lesions, redness or itchiness of exposed skin, shortness of breath or wheezing that worsens at work, nosebleeds, a whistling sound while inhaling or exhaling).
  - Exposed in an emergency situation.

4.2 CLINICAL EXAMINATION AND RELEVANT INVESTIGATIONS

- Clinical examination with emphasis on respiratory system and skin.
- Detect pre-existing allergies.
- Urinary chromium estimation.
- A work history to determine past exposure to hexavalent chromium compounds
- Smoking history.
- History of skin or pulmonary sensitization to chromium.
- History or presence of dermatitis, skin ulcers or lesions of the nasal mucosa and/or perforation of the septum.
- Spirometry.
- Chest X-ray.

4.3 PRE-PLACEMENT MEDICAL EXAMINATION

- Clinical Examination and Relevant Investigations as listed in 4.2.
- Decision for fitness to work and frequency of periodic medical examination:
  - Employees with disease of skin and respiratory system, should not work in areas where there is significant hexavalent chromium compound exposure.

4.4 PERIODIC MEDICAL EXAMINATION

- Clinical Examination and Relevant Investigations as listed in 4.2.
- Frequency of periodic medical examination - annually.

4.5 BIOLOGICAL MONITORING

Determinants	Sampling time	BEL	Notation
Total chromium in urine	End of shift at the end of workweek	0.7 µg/L	Pop

Note:

Determinant being tested is total chromium in urine. Total chromium in urine consists of organic chromium (III) and inorganic chromium (VI). Interpretation without an unexposed baseline will be inaccurate in assessing the chromium (VI).

- **Laboratory Method**
  - Method reference: NMAM 8310
  - Sampling procedure

Total Chromium in Urine	
Container	Polyethylene or plastic container. Note: *Request container from the laboratory. *Own container can be used (provide at least one unit container to the lab for blank tests).
Transportation	Specimens should be refrigerated.
Stability	• Urine specimens are stable at room temperature for 5 days. • Urine specimens are stable for 1 month when refrigerated and frozen.
Preservation	No specific preservative mentioned.
Sample volume	Requested volume: 30 mL Minimum volume: 10 mL

  - Analytical equipment/procedure
    Chromium
    - Atomic absorption spectrometry (AAS).
    - Inductively coupled plasma mass spectrometer (ICP-MS).

5.0 MEDICAL REMOVAL PROTECTION

- **Indications for removal**
  - Clinical findings
    - Evidence of health effects that is suspected to be caused by CHTH or may be worsened by CHTH.
  - Target organ function abnormalities.
  - BEL
    - Exceed BEL
  - Pregnancy and breastfeeding.
  - Others:
    - Medical conditions of the skin and respiratory system.
- All cases recommended for MRP, suspected cases of poisoning and excessive absorption must be notified to the DG of DOSH.

6.0 RETURN TO WORK

- Duration of the temporary MRP, based on the medical conditions, considering the expected duration of diagnostic procedures and the duration of recovery. Return to work when there are NO permanent medical conditions or NO possibility of worsening when further exposed.
- Duration of temporary MRP based on BM, is the duration of the repeat BM sample to be available. Return to work when the results are below the BEL.
- Workplace management:
  - Ensure workplace hygiene is safe and healthy and does not place the employee at increased risk of material impairment to health from exposure to hexavalent chromium compound [Cr(VI)].

7.0. NOTIFICATIONS TO DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed in NADOPOD 2004 using the relevant notification forms.

8.0 TREATMENT AND MANAGEMENT

- All cases of acute poisoning must be immediately removed from exposure, provide appropriate first aid measures, and refer for hospital treatment.
- Treatment based on specific medical conditions arising from exposure to the chemical.

9.0 PREVENTIVE MEASURES

- Good workplace and personal hygiene and safety.
- Approved personal protective equipment.

10.0 REFERENCES

ACGIH. 2022. TLVs® and BEIs® Threshold Limit Values for Chemical Substances and Physical Agents & Biological Exposure Indices. Ohio: American Conference of Governmental Industrial Hygienists.

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ISOCYANATES

1.0 DESCRIPTION

- This refers to a group of chemicals grouped as Isocyanates. Isocyanates are a family of highly reactive organic compounds that contain the isocyanate functional group of the formula R-N=C=O. Isocyanates include isocyanates and poly-isocyanates, which contain two or more isocyanate functional groups. This document contains generalised information which is adequate to perform medical surveillance for any of the chemicals in the family.

- Isocyanates are potent sensitizers and remain one of the most commonly reported causes of occupational asthma worldwide. The reported prevalence of isocyanate asthma in exposed employees is highly variable, ranging from less than 1% to over 30% in end-user settings such as spray applications or foam production.

- The OHD should also be aware of idiosyncratic response from the exposure to isocyanate, where some employees are not affected, while some can be affected in a short period of time.

1.1 SYNONYMS

4,4' Diphenylmethane diisocyanate (MDI); Hexamethylene diisocyanate (HDI); Methyl isocyanate (MIC); Methylene diisocyanate (MDI); I,5' Napthalene diisocyanate (NDI); Toluene diisocyanate (TDI) and many others.

1.2 OCCUPATIONAL EXPOSURE LIMITS (OEL)

USECHH 2000 (Eight-hour time weighted average limit)	
Methyl isocyanate	0.02 ppm 0.047 mg/m^3
Methylene bisphenyl isocyanate (MDI)	0.005 ppm 0.051 mg/m^3
Toluene-2, 4-diisocyanate (TDI)	0.005 ppm 0.04 mg/m
Hexamethylene diisocyanate (HDI)	0.005 ppm 0.034 mg/m^3
USECHH 2000 (15 min - Short term exposure limit)	
Methyl isocyanate	0.06 ppm 0.14 mg/m^3
Toluene-2, 4-diisocyanate (TDI)	0.02 ppm 0.1 mg/m^3

1.3 PHYSICOCHEMICAL PROPERTIES

- Methyl isocyanate is a volatile colourless liquid with pungent odour.
- Methylene bisphenyl isocyanate (MDI) exists as crystals or flakes with white to pale yellow colour. Toluene-2, 4-diisocyanate (TDI) may exist as colourless to pale-yellow solid or liquid above 71 degrees F and has a sharp, pungent odour. They are reactive with alcohol to produce polyurethan polymers.
- Hexamethylene diisocyanate (HDI) is a clear, colourless to slightly yellow liquid which gives off a pungent odour.

2.0 TOXICITY

2.1 SOURCE OF POTENTIAL OCCUPATIONAL EXPOSURE

- Spray painters using two-pack polyurethane paints are the group at highest risk of exposure to isocyanates. The repair and refinishing of cars entails the sprayed-on application of isocyanate-containing coatings on almost every vehicle.
- The largest volume use of isocyanates is in the production of polyurethane foams.

- Examples of work activities involving isocyanates that require special attention when assessing exposure include:
  - all stages of manufacture and use where free isocyanates are released as vapours.
  - aerosols and mists.
  - spray painting, using two-pack paints with an isocyanate hardener, like in vehicle paints.
  - use of rigid foams for thermal insulation in refrigerators, storage tanks, packaging, and furniture.
  - use of flexible foams for bedding and upholstery.
  - use of hard-wearing coatings for furniture and floors.
  - manufacture of sporting goods such as skis, surfboards, and footwear.
  - spray on polyurethane products used as protective coatings for truck beds, trailers, boats, foundations, and decks.

- Processes where heat decomposition of polyurethane products occurs, such as welding, heat removal of electrical insulating varnishes and hot wire cutting of foam.
- Foundry operations, in particular core making, where resins used to bind the sand may contain isocyanates (for example the ‘Iso-Cure process’).
- Special attention should also be given to acute exposures that may occur in the above processes.

2.2 ROUTE OF EXPOSURE

- Inhalation (primary)
- Skin
- Oral

2.3 TOXICOKINETIC

Isocyanates	
Absorption	• Predominantly absorbed through the respiratory tract.
Distribution	• Conjugates directly with glutathione in the lungs after inhalation following mercapturic acid metabolic pathway. • GSH-isocyanate conjugate is distributed to other tissues. • Target organ for MIC, TDI and HDI are the eyes, skin, and respiratory tract. • Target organ for MDI are the eyes and respiratory system.
Metabolism	• Mono isocyanates undergo metabolism via the mercapturic pathway to form N-acetylated cysteine conjugates. • Metabolism of diisocyanates remain unclear.
Excretion	• Excreted in the urine as diamine and conjugate (N-acetylated cysteine conjugates). • Isocyanates-derived amines released from metabolism of isocyanate has an estimated half life of 2 to 5 hours in the urine. The estimated half life of plasma adducts is 20 to 25 days (Cocker., 2011).

- Once absorbed into the body, isocyanate builds covalent bonds with proteins (albumin, laminin, or cell membrane protein) to form an antigen and induce immune reaction.
- Sensitization to isocyanate occurs due to this covalent bond.

2.4 HAZARD CLASSIFICATION

- **Methyl Isocyanate**

Classification Code	Hazard Classification	H-Code	Signal
Flam. Liq. 2	Flammable liquids category 1	H275	Danger
Repr. 2	Reproductive toxicity category 2	H361d	
Acute Tox. 2 (inh)	Acute toxicity category 2	H330	
Acute Tox. 3 (dermal)	Acute toxicity category 3 dermal	H311	
Acute Tox. 3 (oral)	Acute toxicity category 3 oral	H301	

Resp. Sens. 1	Respiratory sensitization category 1	H334	Danger
Skin Sens. 1	Skin sensitization category 1	H317	
STOT SE 3	Specific Target Organ Toxicity-single exposure category 3	H335	
Acute Tox. 3 (oral)	Acute toxicity category 3 oral	H301	
Skin Irrit. 2	Skin corrosion or irritation category 2	H315	
Eye Dam. 1	Serious eye damage or eye irritation category 1	H318	

Source: ICOP on Chemicals Classification and Hazard Communication (Amendment) 2019.

- **Hexamethylene diisocyanate**

Classification Code	Hazard Classification	H-Code	Signal
Acute Tox. 3 (inh)	Acute toxicity category 3	H331	Danger
Eye Irrit. 2	Serious eye damage or eye irritation category 2	H319	
STOT SE 3	Specific target organ toxicity-single exposure category 3	H335	
Skin Irrit. 2	Skin corrosion or irritation category 2	H315	
Resp. Sens. 1	Respiratory sensitization category 1	H334	
Skin Sens. 1	Skin sensitization category 1	H317	

Source: ICOP on Chemicals Classification and Hazard Communication (Amendment) 2019.

- **Cancer Classification IARC**

  - TDI - Group 2B carcinogen (possibly carcinogenic to humans).
  - 4,4'-MDI - Group 3 carcinogen (not classifiable as to its carcinogenicity to humans).
  - Sensitization effects to respiratory tract and skin may be observed in exposure to isocyanates.

3.0 HEALTH EFFECTS MONITORING

Route of occupational exposure

- The primary route of isocyanate exposure is via inhalation. However, skin absorption can also be an important route of exposure.
- The risk of exposure depends on the volatility of the compound and the application process. The most commonly used isocyanates are:

  - toluene diisocyanate (TDI)
  - methylene diphenyl diisocyanate (MDI)
  - hexamethylene diisocyanate (HDI)

3.1 ACUTE EFFECTS

System/Organ	Chronic Effects
Ear, Nose and Throat	• Irritation to nose, eye and throat is an indication of overexposure.
Eyes	• Eye irritation • Tearing • At high exposures may cause: ○ Corneal ulceration ○ Blurred vision ○ Blindness ○ Permanent damage
Gastrointestinal	• Corrosive to the GI tract • Loss of appetite • Abdominal pain • Nausea • Vomiting
Hematological	• Increased blood acidity
Nervous System CNS and PNS	At high exposures may cause: • Weakness • Tremors • Depression • Anxiety • Tetany • Sleepiness

Reproductive	• Disrupted menstrual cycle.
	• Leucorrhoea and dysmenorrhoea.
	• Studies observed in MIC-exposed Bhopal population:
	  ○ Stillbirths
	  ○ Miscarriages
	  ○ Neurological effects on children of exposed parents.

Respiratory	• Corrosive to respiratory tract causing:
	  ○ Shortness of breath
	  ○ Coughs
	  ○ Bronchitis
	  ○ Increased secretion
	  ○ Chest pain
	• Irritation to lungs
	• Trigger asthmatic response as an allergic response in sensitized individuals.
	• At high exposure may cause pulmonary edema (delayed onset of a few hours) that may be fatal.
	  ○ Symptoms worsened by physical effort.

Skin	• Chemical burns. • At high doses may cause ulcers.

3.2 CHRONIC EFFECTS

Chronic exposure to isocyanates can cause contact dermatitis, immune sensitisation, and asthma and less commonly hypersensitivity pneumonitis.

Isocyanates generally appear to be weak human skin irritants and sensitisers. Sensitisation of the skin is not common and if this occurs it is usually due to inadequate work hygiene giving rise to extensive skin contamination with diisocyanates, solvents and additives. Sensitised people react with symptoms of skin irritation including blistering and swelling.

4,4'-diisocyanate dicyclohexylmethane, however, is a potent skin sensitiser. Smoking may be a risk factor for sensitisation to isocyanates.

A rare consequence of chronic isocyanate exposure is hypersensitivity pneumonitis, a granulomatous inflammatory reaction in terminal airways, alveoli and surrounding interstitium. Symptoms include dyspnoea, malaise and fever occurring several hours after work with isocyanates. There is a restrictive pattern on spirometry. Chest X-ray demonstrates a reticular or nodular lung pattern.

Interstitial pulmonary fibrosis has been reported as a long-term health outcome. Adverse health effects resulting from exposure to isocyanates normally arise during the ordinary working period, soon after contact occurs. Occasionally, as with hypersensitivity pneumonitis, symptoms may not appear for several hours following exposure.

Therefore, a correlation of symptoms with workplace exposure may not be obvious. It is important employees are informed of the potential for the delayed onset of adverse health effects and they should report adverse health effects that they think may be related to isocyanate exposure so the root-cause can be investigated.

System/Organ	Chronic Effects
Eyes	Studies on Methyl Isocyanate MIC-exposed Bhopal population: ○ Photophobia ○ Corneal ulcer ○ Cataract ○ Burning and watering ○ Discharge
Nervous System CNS and PNS	Studies on MIC-exposed Bhopal population: ○ Muscle weakness ○ Poor memory ○ Depression ○ Emotional disturbance ○ Cognitive impairment
Renal and Genitourinary	Studies on MIC-exposed Bhopal population: ○ Renal failure and chronic kidney disease
Respiratory	● Allergic sensitization of the respiratory tract resulting in asthma attacks for subsequent exposures even at low doses ● Chronic lung disease ● Tissue lesion in the lungs ● Increased risk of lung infection
Reproductive	Evidence suggesting it may cause miscarriages. Studies on MIC-exposed Bhopal population: ○ Increased infant mortality of exposed parents.
Skin	Allergic sensitization of the skin.

4.0 MEDICAL SURVEILLANCE PROGRAMME

4.1 INDICATIONS

Any occupational exposure to isocyanates above 50% of PEL TWA, and/or exceed the MEL and STEL and/or possibility of absorption.

4.2 CLINICAL EXAMINATION AND RELEVANT INVESTIGATIONS

- Pulmonary function testing - Spirometry
- Chest X-ray, as baseline and thereafter when there is clinical condition.
- BM sampling

4.3 PRE-PLACEMENT MEDICAL EXAMINATIONS

- Clinical examination and relevant investigation as listed in 4.2.
- Decision for fitness to work.
    - Relative contraindications i.e., conditions likely to be regulated as rendering those with them less fit for exposure to isocyanates are:
        - Hay fever, recurrent bronchitis, asthma, chronic pre-existing lung disease.
        - Some types of eczema.
        - Poor lung function test (i.e., man with FEV1 1 litre or more below normal or woman with FEV1 0.8 litre or more below normal).

4.4 PERIODIC MEDICAL EXAMINATION

- Clinical examination and relevant investigation as listed in 4.2.
- Chest X-ray (if clinically indicated)
- Frequency of periodic medical examination: every 6 months.

4.5 BIOLOGICAL MONITORING

- ACGIH TLVs & BEIs 2022 proposed specific determinant and BEI for 1,6-hexamethylene diisocyanate (HDI) and Toluene Diisocyanate (TDI).

Form of Isocyanate	Determinants	BEL	Sampling Time	Signal
1,6-Hexamethyle ne Diisocyanate for HDI	1,6-Hexamethyle ne Diisocyanate diamine in urine	15 µg/g creatinine (15 µmol/mol creatinine)	End of shift	-
Toluene Diisocyanate (TDI)-2,4 or 2,6- or as mixture of isomers	Toluene Diisocyanate diamine in urine	5 µg/g creatinine (5 µmol/mol creatinine)	End of shift	Ns

Source: ACGIH 2022 TLVs® and BEIs®

Note:
Urine samples represent current exposure and do not reflect long-term exposure.
Selection of the determinant will depend on the availability of the laboratory test.

- **Laboratory method**
  - Method reference: HSE Health & Safety Laboratory Guidance sheet for Isocyanates.
  - Sampling Procedure

Isocyanate Metabolites in Urine	
Container	Universal polystyrene container Note: *Request container from the laboratory *Own container can be used (provide at least one unit container to the lab for blank test).
Transport	Must arrive at laboratory within 48 hours of sampling.
Stability	Ambient temperature: 2 days Refrigerated: more than 3 months
Preservation	Addition of 0.5 g citric acid
Volume	Requested volume: 30 mL Minimum volume: 10 mL Creatinine correction is advised.

Source: HSE Health & Safety Laboratory

Note:
The creatinine in the urine should be measured within 24 hours of sample collection.

  - Analytical equipment/procedure

Determinants	Detection procedure
Isocyanate metabolites in urine	Mass spectrometry with negative ion chemical ionisation (methane).

Source: Health and Safety Executive (HSE), Health and Safety Laboratory (n.d)

5.0 MEDICAL REMOVAL PROTECTION

- Indications for removal

    - Where a medical examination indicates the employee is displaying symptoms of exposure to isocyanates.
    - Exceeds BEL.
    - Abnormal lung function test.
    - Pregnancy and breastfeeding.
    - Newly diagnosed asthma warrants a permanent removal.

    Note:
    The development of respiratory sensitisation is an idiosyncratic response that may affect some individuals at a specific exposure level while others remain unaffected.

- Temporary MRP

    - MRP based on medical condition.
        - The determination of the temporary MRP duration should consider the following aspects:
            - expected duration of diagnostic procedures.
            - duration of recovery.

    - MRP based on BEL.
        - The determination of the temporary MRP duration should consider the following aspects:
            - Availability of the repeat BM sample results

6.0 RETURN TO WORK

- Return to work is based on:

    i. Duration of temporary MRP based on BM, is the duration of the repeat BM sample to be available. This applies only for employees with NO isocynate related symptoms.

    ii. BEL
        - Return to work when the results are below the BEL.

    iii. Employees sensitised to isocyanates should be permanently removed.

iv. Abnormal lung function test associated with isocyanate should be permanently removed.

v. Employees with health conditions or continuing symptoms due to exposure to isocy nates should be advised to seek continuing medical examinations as organised by the OHD supervising the medical surveillance program.

vi. Workplace management
   - Ensure workplace hygiene is safe and healthy and does not place the employee and increased risk of material impairment to health from exposure to isocyanates.

7.0 NOTIFICATION TO DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed in NADOPOD 2004 using the relevant notification forms.

8.0 PREVENTIVE MEASURES

- CHRA of spray paintings and foam production that contains isocyanates should be done carefully with detailed information of the tasks and the possible exposures. Non routine exposures may also expose the employees to exposures that can lead to health effects

- Workplace and personal hygiene as well as safe work practices are essential.
  - Wash skin immediately with soap and water if contaminated.
  - Emergency showers and eyewash made available.

- Avoid wearing contact lenses when working with isocyanates.

- Protective clothing must be worn to prevent skin contact: suits, gloves, footwear, and headgear. All protective clothing must be kept clean.
  - When working with liquids, wear splash-proof chemical goggles and face shield or full-face-piece respiratory protection.

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LEAD:
INORGANIC LEAD

1.0 DESCRIPTION

The three lead compounds are organic lead, elemental and inorganic lead. Lead blood level means a total lead (Pb) level in blood and does not inform us on the type of lead, whether it is elemental, organic, or inorganic lead.

Total body burden of Lead in the human body, is both from occupational and non-occupational sources.

Human exposure to lead occurs through a combination of inhalation and oral exposure, with inhalation generally contributing a greater proportion of the dose for occupationally exposed groups, and the oral route generally contributing a greater proportion of the dose for the general population. The effects of lead are the same regardless of the route of exposure (inhalation or oral) and are correlated with internal exposure, as blood lead levels. For this reason, blood lead levels are often used to characterize exposure.

1.1 SYNONYMS

Lead metal; Plumbum.

1.2 OCCUPATIONAL EXPOSURE LIMIT (OEL)

USECHH 2000 (Eight-hour time weighted average limit)	
Lead Elemental and Inorganic, Pb	0.05 mg/m^3 (TWA)

1.3 PHYSICOCHEMICAL PROPERTIES

- Metallic lead is a bluish-white soft metal.
- Inorganic lead compounds (lead salt & lead soaps) and lead arsenate are white to yellowish orange crystals.

2.0 TOXICITY

2.1 SOURCES OF POTENTIAL OCCUPATIONAL EXPOSURE

- General industry (metal shield, plumbing and covering)
- Construction and shipyard (repair and renovation)

- Lead-related product manufacture
- Agriculture, automotive and organic lead production

2.2 ROUTE OF EXPOSURE

- Inhalation and ingestion of dust (primarily for solid lead compounds)
- Skin/eye contact and absorption (primarily for liquid organic lead compounds).

2.3 TOXICOKINETICS

Absorption	• Absorption of particulate lead following inhalation involves the deposition of airborne lead particles in the respiratory tract. • Gastrointestinal absorption of lead is affected by physicochemical characteristics of the lead particles and by physiological factors including age, fasting, nutritional calcium and iron status.
Distribution	• Transported primarily in the red blood cells bound to plasma proteins and distributed by blood to mineralising systems (bone, teeth) and soft tissues (e.g., liver).
Metabolism	• Metabolized via protein (metallothionein) and thiols binding. • It is not metabolized by the human body into other elements.
Excretion and Half- Life	• Based on studies, the half life was found to be: ○ Half Life in blood: 36 days ○ Half Life in soft tissue: 40 days ○ Half Life in bone: 27 years • Unabsorbed lead eliminated through faeces. • Absorbed lead is mainly excreted in urine with sweat, saliva, hair and nails and breast milk as minor routes.

2.4 HAZARD CLASSIFICATION

Best to refer to the SDS of respective compounds.

- **Cancer Classification IARC**

Inorganic Lead	Organic Lead
Group 2A (Probably Carcinogenic to Humans)	Group 3 (Not classifiable as to its carcinogenicity to humans)

3.0 HEALTH EFFECTS MONITORING

3.1 ACUTE EFFECTS

System/Organ	Acute Effects
Cardiovascular	Hypertension
Eyes	Eye irritation

Gastrointestinal	• Gastrointestinal disturbances:
	  ○ Constipation
	  ○ Abdominal pains
	  ○ Decreased appetite

Hepatobiliary	Hepatic damage
Musculoskeletal	Aching bones and muscles

Nervous System CNS and PNS	• Tiredness
	• Sleep disturbance
	• Irritability and depression
	• Headaches
	• Encephalopathy
	• Extremely high levels of exposure:
	  ○ Seizures
	  ○ Coma
	  ○ Death
	• Fatigue
	• Restlessness
	• Poor attention span
	• Hallucinations

Renal and Genitourinary	Renal damage
Skin	Skin irritation

3.2 CHRONIC EFFECTS

System/Organ	Chronic Effects
Cardiovascular	• Cardiovascular toxicity • Hypertension

Gastrointestinal	Gastrointestinal toxicity causing: • Abdominal pain • Nausea • Blue line at gum margin • Severe constipation • Vomiting
Hematological	• Anaemia • Basophilic stippling • Pale skin
Nervous System CNS and PNS	Neurological disturbances • Severe headache • Convulsions • Muscle weakness • Tremors • Irritability and Personality changes • Paralysis (forearm, wrist joint, fingers) • Lethargy • Coma • Ataxia Brain damage which may be permanent.
Renal and Genitourinary	Renal toxicity Kidney damage which may be permanent. Chronic nephritis and tubular degeneration
Reproductive	Adverse effects to male and female reproductive functions: • Reduced libido • Low semen volume and sperm counts. • Increased abnormal sperm morphology. • Decreased sperm motility Occupational exposure of pregnant women associated with increased risk of: • Spontaneous abortion • Preterm delivery • Low birth weight Lead can cross the placenta and may cause neurological damage to the fetus.

4.0 MEDICAL SURVEILLANCE PROGRAMME

4.1 INDICATIONS

Airborne exposure exceeding the 50% of PEL and/ or significant risk of hand to oral contacts.

4.2 CLINICAL EXAMINATION AND RELEVANT INVESTIGATION

- Clinical examination with emphasis on renal, haematological, gastrointestinal, and nervous system.
- Current usage of medication with potential nephrotoxic side-effects
- Blood lead (Pb) level (venous blood in heparinised container).
- Full blood examination
- Renal function test
- Routine urinalysis
- Pulmonary function tests (where exposure may be via airborne lead fume or dusts or where respiratory protection is required).

4.3 PRE-PLACEMENT MEDICAL EXAMINATION

- Clinical examination and relevant investigations as listed in 4.2.

- Decision for fitness to work:
  - Persons with a history of renal, haematological, gastrointestinal, and nervous system dysfunction are unfit for exposure to Lead.
  - Pre-placement medical examination should be conducted **prior to exposure.**

Note:
Persons with thalassaemia minor may work with inorganic lead.

4.4 PERIODIC MEDICAL EXAMINATION

- Generally, once a year. More frequent examination when abnormal results was detected. The OHD should decide based on the findings, until problem is resolved.

4.5 BIOLOGICAL MONITORING

Determinant	Sampling time	BEL	Notation
Lead in blood	Not critical	50 µg/dL	-

Note:
Persons applying this BEL over the current CDC reference value (CDC: Guidelines for the identification and management of lead exposure in pregnant and lactating women, 2010) are encouraged to counsel female employees of child-bearing age about the risks.

Repeat test frequency

Category	Level of last BEI determinant and the frequency
Males and females not of reproductive capacity	• less than 30 µg/dL: six (6) months • 30 µg/dL or more but less than 40 µg/dL: three (3) months • 40 µg/dL (1.93 µmol/L) or more: six (6) weeks
Females of reproductive capacity	• less than 10 µg/dL (0.48 µmol/L): 3 months • 10 µg/dL (0.48 µmol/L) or more: 6 weeks
Females who are pregnant or breastfeeding	15 µg/dL

- **Laboratory method**
  - Method reference: NMAM 8003
  - Sampling procedures

Lead in Blood	
Container	Heparinized, lead-free, “blue-top” blood collection tubes Polyethylene bottles (wide mouth) for urine Note: *Request container from the laboratory *Own container can be used (provide at least one unit container to the lab for blank test).
Transportation	Polyethylene shippers
Stability	Blood stable for 3 days when refrigerated
Preservation	Heparin anticoagulant for blood
Volume	Blood samples Requested volume: 7 mL Minimum volume: 2 mL

  - Analytical equipment/procedure

Lead level	• Atomic Absorption, Flame • Inductively Coupled Plasma Mass Spectrometer (ICP-MS)

5.0 MEDICAL REMOVAL PROTECTION

MRP of an employee from a lead risk work

Category	Level for immediate MRP
Males and females not of reproductive capacity	50 µg/dL
Females of reproductive capacity	20 µg/dL
Females who are pregnant or breastfeeding	15 µg/dL

A second medical examination should be conducted **within seven days** after the day the employee is removed from lead risk work. The frequency should be done at least every three to six weeks until the appropriate fall in blood lead levels has occurred.

- All employees undergoing MRP must be investigated to determine the cause of anemia.
- All cases recommended for MRP, suspected cases of lead poisoning and excessive absorption must be notified to the DG of DOSH.

6.0 RETURN TO WORK

i. An employee must not return to lead risk work until the employee's blood lead level is less than:

Category	Level to RTW
Males and females not of reproductive capacity	40 µg/dL
Females of reproductive capacity	10 µg/dL

And has been assessed as medically fit to return to lead risk work by the OHD supervising the health monitoring.

ii. Medical condition
   - No longer detected having any medical conditions.

iii. Workplace management
   - Ensure workplace hygiene is safe and healthy and does not place the employee at increased risk of material impairment to health from exposure to inorganic lead.

7.0 NOTIFICATION TO DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed in NADOPOD 2004 using the relevant notification forms.

8.0 TREATMENT AND MANAGEMENT

- All cases of lead poisoning must be immediately removed from exposure, provided appropriate first aid measures, and refer for hospital treatment.
- Treatment based on specific medical conditions arising from exposure to the chemical.

9.0 PREVENTIVE MEASURES

- Good workplace and personal hygiene and safety.
- Approved personal protective equipment.
- Employees should be encouraged to use washing or showering facilities at the workplace and change clothes prior to going home to minimise secondary lead exposure from contaminated clothing and minimise ingestion of lead.
- Employees should be reminded:

    i. they are not permitted to smoke, carry materials used for smoking, eat, chew gum or drink in a lead process area.
    ii. the importance of removing lead contaminated clothing and equipment.
    iii. to wash their hands and faces, before entering areas designated for eating and drinking.

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LEAD:
ORGANIC LEAD

1.0 DESCRIPTION

The three lead compounds are organic lead, elemental and inorganic lead. Blood lead level means a total lead (Pb) level in blood but it does not tell us on the type of lead, whether it is elemental, organic or inorganic lead.

Total body burden of lead in the human body, is both from occupational and non occupational sources.

Human exposure to lead occurs through a combination of inhalation and oral exposure, with inhalation generally contributing a greater proportion of the dose for occupationally exposed groups, and the oral route generally contributing a greater proportion of the dose for the general population. The effects of lead are the same regardless of the route of exposure (inhalation or oral) and are correlated with internal exposure, as blood lead levels. For this reason, blood lead levels are often used to characterize exposure.

Two examples of organic lead compounds are tetraethyl lead (TEL) and tetramethyl lead (TML). Both of these compounds were commonly used as gasoline additives in the past but were gradually phased out in the 1980's due to environmental concerns.

1.1 SYNONYMS

Lead metal; Plumbum.

1.2 OCCUPATIONAL EXPOSURE LIMIT (OEL)

USECHH 2000 (Eight-hour time weighted average limit)	
Organic lead	Not available

1.3 PHYSICOCHEMICAL PROPERTIES

- Organic lead compounds (alky-lead) are liquid at room temperature.

2.0 TOXICITY

2.1 SOURCES OF POTENTIAL OCCUPATIONAL EXPOSURE

- Leaded fuel; the cleaning of gasoline tanks.
- Occupational exposure is rarer now.

2.2 ROUTE OF EXPOSURE

- Inhalation and ingestion of dust (primarily for solid lead compounds)
- Skin/eye contact and absorption (primarily for liquid organic lead compounds).

2.3 TOXICOKINETICS

Absorption	• Absorption of particulate lead following inhalation involves the deposition of airborne lead particles in the respiratory tract. • Gastrointestinal absorption of lead is affected by physicochemical characteristics of the lead particles and by physiological factors including age, fasting, nutritional calcium and iron status.
Distribution	• Transported primarily in the red blood cells bound to plasma proteins and distributed by blood to minerlising systems (bone, teeth) and soft tissues (e.g., liver).
Metabolism	• Metabolized via protein (metallothionein) and thiols binding. • It is not metabolized by the human body into other elements.
Excretion and Half- Life	• Based on studies, the half life was found to be: ○ Half Life in blood: 36 days ○ Half Life in soft tissue: 40 days ○ Half Life in bone: 27 years • Unabsorbed lead eliminated through faeces. • Absorbed lead is mainly excreted in urine with sweat, saliva, hair and nails and breast milk as minor routes.

2.4 HAZARD CLASSIFICATION

Best to refer to the SDS of respective compounds.

- **Cancer Classification IARC**

Inorganic Lead	Organic Lead
Group 2A (Probably Carcinogenic to Humans)	Group 3 (Not classifiable as to its carcinogenicity to humans)

3.0 HEALTH EFFECTS MONITORING

3.1 ACUTE EFFECTS

System/Organ	Acute Effects
Cardiovascular	Hypertension
Eyes	Eye irritation

Gastrointestinal	• Gastrointestinal disturbances:
	  ○ Constipation
	  ○ Abdominal pains
	  ○ Decreased appetite

Hepatobiliary	Hepatic damage
Musculoskeletal	Aching bones and muscles

Nervous System
CNS and PNS
	• Tiredness
	• Sleep disturbance
	• Irritability and depression
	• Headaches
	• Encephalopathy
	• Extremely high levels of exposure:
	  ○ Seizures
	  ○ Coma
	  ○ Death
	• Fatigue
	• Restlessness
	• Poor attention span
	• Hallucinations

Renal and Genitourinary	Renal damage
Skin	Skin irritation

3.2 CHRONIC EFFECTS

System/Organ	Chronic Effects
Cardiovascular	• Cardiovascular toxicity • Hypertension
Gastrointestinal	Gastrointestinal toxicity causing: • Abdominal pain • Nausea • Blue line at gum margin • Severe constipation • Vomiting
Hematological	• Anaemia • Basophilic stippling • Pale skin
Nervous System CNS and PNS	Neurological disturbances • Severe headache • Convulsions • Muscle weakness • Tremors • Irritability and Personality changes • Paralysis (forearm, wrist joint, fingers) • Lethargy • Coma • Ataxia Brain damage which may be permanent.
Renal and Genitourinary	Renal toxicity Kidney damage which may be permanent. Chronic nephritis and tubular degeneration
Reproductive	Adverse effects to male and female reproductive functions: • Reduced libido • Low semen volume and sperm counts. • Increased abnormal sperm morphology. • Decreased sperm motility Occupational exposure of pregnant women associated with increased risk of: • Spontaneous abortion • Preterm delivery • Low birth weight Lead can cross the placenta and may cause neurological damage to the fetus.

4.0 MEDICAL SURVEILLANCE PROGRAMME

4.1 INDICATIONS

Significant exposures that can lead to excessive absorption.

4.2 CLINICAL EXAMINATION AND RELEVANT INVESTIGATION

- Clinical examination with emphasis on renal, haematological, gastrointestinal, and nervous system.
- Current usage of medication with potential nephrotoxic side-effects.
- Full blood examination
- Renal function test
- Routine urinalysis
- Pulmonary function tests (where exposure may be via airborne lead fume or dusts or where respiratory protection is required).
- Estimation of urinary Pb concentration in an early morning urine specimen collected at the end of the work week. Urine lead is more accurate to reflect organic lead exposure.

4.3 PRE-PLACEMENT MEDICAL EXAMINATION

- Clinical examination and relevant investigations as listed in 4.2.
- Decision for fitness to work:
  - Persons with a history of renal, haematological, gastrointestinal, and nervous system dysfunction are unfit for exposure to Lead.
  - Pre-placement medical examination should be conducted **prior to exposure.**

Note:
Persons with thalassaemia minor may work with inorganic lead.

4.4 PERIODIC MEDICAL EXAMINATION

- Generally, once a year. More frequent examination when abnormal results are detected. The OHD should decide based on the findings, until the problem is resolved.

4.5 BIOLOGICAL MONITORING

Determinant	Sampling time	BEL
Urine Pb level	Random	● 110 µg/L urine (male) ● 25 µg/L urine (female)

Source: Singapore Workplace Safety and Health Guidelines.

- **Laboratory Method:**
  - Method reference: NMAM 8003
  - Sampling Procedure

Lead in Urine	
Container	Polyethylene or trace metal-free plastic container Note: *Request container from the laboratory *Own container can be used (provide at least one unit container to the lab for blank test).
Transportation	Urine specimens should be refrigerated.
Stability	• Urine specimens are stable at room temperature for 5 days. • Urine specimens are stable for 14 days when refrigerated. • Urine specimens are stable for 30 days when frozen.
Sample Volume	• Requested volume: 30 mL • Minimum volume: 10 mL

- Analytical equipment/procedure:

Chemical	Detection Procedure
Lead in urine	Inductively Coupled Plasma Mass Spectrometer (ICP-MS)

5.0 MEDICAL REMOVAL PROTECTION

Cases of definite or suspected lead poisoning and excessive absorption.	All cases
Elevated Pb urine	
Urine Pb level in 2 successive examinations.	>110 µg/L urine (male). >25 µg/L urine (female).

- All cases recommended for MRP, and suspected cases of lead poisoning/excessive absorption must be notified to the DG of DOSH.
- All employees undergoing MRP should have repeat urine lead examinations and follow-up at monthly intervals
- They should not return to lead work until the urine lead level has fallen to below the return levels (see below), all other biochemical results have returned to normal, and any related signs and symptoms have disappeared.
- Renal function test
- Review of abnormal results includes:
  - Investigating the cause of anemia.
  - Repeating hemoglobin level in 3 months.

6.0 RETURN TO WORK

- Return to Work is based on:
  i. Urine Pb Level

Category	Level
Urine Pb Level below	110 µg/L urine (male). 25 µg/L urine (female).

Monthly follow up with sampling of urine lead is recommended until the level is consistently lower than the BEL.

ii. Medical condition
  - No longer detected having any medical conditions.

iii. Workplace management
  - Ensure workplace hygiene is safe and healthy and does not place the employee at increased risk of material impairment to health from exposure to organic lead.

7.0 NOTIFICATION TO DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed in NADOPOD 2004 using the relevant notification forms.

8.0 TREATMENT AND MANAGEMENT

- All cases of lead poisoning must be immediately removed from exposure, provided appropriate first aid measures, and refer for hospital treatment.
- Treatment based on specific medical conditions arising from exposure to the chemical.

ORGANIC LEAD

- Treatment with chelating agents does not appear to be useful for organic lead poisoning. Symptomatic and supportive treatment is indicated.
- Several weeks to years may be necessary for recovery, which may not be complete.

9.0 PREVENTIVE MEASURES

- Good workplace and personal hygiene and safety.
- Approved personal protective equipment.
- Employees should be encouraged to use washing or showering facilities at the workplace and change clothes prior to going home to minimise secondary lead exposure from contaminated clothing and minimise ingestion of lead.
- Employees should be reminded:
  i. they are not permitted to smoke, carry materials used for smoking, eat, chew gum or drink in a lead process area.
  ii. the importance of removing lead contaminated clothing and equipment.
  iii. to wash their hands and faces, before entering areas designated for eating and drinking.

10.0 REFERENCES

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MANGANESE

1.0 DESCRIPTION

Most manganese is used in the production of a range of steel alloys such as stainless steels, tool steels and high temperature steels. Manganese is used as it hardens and strengthens the steel. It is also used in dry cell batteries, glass, and ceramics.

1.1 SYNONYMS

- Manganese dioxide, potassium permanganate, pyrolusite.

1.2 OCCUPATIONAL EXPOSURE LIMIT (OEL)

USECHH 2000 (Eight-hour time weighted average limit)	
Manganese, elemental and inorganic compound, as Mn	0.2 mg/m^3 (TWA)

1.3 PHYSICOCHEMICAL PROPERTIES

- Exist as a metal (metallic manganese, ferromanganese), as inorganic manganese (chloride, sulfate salts) or as organic manganese.

Manganese, elemental & inorganic compounds (as Mn)
- A brittle and very hard metal which is silver-white in appearance. Manganese possesses a wide range of oxidation states. (Lang & Dietrich, 2013).
- Manganese metal is a combustible solid.

Manganese dioxide
- Black crystalline solid or powder.
- A strong oxidant which exposed to will generate fire and explosion hazard.

Manganese cyclopentadienyl tricarbonyl
- Exist as a yellow, crystalline solid which has a distinct/characteristic odour (Centers for Disease Control and Prevention, n.d.)
- Combustible solid.

2.0 TOXICITY

2.1 SOURCE OF POTENTIAL OCCUPATIONAL EXPOSURE

Manganese, elemental & inorganic compounds (as Mn)
- Exposure to manganese is usually by inhalation of dust and fume from mining, metal processing, grinding, and welding.

- In mining industries, the Manganese known as Manganese ores are the oxides pyrolusite, romanechite, manganite, and hausmannite and the carbonate ore rhodochrosite. Rhodonite and braunite, both silicate ores, are frequently found with the oxides.

Manganese dioxide

- Manufacturing dry cell batteries, pyrotechnics, and matches.
- Other processes involving manganese.
- Laboratory use

Manganese cyclopentadienyl tricarbonyl (organic Mn compounds)

- Gasoline or automobile mechanics.
- Occupation involving fuel oil and distilled fuel.

2.2 ROUTE OF EXPOSURE

Manganese, elemental & inorganic compounds (as Mn)

- Inhalation of dust or fume.
- Liquid poorly absorbed through the skin.
- Ingestion

Manganese cyclopentadienyl tricarbonyl (organic Mn compounds)

- Inhalation
- Skin absorption
- Ingestion
- Skin and/or eye contact

2.3 TOXICOKINETICS

Absorption	• Absorbed in the gastrointestinal system and lungs with inhaled manganese more rapidly absorbed than ingested manganese. • Approximately 3-5% manganese is absorbed in the gastrointestinal tract. • Smaller particles of inhaled manganese travel to lower airways and absorbed into blood and lymph fluids. • Larger particles are transported to the upper airways and enter the gastrointestinal tract by mucociliary transport to the throat.
Distribution	• Reduced to Mn 2+ and distributed through blood circulation on albumin, beta-globulin proteins and as stable complexes with weak acids and bases. • May enter the central nervous system through transport in the form of Mn-citrate complex or directly via the choroid plexus. • May cross the blood-placental barrier and interrupt fetal development. • Distributed to all tissues in the body with higher levels found in the liver, kidney, pancreas, and adrenals. Also found in the brain, heart, and lungs.

Metabolism	• Manganese is not metabolically converted to other products.
Excretion	• Conjugated in the liver and sent to the bile where it is eliminated via faeces. • Half-Life varies according to target organs. Manganese is usually removed from the body within a few days (ATSDR 2012). Animal studies have found the half-life in the brain parenchyma is around 5 – 7 days. Half life in the bones is around 8.5 years.

- Disrupts energy production, gene expression and increases reactive oxygen species (ROS).
- High level of Mn in the brain may cause manganism or manganese poisoning that mimics Parkinson's disease.
- Deposited in the globus pallidus and corpus striatum in the brain causing reduction and damage of catecholamines like dopamine.
- Replaces Mg2+ in some enzymes subsequently disrupting calcium metabolism.

2.4 HAZARD CLASSIFICATION

Manganese Compound	Classification Code	Hazard Classification	Hazard Code	Signal
Manganese dioxide	Acute Tox. 4 (inh)	Acute toxicity category 4 (inhalation)	H332	Warning
	Acute Tox. 4 (oral)	Acute toxicity category 4 (oral)	H302	
Manganese sulphate	STOT RE 2	Specific Target Organ Toxicity-Repeated Exposure category 2	H373[(a)]	

*(a) - State the target organ
Source: ICOP on Chemicals Classification and Hazard Communication (Amendment) 2019

- Cancer Classification IARC
  Not listed

3.0 HEALTH EFFECTS MONITORING

Inhalational exposure to manganese at high levels may result in adverse effects to the nervous system termed 'manganism'. Less severe central nervous system effects include slowed hand movements. May also cause respiratory effects like lung irritation and subsequently pneumonia.

3.1 ACUTE EFFECTS

Elemental manganese and inorganic compounds (as Mn) as dust and fumes.

SYSTEM/ORGAN	Acute Effects
Eyes	Irritating to eyes.
Respiratory	• Pneumonitis (possibly delayed). • Irritation to mucous membrane of respiratory tract.

Manganese dioxide

SYSTEM/ORGAN	Acute Effects
Nervous System CNS and PNS	• Flu-like illness: ○ Chills ○ Aching ○ Fever
Respiratory	• Irritation of respiratory system • Chest congestion with cough and shortness of breath • Asthma-like lung allergy (delayed effects)

Manganese cyclopentadienyl tricarbonyl

SYSTEM/ORGAN	Acute Effects
Eye	Irritation to eyes
Gastrointestinal	• Signs following skin contact: ○ Thick tongue
Nervous System CNS and PNS	• Signs following skin contact: ○ Dizziness ○ Nausea ○ Headache
Respiratory	Irritation to respiratory system.
Skin	Irritation to skin.
Others	• Signs following skin contact: ○ Tissue lesions

3.2 CHRONIC EFFECTS

Elemental manganese and inorganic compounds (as Mn) as dust and fumes.

SYSTEM/ORGAN	Chronic Effects
Hematological	Anemia
Hepatobiliary	Permanent damage to liver.
Nervous System CNS and PNS	• Permanent brain damage ○ Neurologic and neuropsychiatric disorder or known as manganism. • Three Phases of Manganese Poisoning: First (Early) Phase • Apathy, anorexia, absthenia. Headache, hypersomnia, cramps, weakness of the legs, athralgia and irritability. Second Phase • Manganese psychosis which may disappear if exposure ceases. Symptoms are euphoria, impulsive actions, mental confusion, hallucination, and aggressiveness. Late Phase • Speech disturbances, mask-like face, microphagia (abnormal handwriting). • Disturbances in balance and movement such as tremor and difficulty in walking and speaking. • Excessive salivation and sweating, removal from reality and spasmodic laughter.
Renal and Genitourinary	Permanent damage to kidneys.
Reproductive	Animal studies indicate reproductive effects.
Others	Permanent damage to lungs: • Bronchitis • Pneumonitis

Manganese dioxide

SYSTEM/ORGAN	Chronic Effects
Hepatobiliary	Liver damage
Nervous System CNS and PNS	• Permanent brain damage

	• Neurologic and neuropsychiatric disorders or known as manganism. • Early symptoms: ○ Poor appetite ○ Weakness ○ Sleepiness • Manganese substance that damages the axon causing symptoms resembling Parkinson's disease.
Renal and Genitourinary	Kidney damage
Reproductive	Animal tests indicate reproductive effects.
Respiratory	• Permanent lung damage • Bronchitis • Pneumonitis

Manganese cyclopentadienyl tricarbonyl

SYSTEM/ORGAN	Chronic Effects
Renal and Genitourinary	Kidney damage
Respiratory	Lung damage

4.0 MEDICAL SURVEILLANCE PROGRAMME

4.1 INDICATIONS

Any work where employees are exposed to levels of airborne levels, which are liable to be in excess of half the -in-air standard, and/or where there is significant risk of absorbing it.

4.2 CLINICAL EXAMINATION AND RELEVANT INVESTIGATION

- Clinical examination and baseline data with particular attention to behavioural and neurological changes (speech and emotional disturbances, hypersonic tremor, equilibrium, gait, handwriting and adiadochokinesis), renal and genitourinary, liver, and respiratory system.
- Urine manganese (U-Mn) estimation on post-shift urine specimen collected at end of workweek.
- Kidney function test (manganese dioxide and Manganese cyclopentadienyl tricarbonyl.
- Liver function test.
- Complete Blood Count (CBC) including total white and differential count.

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4.3 PRE-PLACEMENT MEDICAL EXAMINATION

- Clinical examination and relevant investigation as listed in 4.2.
- Baseline urine manganese.
- Decision for fitness to work:
  - Individuals with disease of liver, kidneys and central nervous system or alcoholism should not be exposed to manganese.

4.4 PERIODIC MEDICAL EXAMINATION

- Clinical examination and relevant investigations as listed in 4.2.
- Frequency of periodic medical examination: annual
- More frequent assessment when abnormalities are detected.

4.5 BIOLOGICAL MONITORING

- No established BEI for manganese and inorganic compounds from ACGIH 2022.
- The European Commission cites limited methods for biological monitoring of manganese exposure.
- However, manganese levels in blood and urine are recommended as confirmation of increased manganese exposure at the group level (Lauwerys and Hoet 2001).

Determinant	Sampling time	BEL
Urine as U-Mn	End of workweek, post shift	50 mcg/L

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Source: BTLV Workplace Safety and Health Council of Singapore

- Repeat urine manganese and medical examination if U-Mn >40 mcg/L but less than 50 mcg/L, at more frequent intervals, such as 3-month intervals. Levels below 50 mcg/L, does not require MRP.

- Laboratory method
  - Method reference: NMAM 8310
  - Sampling procedures

Manganese in Urine	
Container	Polyethylene or trace metal-free plastic container Note: *Request container from the laboratory *Own container can be used (pprovide at least one unit container to the lab for blank test).
Transport	Urine specimens should be refrigerated.
Stability	● Urine specimens are stable at room temperature for 5 days. ● Urine specimens are stable for 14 days when refrigerated. ● Urine specimens are stable for 30 days when frozen.
Volume	Requested volume: 30 mL Minimum volume: 10 mL

Source: NMAM 8310

- Analytical equipment/procedure

Chemical	Detection Procedure
Manganese in urine	Inductively Coupled Plasma Mass Spectrometer (ICP-MS)

5.0 MEDICAL REMOVAL PROTECTION

- Indications for removal
  - Clinical findings
    - Evidence of health effects that is suspected to be caused by CHTH or may be worsened by CHTH.
  - Target organ function abnormalities.
  - BEL
    - Exceed BEL
  - Others:
    - Medical conditions of the liver, kidneys and central nervous system or alcoholism.

- Duration of temporary MRP based on BM, is the duration of the repeat BM sample to be available. Return to work when the results are below the BEL.
- Duration of the temporary MRP based on the medical conditions, considering the expected duration of diagnostic procedures and the duration of recovery.
- All cases recommended for suspension and suspected cases of poisoning and exces sive absorption must be notified to the DG of DOSH.

6.0 RETURN TO WORK

- Return to Work is based on:
  - i. Urine Mn levels and other biochemical results.
    - Abnormal results have returned to normal.
  - ii. Medical condition
    - No longer detected of having a medical condition.
  - iii. Workplace management
    - Ensure workplace hygiene is safe and healthy and does not place the employee and increased risk of material impairment to health from exposure to manganese.

7.0 NOTIFICATION TO DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed in NADOPOD 2004 using the relevant notification forms.

8.0 TREATMENT AND MANAGEMENT

Seek medical attention immediately if a chemical gets into eyes, contacts the skin or has been inhaled.

- Supportive, irrigate eyes with water.
- Wash contaminated areas of body with soap and water.
- Gastric lavage, if ingested, followed by saline catharsis.
- Oxygen and artificial respiration.
- Supportive measures.
- Refer to hospital.

9.0 PREVENTIVE MEASURES

- Adequate ventilation.
- Protective clothing including chemical goggles, chemical cartridge respirator, polyvinyl gloves.
- Appropriate signage.

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MERCURY (Elemental and Inorganic Mercury)

Occupational sourced form of mercury is usually elemental and inorganic mercury. However, the total burden of mercury toxicity of an employee is the results of all types of mercury in his body.

In the environment, elemental mercury may be converted to inorganic mercury, where inorganic mercury will also be converted to methyl mercury (organic mercury). Methyl mercury is normally found as a contamination in the foods especially marine sourced.

The biological exposure determinant is the total mercury in urine.

The OHD should always remember these facts when managing abnormal findings related to mercury.

1.0 DESCRIPTION

Mercury exists as elemental mercury, inorganic mercury, and organic mercury (WHO 2021).

1.1 SYNONYMS

Elemental Mercury

- Quicksilver, Hydrargyrum, metallic mercury, liquid silver (National Library of Medicine 2021) Colloidal mercury.

Inorganic Mercury

- Colloidal mercury, Hydragyrum, Liquid silver, Metallic mercury, mercuric chloride, mercurous chloride, calomel
- Methyl mercury chloride: Chloromethylmercury; Methylmercuric chloride; Methylmercury chloride; MMC; Monomethyl mercury chloride, Dimethyl mercury: Mercury dimethyl

Organic Mercury

- Methylmercury, which is known to be the most poisonous among the mercury compounds is created (by microorganisms) when inorganic mercury circulating in the general environment is dissolved into freshwater and seawater. It is known to become condensed through the ecological food chain and ingested into humans. Accordingly, methylmercury can be ingested through food intake by people whose occupations are not directly related to mercury exposure, and this can affect human health.

1.2 OCCUPATIONAL EXPOSURE LIMITS (OEL)

USECHH 2000 (Eight-hour time weighted average limit)	
Alkyl compounds	0.01 mg/m³ (TWA) 0.03 mg/m³ (STEL)
Aryl compounds	0.1 mg/m³ (TWA)
Other Inorganic forms including metallic mercury	0.025 mg/m³ (TWA)

1.3 PHYSICOCHEMICAL PROPERTIES

Elemental Mercury

- Exists as a shiny, silver-white metal.
- Liquid at room temperature.
- Elemental mercury has a low latent heat of evaporation which contributes to its ease of evaporation at room temperature.
- Non-combustible but emits irritating or toxic fumes.

Inorganic Mercury

- May exist in two oxidative states which is mercurous (Hg^{+}) and mercuric (Hg^{2+}) salts in solid state.
- Mercuric chloride exists as white crystals or dust.
- Non-combustible but may emit irritating fumes and gas in a fire.
- May degrade when exposed to light or heat producing toxic fumes of mercury and chlorine.

1.4 MATERIAL USE

Elemental Mercury

- A component in:
  - Thermometer
  - Dental amalgam or “silver filling”
  - Aritisnal gold mining and production of mercury-gold amalgam
  - Cement production
  - Contaminants in crude oil

Inorganic Mercury

- Found in:
  - Small scale artisanal gold mining
  - Dental amalgam
  - Pharmaceutical products
  - Electrical component like switches and relays
  - Used as a catalyst in several manufacturing processes
  - Mercury lamps
  - Chlor-alkali production
  - Semiconductors in solar cells
  - Battery, thermometer, barometer, and thermostat

2.0 TOXICITY

2.1 SOURCE OF OCCUPATIONAL EXPOSURE

Elemental Mercury

- Mining of mercury containing ores.
- Manufacture of dental amalgam, barometer, battery, thermometer, fluorescent lamps, insecticide.
- Petrochemical industry using chlor-alkali.
- Gold and silver extraction.
- Oil and gas upstream activities, as Contaminants in crude oil.

Inorganic Mercury

- Manufacture of electrical instruments (lamps, rectifier, battery), switches, thermometer, barometer, paint pigment, antifouling paint, vermilion.
- Gold and silver extraction.
- Dentistry and pharmaceutical industries.
- Laboratory works using mercury.
- Agriculture
- Filtering, production, and processing of natural gas.

2.2 ROUTE OF EXPOSURE

- Inhalation
- Skin absorption
- Skin and/or eye contact

2.3 TOXICOKINETICS

Absorption	**Elemental Mercury** ● Mercury inhaled as vapours are readily absorbed into the respiratory system and diffused through cell membranes. **Inorganic Mercury** ● Inorganic mercury can be absorbed through the skin, where mercuric chloride is more readily absorbed than mercurous chloride. ● Approximately 7% to 15% are absorbed in the gastrointestinal tract following ingestion.
Distribution	**Elemental Mercury** ● It can easily pass through the blood-brain barrier and blood-placental barriers. **Inorganic Mercury** ● May also be distributed throughout the body via plasma. ● Does not readily cross cell membranes due to low lipophilicity. ● Large amount of mercuric mercury deposited in the proximal convoluted renal tubule.

Metabolism	**Elemental and Inorganic Mercury** ● Undergo oxidation-reduction cycle. ● Elemental mercury transforms into divalent inorganic cation via oxidation in red blood cells and lungs. ● The divalent cation is then reduced to metallic or monovalent form and released as exhaled elemental mercury vapour.
Excretion and Half Life	Main pathway of inorganic and elemental mercury elimination from the body is through the urine and feces. Inorganic mercury has a half-life of around 60 days.

- Inhaled elemental mercury vapours enter the bloodstream and undergo rapid oxidation mainly in red blood cells to inorganic divalent form.
- Unoxidized elemental mercury in the brain can be oxidised to the inorganic form, resulting in trapped inorganic mercury in the brain.

2.4 HAZARD CLASSIFICATION

Mercury, as Hg

- Alkyl compounds
- Aryl compounds
- Elemental mercury and inorganic compounds

Classification Code	Hazard Classification	H-Code	Signal
Repr. 1B	Reproductive toxicity category 1B	H360D	Danger
Acute Tox. 2 (inh)	Acute toxicity category 2	H330	
STOT RE 1	Specific target organ toxicity-repeated exposure category 1	H372 (CNS, peripheral nervous system, kidney, gingiva, cardiovascular system, blood system, liver) (inh)	

Source: ICOP on Chemicals Classification and Hazard Communication (Amendment) 2019.

- Cancer Classification IARC
  Elemental mercury and inorganic mercury compound:
  IARC: Group 3 (not classifiable as to their carcinogenicity to humans)

- Lethal blood in humans: 0.4-22 mg/ml

3.0 HEALTH EFFECTS MONITORING

Acute and chronic exposure to mercury causes adverse effects to the central nervous system characterised by tremors, neuromuscular disturbances, headaches, alteration in nerve responses and others. Exposure to higher levels of mercury may affect the kidney and cause respiratory failure.

3.1 ACUTE EFFECTS

System/Organ	Acute Effects
Eyes	Irritation to eyes
Gastrointestinal	Exposure to high levels may cause: ○ Abdominal cramps ○ Nausea ○ Metallic taste ○ Gingivitis ○ Diarrhoea ○ Vomiting ○ Stomatitis and gastroenteritis ○ Increase salivation.
Musculoskeletal	● Muscle cramps ● Decrease in arm and leg sensation and strength.
Nervous System CNS and PNS	Exposure to high levels may cause: ○ Headache ○ Chills ○ Fatigue ○ Decreased nerve conduction. ○ Tremors ○ Fever ○ Polyneuropathy
Renal and Genitourinary	Acute renal failure
Respiratory	○ Dyspnoea ○ Coughing up blood ○ Difficulty breathing ○ Pneumonitis which may be fatal. ○ Cough ○ Chest tightness ○ Lung and bronchial irritation with possible damage.
Skin	Irritation to skin

3.2 CHRONIC EFFECTS:

System/Organ	Acute Effects
Ear, Nose and Throat	Irritation of the gums with blue lines between teeth and gums.
Eyes	Clouding of the eyes
Gastrointestinal	Accumulation in the liver and kidneys will cause: ● Increased salivation ● Loss of appetite ● Vomiting ● Gum irritation with blue line between teeth and gums. ● Loose teeth ● Nausea ● Diarrhoea
Hepatobiliary	May accumulate in the liver and cause damage.
Nervous System CNS and PNS	May accumulate in the brain at high concentrations with very slow excretion causing: ○ Impaired memory ○ Possible permanent brain damage ○ Personality changes Accumulation in the liver and kidneys will cause: ○ Headache ○ Restlessness ○ Sleepiness ○ Disturbances in muscle control ○ Dizziness ○ Irritability ○ Tremors ● Exposure to >500 µg/m3 of mercury vapor may cause: ○ Peripheral neuropathy ● Exposure to >500 µg/m3 of inorganic mercury may cause: ○ Acrodynia
Renal and Genitourinary	● May accumulate in the kidneys and cause urinary alterations. ● Exposure to >500 µg/m3 of mercury vapor may cause: ○ Proteinuria
Reproductive	Elemental mercury cross placental barrier and may cause developmental defects.
Skin	● May cause skin allergy. ● Raised red areas and blisters. ● Grey skin colour.
Others	Weight loss

4.0 MEDICAL SURVEILLANCE PROGRAMME

4.1 INDICATIONS

Any work where employees are exposed to levels of airborne mercury which are liable to be more than 50% of the permissible exposure level and/or where there is significant risk of skin absorption and ingesting it.

4.2 CLINICAL EXAMINATION AND RELEVANT INVESTIGATIONS

- Clinical examination and baseline data with particular attention to:
  - Nervous system including effects to the central nervous system.
  - Gastrointestinal system
  - Respiratory system
  - Renal system

- Symptoms of weight loss, insomnia, and personality changes.
- Urinary mercury (total Hg) estimation (early morning specimen corrected for creati nine). Ensure employee avoids seafood for 3 days prior to urine collection.
- Renal function tests
- Baseline lung function test and chest X-ray (periodic examination does not require these tests, unless indicated).

4.3 PRE-PLACEMENT MEDICAL EXAMINATION

- Clinical Examination and Relevant Investigation as listed in 4.2.
- Baseline blood mercury.
  - The baseline may be influenced by diet. Interpretation of mercury in blood, require careful evaluation of all confounding factors.
- Baseline spirometry and chest X-ray.
  - These may be needed as a reference during medical assessment of post high exposure incidents.
- Decision for fitness to work:
  - Employees with disease of the central nervous, gastrointestinal, respiratory, and renal system should not work in areas where there is significant mercury exposure.

4.4 PERIODIC MEDICAL EXAMINATION

- Clinical Examination and Relevant Investigation as listed in 4.2.
- Frequency of periodic medical examination – annual.
- More frequent assessment when abnormalities are detected

4.5 BIOLOGICAL MONITORING.

Determinant	Sampling Time	BEL	Notes	Notation
Mercury in urine	Prior to shift	20 µg/g creatinine	Recommended for periodic assessment	-

Note: Employee with dental amalgams must be noted as it may contribute to urinary mercury levels.

- Additional Test

Determinant	Sampling Time	BEL	Notes
Blood total mercury	Not stated	15 µg/L	Indicate recent exposure to all types of mercury. Practical use is when suspicious of post high exposures incident.

Source: Safe Work Australia (2020)

Note:

1. Level of mercury in the urine is an indication of chronic exposure which is the average exposure for the past few months and not indicative of the mercury level at time of sampling (Safe Work Australia, 2020).
2. Level of mercury in the blood is not specific to occupational exposure as it can detect organic mercury exposure through diet. Employee's diet specifically seafood must be considered.

- Laboratory Method
  - Sampling Procedure

Mercury in Urine	
Container	Polyethylene or trace metal-free plastic container Note: *Request container from the laboratory *Own container can be used (provide at least one unit container to the lab for blank test).
Transport	Urine specimens should be refrigerated.
Stability	• Urine specimens are stable at room temperature for 5 days. • Urine specimens are stable for 14 days when refrigerated. • Urine specimens are stable for 30 days when frozen.
Volume	Requested volume: 30 mL Minimum volume: 10 mL Samples must be creatinine corrected.

Note:

The creatinine in the urine should be measured within 24 hours of sample collection.

Mercury in Blood	
Container	EDTA tube Note: *Request container from the laboratory. *Own container can be used (provide at least one unit container to the lab for blank test).
Transport	Blood specimens should be refrigerated.
Stability	● Blood specimens are stable for 30 days at room condition or when refrigerated. ● The specimens are stable at least for 3 months when frozen.
Volume	Requested volume: 7 ml Minimum volume: 2 ml

- Analytical equipment/procedure

Chemical	Detection Procedure
Mercury in urine	▪ Inductively Coupled Plasma/Mass Spectrometry (ICP/MS) ▪ Cold Vapor Atomic Aborption Spectrometry (CVAAS)
Creatinine	▪ Colorimetry
Mercury in blood	▪ Inductively Coupled Plasma/Mass Spectrometry (ICP/MS) ▪ Cold Vapor Atomic Aborption Spectrometry (CVAAS)

5.0 MEDICAL REMOVAL PROTECTION

- Indications for removal:
  - Clinical findings
    - Evidence of health effects that is suspected to be caused by CHTH or may be worsened by CHTH.
  - Target organ function abnormalities.
  - BEL
    - Exceed BEL
  - Pregnancy and breastfeeding
- All employees undergoing MRP should have repeat urine examinations (and relevant biochemical tests where indicated) once every 6 weeks with their health being monitored every 30 days until urine Hg level falls below the level (see below).
- Duration of the temporary MRP, based on the medical conditions, considering the expected duration of diagnostic procedures and the duration of recovery.

6.0 RETURN TO WORK

- Return to Work is based on:

  i. Biochemical results which include urine Hg level:

Categories	Level
Urine Hg level	<20 µg/g creatinine creatinine on two successive occasions.

  ii. Medical condition
    - No longer detected of having a medical condition.

  iii. Workplace management
    - Ensure workplace hygiene is safe and healthy and does not place the employee and increased risk of material impairment to health from expo sure to mercury.

7.0 NOTIFICATION TO DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed in NADOPOD 2004 using the relevant notification forms.

8.0 TREATMENT AND MANAGEMENT

- All cases of acute poisoning must be immediately removed from exposure and referred for hospital treatment. Contaminated areas of the body must be washed with soap and water.
- Chelation in the early stages e.g., Calcium EDTA; oral L-dopa reduces hypertonia, contractions, and speech disturbances.

9.0 PREVENTIVE MEASURES

- Women of reproductive age should not work in areas where there is significant Hg exposure (particularly alkyl Hg).
- Provide adequate and efficient ventilation. Air quality should be continually monitored.
- Mercury-related work areas should be isolated from other areas.
- Correct use of appropriate PPE like gloves, chemical goggles and breathing apparatus when required.
- Periodic medical surveillance of employees.
- Placing appropriate signages to inform of hazards.

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MINERAL OIL MIST, REFINED MINERAL

1.0 DESCRIPTION

CAS number: 8012-95-1

Mineral oil is a generic term used to group several petroleum derived liquids with “oil-like viscosity” manufactured by atmospheric and vacuum distillation (at temperatures between ~300ºC and ~700ºC) of crude oil and then further refined.

Unrefined crude oil is not used in the formulation of products in contact with the human body or in food related applications.

Employees at the oil drilling activities and oil and gas upstream activities may be exposed to the crude oil. Hazardous contents of the crude oil may include aromatic hydrocarbons, PAH, metallic and inorganic mercury, and other hazardous substances. Due to complexity of the chemical mixtures, the medical surveillance program of crude oil, or poorly refined mineral oil, is not explained in this document.

Commercially available Mineral oils differ in their physical chemical properties (e.g., viscosity) and chemical composition (e.g., aromatic content) and cannot thus be described with a single chemical formula.

The feedstock used in manufacturing mineral oil contains poly-aromatic hydrocarbons, some of which are classified as hazardous. However, these compounds are either removed using solvent extraction or converted using catalytic hydrotreatment to produce the refined mineral oils. The remaining aromatics found in the refined mineral oils are mainly 1-2 ring highly alkylated structures, which are not carcinogenic. From petroleum to mineral oils, white oils and waxes are chemically very inert substances.

What is thus used for instance, in the cosmetics, pharmaceutical or food contact, are highly refined specialty products derived from petroleum.

The use of these highly refined products has a very long history and enjoys an impeccable human safety record. In addition to offering interesting lubricating and moisture barrier properties, they are not allergenic.

This document only explains the medical surveillance program for Oil mist as regulated in the USECHH 2000 as Oil mist, mineral at 5 mg/m3 8-hour TWA.

1.1 SYNONYMS

- Not applicable

1.2 OCCUPATIONAL EXPOSURE LIMIT (OEL)

USECHH 2000 (Eight-hour time weighted average limit)	
Mineral Oil mist	5 mg/m^3 (TWA)

1.3 PHYSICOCHEMICAL PROPERTIES

- Mineral oil mist is a colourless, oily liquid aerosol dispersed in air with an odour like burned lubricating oil.

1.4 MATERIAL USE

- Used in cosmetics, pharmaceutical bases, food, and fibre production.
- Used primarily as lubricant base oils to produce further refined oil products.
- Used as carrier and bases.
- Used as solvent for inks in the painting industry.

2.0 TOXICITY

2.1 SOURCES OF POTENTIAL OCCUPATIONAL EXPOSURE

- Automotive industry
- Manufacturing industry
- Painting industry
- Mining
- Construction

2.2 ROUTE OF EXPOSURE

- Inhalation of the oil mist

2.3 TOXICOKINETICS

Absorption	Mineral oil readily partition into the uppermost cell layers of the stratum corneum.

Distribution	• Mineral oils ingested orally follow the route of intestinal resorption of dietary fats and resorbed by the small intestine primarily into the lymphatic system and to a lower extent into the liver portal vein. • A study on the lipid composition of human serum detected mineral oil in low- and high-density lipoprotein classes (LDL, HDL) and in the albumin-containing fraction. • In pharmacokinetic studies, mineral oil was undetectable in blood samples. Some studies showed mineral oil bioaccumulation in humans as a result of exposure to industrial-/technical- or food-grade mineral oils, which have been detected in fat, mesenteric lymph nodes, liver, and spleen, with lower levels in the lung, kidney, brain, and heart.
Metabolism and Excretion	• Metabolism is the dominant elimination process as there is no evidence for the excretion of non-metabolized alkanes via the urinary tract.

2.4 HAZARD CLASSIFICATION

- GHS Classification: to refer to each product classifications. Most refined mineral oil products are not classified as hazardous. This document refers to the oil mist that is formed in the breathing air of a workplace from refined mineral oil.

- SDS of a product should be the main source of information about the carcinogenicity because possible addition of hazardous chemicals or the mineral oil is less refined.

- Cancer Classification IARC
  - Not available.
  - Untreated and mildly treated mineral oils are carcinogenic to humans (Group 1) that does not apply to refined mineral oil. (IARC monograph mineral oil).

3.0 HEALTH EFFECTS MONITORING

3.1 ACUTE EFFECTS

SYSTEM/ORGAN	Acute Effects
Overexposures	**Adverse respiratory effects**
Respiratory	Irritations effects to the respiratory system.
Ear, Nose and Throat	• May irritate nose and throat. • Burning sensation to mouth and throat if swallowed. • Ringing in the ears

Eyes	• May cause severe irritation if not promptly removed
Skin	• Skin irritation. • Prolong exposure may cause dermatitis. • People with pre-existing skin disorders may be more susceptible.

3.2 CHRONIC EFFECTS

SYSTEM/ORGAN	Chronic Effects
Respiratory	• Obstructive airway disease
Skin	• Prolonged contact may cause skin irritation; acne-like rash may develop. • May cause skin allergy with itching and rash.

4.0 MEDICAL SURVEILLANCE PROGRAMME

4.1 INDICATIONS

Any work where employees are exposed to levels of airborne levels which are liable to be in excess of half the -in-air standard.

4.2 CLINICAL EXAMINATION AND RELEVANT INVESTIGATION

- Clinical examination with emphasis on respiratory system and skin diseases.
- Lung function test
- Chest X-ray (when indicated)

4.3 PRE-PLACEMENT MEDICAL EXAMINATION

- Clinical examination and relevant investigations as listed in 4.2.
- Decision for fitness to work and frequency of periodic medical examination:
  - Employees with disease of skin and respiratory system should not work in areas where there is significant mineral oil mist exposure.

4.4 PERIODIC MEDICAL EXAMINATION

- Clinical examination and relevant investigations as listed in 4.2.
- Frequency of periodic medical examination – annual.
- More frequent assessment when abnormalities are detected.

4.5 BIOLOGICAL MONITORING

- No biological exposure indices determinant based on ACGIH 2022 TLVs and BEIs.

5.0 MEDICAL REMOVAL PROTECTION

- Indications for removal:
  - Clinical findings
    - Evidence of health effects that is suspected to be caused by CHTH or may be worsened by CHTH.
  - Target organ function abnormalities.
  - Others:
    - Medical conditions of the skin and respiratory system.
- Temporary MRP
  - MRP based on medical condition.
    - The determination of the temporary MRP duration should consider the following aspects:
      - expected duration of diagnostic procedures
      - duration of recovery.
- All cases recommended for MRP, suspected cases of poisoning and excessive absorption must be notified to the DG of DOSH.

6.0 RETURN TO WORK

- Return to Work is based on:
  i. Medical condition
     - Normalised lung functions.

  ii. Workplace management
     - Ensure workplace hygiene is safe and healthy and does not place the employee at increased risk of material impairment to health from exposure to mineral oil mist.

7.0 NOTIFICATION TO DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed in NADOPOD 2004 using the relevant notification forms.

8.0 TREATMENT AND MANAGEMENT

- All cases of poisoning must be immediately removed from exposure and referred for hospital treatment.

9.0 PREVENTIVE MEASURES

- Adequate ventilation.
- Encourage personal hygiene.
- Appropriate signage.
- Use protective clothing in conditions of high exposures.
- Use particulate respirator R95 or P95.
- Use of barrier creams.
- Educate employees to report all early skin lesions.

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NAPHTHYLAMINE AND ITS ISOMERS: 1-NAPHTHYLAMINE

1.0 DESCRIPTION

1-Naphthylamine is an aromatic amine derived from naphthalene. It can cause bladder cancer (transitional cell carcinoma). It crystallises in colourless needles which melts at 50 °C. It possesses a disagreeable odour, sublimes readily, and turns brown on exposure to air. It is the precursor to a variety of dyes.

1.1 SYNONYMS

1-Aminonaphthalene; α-Naphthylamine.

1.2 OCCUPATIONAL EXPOSURE LIMIT (OEL)

USECHH 2000 (Eight-hour time weighted average limit)	
1-Naphthylamine	Not listed

Note:

- Not listed in USECHH Regulations 2000 or ACGIH TLV-TWA 2022.
- No numerical OELs have been established.
- No safe level of exposure for potential carcinogen.

1.3 PHYSICOCHEMICAL PROPERTIES

- Exists as white needle-like crystals which turn red on exposure to air.
- Has a weak ammonia-like odour.

1.4 MATERIAL USE

- Used as an intermediate in dye production.
- Used for manufacturing herbicides and antioxidants.
- Used in the manufacture of condensation colours, rubber, and in the synthesis of many chemicals (eg. α-naphthol, sodium naphthionate, o-naphthionic acid, Neville and Winther's acid, sulfonated naphthylamines, α-naphthylthiourea (a rodenticide), and N-phenyl- α-naphthylamine).

2.0 TOXICITY

2.1 SOURCES OF POTENTIAL OCCUPATIONAL EXPOSURE

- Dye manufacturing.
- Rubber manufacturing.

2.2 ROUTE OF EXPOSURE

- Inhalation (primary).
- Ingestion.
- Skin and/or eye contact.
- Percutaneous absorption.

2.3 TOXICOKINETICS

1-Naphthylamine	
Absorption and Distribution	• 1-Naphthylamine can be absorbed into the body by inhalation, through the skin and by ingestion
Metabolism	• 1-Naphthylamine was metabolized to 2-amino-1-naphthyl monophosphate ester and para-hydroxy conjugates. • The aromatic amines in the urine were metabolized but the active carcinogens of these aromatic amines are still to be found. • 1-naphthylamine is metabolized, by rats and other mammals, into 1-amino-2-naphthol and 1-amino-4-naphthol which are excreted in urine as glucuronide and sulfate conjugates, together with n-glucuronide, the n-sulfate, and somewhat unexpectedly, the n-glucoside of 1-naphthylamine.
Excretion and Half-life	• 1-Naphthylamine is partly oxidized to 1-amino-2-naphthol, which is eliminated in the urine but mainly as a glucuronide. 0.2% from 5.0 mg dose was shown to be excreted in the urine. A part of 1-naphthylamine is excreted unchanged. • Half-life: 13 hours

2.4 HAZARD CLASSIFICATION

Classification code	Hazard classification	H-Code	Signal
Acute Tox. 4 (oral)	Acute toxicity category 4 - oral	H302	Warning

Source: European Union, Commission Regulations (EU) 2018/669.

- Cancer Classification IARC
  - Group 1 (Bladder cancer)

3.0 HEALTH EFFECTS MONITORING

3.1 ACUTE EFFECTS

System/Organ	Acute Effects
Haematological	Methemoglobinemia
Renal & Genitourinary	• Dysuria • Haematuria

3.2 CHRONIC EFFECTS

System/Organ	Chronic Effects
Renal & Genitourinary	• Haemorrhagic cystitis • Bladder cancer
Skin	• Dermatitis • Skin cancer

4.0 MEDICAL SURVEILLANCE PROGRAMME

4.1 INDICATIONS

- Airborne exposures which are liable to be absorbed and/or where there is significant risk of ingesting it.

4.2 CLINICAL EXAMINATION AND RELEVANT INVESTIGATIONS

- Clinical examination with emphasis on renal genitourinary system and skin.
- Full blood count.
- Urine cytology

4.3 PRE-PLACEMENT MEDICAL EXAMINATION

- Clinical examination and relevant investigations as listed in 4.2.
- Decision for fitness to work:
    - Employees with disease of skin and renal & genitourinary systems should not work in areas where there is significant 1-Naphthylamine exposure.

4.4 PERIODIC MEDICAL EXAMINATION

- Clinical examination and relevant investigations as listed in 4.2.
- Frequency of periodic medical examination - annual.

4.5 BIOLOGICAL MONITORING

- No biological exposure indices determinant based on ACGIH 2022 TLVs and BEIs.

5.0 MEDICAL REMOVAL PROTECTION

- Indications for removal:
    - Clinical findings
        - Evidence of health effects that is suspected to be caused by CHTH or may be worsened by CHTH.
    - Target organ function abnormalities that is significant.
    - Pregnancy and breastfeeding.

6.0 RETURN TO WORK

- When to return to work based on:

i. Medical condition
    - Signs and symptoms have resolved and abnormal biochemical results have normalised after repeat of the urine examinations (and relevant biochemical tests where indicated)at **3-monthly intervals.**

ii. Workplace management
    - Ensure workplace hygiene is safe and healthy and does not place the employee at increased risk of material impairment to health from exposure to 1-Naphthylamine.

7.0 NOTIFICATION TO DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed in NADOPOD 2004 using the relevant notification forms.

8.0 TREATMENT AND MANAGEMENT

- All cases of poisoning must be immediately removed from exposure and referred for hospital treatment.
- Irrigate eyes with water.
- Wash contaminated areas of the body with soap and water.
- Gastric lavage, if ingested, followed by catharsis

9.0 PREVENTIVE MEASURES

- Adequate ventilation.
- Use of chemical goggles, rubber gloves.
- Appropriate signage.

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The European Commission. 2018. Commission Regulation (EU) 2018/669. Official Journal of the European Union, L 115.

NAPHTHYLAMINE AND ITS ISOMERS: β-NAPHTHYLAMINE

1.0 DESCRIPTION

1.1 SYNONYMS

2-Naphthylamine; 2-Aminonaphthalene.

1.2 OCCUPATIONAL EXPOSURE LIMIT (OEL)

USECHH 2000 (Eight-hour time weighted average limit)	
β-Naphthylamine	Not listed

1.3 PHYSICOCHEMICAL PROPERTIES

- β-Naphthylamine is a white to red crystal with a faint, aromatic odour.
- Darkens in air to a reddish-purple colour.

1.4 MATERIAL USE

- β-Naphthylamine is presently used only for research purposes. It is present as an impurity in α-naphthylamine.
- Used as an intermediate in the preparation of other compounds.
- β-Naphthylamine was widely used in the manufacture of dyestuffs, as an antioxidant fo rubber, and in rubber-coated cables.

2.0 TOXICITY

2.1 SOURCES OF POTENTIAL OCCUPATIONAL EXPOSURE

- Laboratory
- Rubber industry
- Dye industry

2.2 ROUTE OF EXPOSURE

- Inhalation (primary).
- Ingestion
- Skin and/or eye contact
- Percutaneous absorption

2.3 TOXICOKINETICS

β-Naphthylamine	
Absorption and Distribution	Dermal penetration of β-naphthylamine through human skin was studied by use of diffusion cells. It penetrates rapidly (lag time approximately 1.2 hours) and in high percentages (54% of the applied dose within 24 hours).
Metabolism	• Most of the absorbed β-Naphthylamine dose is excreted in the urine, in the form of free metabolites, metabolites conjugated to acids, and even in an unchanged form. • There was little oxidative metabolism of b-naphthylamine in human. In particular, N-hydroxy-2-naphthylamine, a proximate carcinogen of β-naphthylamine could not be detected. In contrast, large amounts of the acetylated metabolites, viz. N-acetylbenzidine, N,N-diacetylbenzidine and N-acetyl-2-naphthylamine were formed in human bladder cultures.
Excretion and Half-life	The excreted β-naphthylamine decomposes in the urine within a few hours.

2.4 HAZARD CLASSIFICATION

Classification code	Hazard classification	H-Code	Signal
Carc. 1A	Carcinogenicity category 1A	H350	Danger
Acute Tox. 4 *	Acute toxicity category 4 - oral	H302	

Source: European Union, Commission Regulations (EU) 2018/669.

- Cancer Classification IARC
  - Group 1 (Bladder cancer)

3.0 HEALTH EFFECTS MONITORING

3.1 ACUTE EFFECTS

System/Organ	Acute Effects
Eyes	Eyes irritation
Haematological	Methemoglobinemia
Renal & Genitourinary	Acute hemorrhagic cystitis

Skin	● Skin irritation ● Contact dermatitis

3.2 CHRONIC EFFECTS

System/Organ	Chronic Effects
Haematological	Methemoglobinemia
Nervous system	CNS - Ataxia
Renal & Genitourinary	● Dysuria ● Haematuria ● Hemorrhagic cystitis ● Bladder cancer
Skin	● Dermatitis

4.0 MEDICAL SURVEILLANCE PROGRAMME

4.1 INDICATIONS

- Any occupation where employees are liable to be exposed to β-Naphthylamine exceeding 50% of PEL and/or possibility of absorption.

4.2 CLINICAL EXAMINATION AND RELEVANT INVESTIGATIONS

- Clinical examination with emphasis on renal and genitourinary system, skin, and haematological system.
- Urine cytology
- Renal function test
- Full blood count

4.3 PRE-PLACEMENT MEDICAL EXAMINATION

- Clinical examination and relevant investigations as listed in 4.2.
- Decision for fitness to work:
    - Employees with disease of the renal and genitourinary system, skin and haematological system should not work in areas where there is significant β-Naphthylamine exposure.

4.4 PERIODIC MEDICAL EXAMINATION

- Clinical examination and relevant investigations as listed in 4.2.
- Frequency of periodic medical examination - annual.

4.5 BIOLOGICAL MONITORING

- No biological exposure indices determinant based on ACGIH 2022 TLVs and BEIs.

5.0 MEDICAL REMOVAL PROTECTION

- Indications for removal
  - Clinical findings
    - Evidence of health effects that is suspected to be caused by CHTH or may be worsened by CHTH.
  - Target organ function abnormalities that is significant.
  - Pregnancy and breastfeeding.

- All cases recommended for MRP, suspected cases of poisoning and excessive absorption must be notified to the DG of DOSH.

6.0 RETURN TO WORK

- When to return to work based on:

  i.Medical condition
  - Signs and symptoms have resolved and abnormal biochemical results have normalised after repeat of the urine examinations (and relevant biochemical tests where indicated)at **3-monthly intervals.**

  ii. Workplace management
  - Ensure workplace hygiene is safe and healthy and does not place the employee at increased risk of material impairment to health from exposure to β-Naphthylamine.

7.0 NOTIFICATION TO DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed in NADOPOD 2004 using the relevant notification forms.

8.0 TREATMENT AND MANAGEMENT

- All cases of poisoning must be immediately removed from exposure and referred for hospital treatment.
- Wash contaminated areas of the body with soap and water.

9.0 PREVENTIVE MEASURES

- Education of employees not to smoke as this chemical is found in cigarette smoke.
- Engineering control.
- Adequate ventilation.
- Approved PPE - any self-contained breathing apparatus with a full facepiece and operated in a pressure demand or positive pressure mode.
- Use of chemical goggles, mechanical filter respirator.
- Appropriate signage – CARCINOGEN.

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NICKEL

1.0 DESCRIPTION

Nickel is extracted from its sulphide ore by the Mond process. As part of this process nickel reacts with carbon monoxide to produce nickel carbonyl (a known carcinogen) as an intermediary in the production of nickel metal. Nickel is used in a range of steel alloys, including stainless steels. It is also used in nickel cadmium batteries, as a coating in electroplating and in ceramics. Exposure to nickel is usually by inhalation of dust and fumes from metal processing, grinding, and welding.

Nickel compounds can be grouped according to their solubility in water: Soluble compounds include nickel chloride, nickel sulfate, and nickel nitrate, and less-soluble compounds include nickel oxide and nickel subsulfide.

Nickel (soluble compounds) are defined to be compounds of nickel with solubility in water of greater than 0.1 moles per liter (mol/L) at 20ºC.

CHRA should classify the compounds into metal, insoluble, and soluble compounds, to help OHD determine the appropriate BEL.

1.1 SYNONYMS

- Nickel metal; elemental nickel; nickel catalyst; nickel subsulfide; raney nickel; Other synonyms vary depending upon the specific nickel compound.

1.2 OCCUPATIONAL EXPOSURE LIMIT (OEL)

USECHH 2000 (Eight-hour time weighted average limit)	
Elemental/Metal	1.5mg/m³ (IF)
Insoluble compounds, Ni (nickel oxide)	0.2 mg/m³ (IF)
Soluble compounds, Ni (include the sulfide, hydroxide, acetate, bromide, iodide, and nitrate)	0.1 mg/m³ (IF)
Nickel carbonyl (soluble)	0.05 ppm / 0.12 mg/m³
Nickel subsulfide (non soluble)	0.1 mg/m³ (IF)

1.3 PHYSICOCHEMICAL PROPERTIES

- Nickel metal is a lustrous, grey-white (silvery) metal, which is ductile, malleable, and with a fibrous structure.
- Insoluble in water and does not react with larger volumes of water.
- Odourless and excellent resistance to corrosion.

1.4 MATERIAL USE

- Nickel is used as an alloy additive in steel manufacture and in the production of coins and other utensils.
- Elemental nickel is used in electroplating, anodizing aluminium casting operations for machine parts; and in coinage; in the manufacture of acid-resisting and magnetic alloys; magnetic tapes; surgical and dental instruments; nickel-cadmium batteries; nickel soaps in crankcase oil; in ground-coat enamels; coloured ceramics; and glass.
- Elemental nickel also used as a catalyst in the hydrogenation synthesis of acrylic esters for plastics.
- Potential exposure by inhalation of dust and fume from metal processing, grinding, and welding as a by-product.

2.0 TOXICITY

2.1 SOURCES OF POTENTIAL OCCUPATIONAL EXPOSURE

- Coin and kitchen utensil manufacture
- Welding
- Manufacture of nickel cadmium batteries
- Pewter articles manufacture

2.2 ROUTE OF EXPOSURE

- Ingestion (primary)
- Inhalation
- Skin and eye absorption

2.3 TOXICOKINETICS

	Nickel
Absorption	• Higher concentrations of urinary nickel were found in employees exposed to soluble nickel compounds (nickel chloride, nickel sulfate) than in those exposed to less-soluble nickel compounds (nickel oxide, nickel subsulfide), indicating that the soluble compounds were more readily absorbed from the respiratory tract.

	• Human studies show that nickel can penetrate the skin, but it could not be determined whether the nickel had been absorbed into the deep layers of the skin or into the bloodstream.
Distribution	• Following inhalation exposure, about 20-35% of nickel deposited in the lungs of humans is absorbed into the bloodstream. The remainder is either swallowed, expectorated, or remains in the respiratory tract. • Post-mortem analysis of tissues from ten individuals who, with one exception, had no known occupational exposure to nickel, showed highest nickel concentrations in the lungs, thyroid gland, and adrenal gland, followed by lesser concentrations in the kidneys, heart, liver, brain, spleen, and pancreas. • The total amount of nickel found in the human body has been estimated as 6 mg or 86 µg/kg for a 70-kg person.
Metabolism	In human serum, nickel binds to albumin, L-histidine and α2-macroglobulin.
Excretion	• Absorbed nickel is excreted in the urine, regardless of the route of exposure. • Sweat constitutes another elimination route of nickel from the body. Nickel concentrations in sweat have been reported to be 10 to 20 times higher than concentrations in urine. • Hair is also an excretory tissue of nickel. However, use of hair as an internal exposure index has not gained wide acceptance due to problems associated with external surface contamination and non-standardized cleaning methods. • Nickel may also be excreted in human breast milk leading to dietary exposure of breast-fed infants.

2.4 HAZARD CLASSIFICATION

Nickel Compound	Classification code	Hazard Classification	H-Code	Signal
Nickel	Carc. 2	Carcinogenicity category 2	H351 (inh)	Danger
	STOT RE 1	Specific target organ toxicity – repeated exposure category 1	H372 (resp. system) (inh)	

	Skin Sens. 1	Skin sensitization category 1	H317	
Nickel subsulfide	Carc. 1A	Carcinogenicity category 1A	H350	Danger
	Muta. 2	Germ cell mutagenicity category 2	H341	
	STOT RE 1	Specific target organ toxicity, repeated exposure category 1	H372**	
	Skin Sens. 1	Sensitization, skin category 1	H317	
Cobalt dimolybdenum nickel octaoxide	Carc. 1A	Carcinogenicity category 1A	H350i	Danger
	STOT RE 1	Specific target organ toxicity, repeated exposure category 1	H372(a)	
	Skin Sens. 1	Sensitization, skin category 1	H317	
Cobalt nickel dioxide	Carc. 1A	Carcinogenicity category 1A	H350i	Danger
	STOT RE 1	Specific target organ toxicity, repeated exposure category 1	H372(a)	
	Skin Sens. 1	Sensitization, skin category 1	H317	
Cobalt nickel gray periclase; C.I. Pigment Black 25	Carc. 1A	Carcinogenicity category 1A	H350i	Danger
	STOT RE 1	Specific target organ toxicity, repeated exposure category 1	H372(a)	
	Skin Sens. 1	Sensitization, skin category 1	H317	
Cobalt nickel oxide	Carc. 1A	Carcinogenicity category 1A	H350i	Danger
	STOT RE 1	Specific target organ toxicity, repeated exposure category 1	H372(a)	
	Skin Sens. 1	Sensitization, skin category 1	H317	

Dialuminium nickel tetraoxide	Carc. 1A	Carcinogenicity category 1A	H350i	Danger
	STOT RE 1	Specific target organ toxicity, repeated exposure category 1	H372(a)	
	Skin Sens. 1	Sensitization, skin category 1	H317	
Diammonium nickel bis(sulfate)	Carc. 1A	Carcinogenicity category 1A	H350i	Danger
	Muta. 2	Germ cell mutagenicity category 2	H341	
	Repr. 1B	Reproductive toxicity category 1B	H360D	
	STOT RE 1	Specific target organ toxicity, repeated exposure category 1	H372(a)	
	Acute Tox. 4 (inh)	Acute toxicity category 4 (inhalation)	H332	
	Acute Tox. 4 (oral)	Acute toxicity category 4 (oral)	H302	
	Resp. Sens. 1	Respiratory sensitizatio category 1	H334	
	Skin Sens. 1	Sensitization, skin category 1	H317	
Diammonium nickel	Carc. 1A	Carcinogenicity category 1A	H350i	Danger
	STOT RE 1	Specific target organ toxicity, repeated exposure category 1	H372(a)	
	Resp. Sens. 1	Respiratory sensitizatio category 1	H334	
	Skin Sens. 1	Sensitization, skin category 1	H317	
Lithium nickel dioxide	Carc. 1A	Carcinogenicity category 1A	H350i	
	STOT RE 1	Specific target organ toxicity, repeated exposure category 1	H372(a)	

	Skin Sens. 1	Sensitization, skin category 1	H317	Danger

Source: ICOP on Chemicals Classification and Hazard Communication (Amendment) 2019.

- **Cancer Classification IARC**
  - Group 1

3.0 HEALTH EFFECTS MONITORING

Main target organs are the respiratory system and the skin. Irritations of the respiratory system are expected at exposures above the PEL.

3.1 ACUTE EFFECTS

System/Organ	Acute Effects
Eyes	Nickel dust and fumes can cause irritation of the eyes.
Respiratory	• Sensitizer by Inhalation • Fumes highly irritating to the respiratory tract. • Metal fume fever. • Lung allergy occasionally occurs with asthma-type effects. Fumes from heated nickel can cause **pneumonia-like illness** with cough and shortness of breath. • Higher exposures to fumes from heated nickel can cause **pulmonary edema**, a medical emergency that can be delayed for several hours or days. This can cause death.
Skin	• Nickel dusts and fumes can cause irritation of the skin. • Skin contact may cause sensitization, skin allergy, with itching, redness and later, rash.

3.2 CHRONIC EFFECTS

System/Organ	Acute Effects
Ear, Nose & Throat	• Breathing nickel dust and fumes can cause a sore or hole in the nasal septum.
Hepatobiliary	May affect liver function.

Renal and Genitourinary	May damage the kidneys.
Respiratory	• May cause respiratory sensitization and allergy. • May cause allergic asthma, pneumonitis. • Occupational exposure to nickel refinery dust contains nickel subsulfide, and is associated with lung cancer.
Skin	• Severe dermatitis and eczema via sensitisation. Sensitisation is permanent. • "Nickel itch" upon repeated exposure. Pink papular erythema of webs of fingers, which may spread to others • May cause skin sensitization and allergy.
Others	Nickel is a carcinogen and may damage the developing fetus.

4.0 MEDICAL SURVEILLANCE PROGRAMME

4.1 INDICATIONS

- Any work where employees are exposed to levels of airborne levels which are liable to be in excess of half the -in-air standard and/ or where there is significant risk of ingesting it.

4.2 CLINICAL EXAMINATION AND RELEVANT INVESTIGATIONS

- Clinical examination with emphasis on skin and respiratory system.
- Chest X-ray (if clinically indicated).
- Pulmonary function tests
- Liver function tests
- Urine test for nickel

4.3 PRE-PLACEMENT MEDICAL EXAMINATION

- Clinical Examination and Relevant Investigations as listed in 4.2.
- Decision for fitness to work and frequency of periodic medical examination:
    - Employees with diseases of skin and respiratory systems should not work in areas where there is significant nickel exposure.

4.4 PERIODIC MEDICAL EXAMINATION

- Clinical examination and relevant investigations as listed in 4.2.
- Chest X-ray (if clinically indicated) – 3 yearly.
- Frequency of periodic medical examination – annual.
- More frequent assessment when abnormalities are detected.

4.5 BIOLOGICAL MONITORING

Determinants	Sampling time	BEL	Notation
Nickel in urine after exposure to elemental Nickel and poorly soluble compounds.	Post-shift at end of workweek	5 µg/L	B
Nickel in urine after exposure to soluble compounds.		30 µg/L	-

- Nickel in urine is actually total nickel in urine, thus for the interpretation to the BEL, OHD need to refer to the type of nickel exposed.

- **Laboratory Methods**
  - Method reference: NMAM 8310
  - Sampling procedure

Nickel in Urine	
Container	Plastic container (Acid washed or Trace metal-free), Plastic container (preservative-free) Note: *Request container from the laboratory. *Own container can be used (provide at least one unit container to the lab for blank test).
Transportation	Urine specimens should be refrigerated.
Stability	• Urine specimens are stable at room temperature for 5 days. • Urine specimens are stable for 30 days when refrigerated. • Urine specimens are stable for 12 months when frozen.
Preservation	• Unpreserved urine should be refrigerated immediately and analysed within 1 week of collection. • Preserved urine should be refrigerated and analysed within 30 days of collection. • Acceptable preservatives include Trace Metal Free Hydrochloric Acid or Nitric Acid (0.1 mL of 12M acid/10 mL urine). • Avoid exposure to gadolinium-based contrast media for 48 hours prior to sample collection.
Volume	Requested volume: 30 mL Minimum volume: 10 mL

- **Analytical equipment/ procedure**
  Nickel
  - Inductively Coupled Plasma/Mass Spectrometry (ICP/MS).

5.0 MEDICAL REMOVAL PROTECTION

- Indications for removal
  - Clinical findings
    - Evidence of health effects that is suspected to be caused by CHTH or may be worsened by CHTH.
  - Target organ function abnormalities.
  - BEL
    - Exceed BEL
  - Pregnancy and breastfeeding.
  - Others:
    - Medical conditions of the skin, respiratory system, and nervous system.

- Temporary MRP
  - MRP based on medical condition.
    - The determination of the temporary MRP duration should consider the following aspects:
      - expected duration of diagnostic procedures.
      - expected duration of diagnostic procedures.
  - MRP based on BEL.
    - The determination of the temporary MRP duration should consider the following aspects:
      - Availability of the repeat BM sample results

- All cases recommended for MRP, suspected cases of poisoning and excessive absorption must be notified to the DG of DOSH.

6.0 RETURN TO WORK

- Return to work based on:
  i. BEL
     - Return to work when the results are below the BEL.
  ii. Medical condition
     - Signs and symptoms have resolved and abnormal biochemical results have normalised.
  iii. Workplace management
     - Ensure workplace hygiene is safe and healthy and does not place the employee at increased risk of material impairment to health from exposure

7.0 NOTIFICATION TO DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed in NADOPOD 2004 using the relevant notification forms.

8.0 TREATMENT AND MANAGEMENT

- All cases of poisoning must be immediately removed from exposure and referred for hospital treatment.
- Wash contaminated areas of the body with soap and water.
- Suggest the use of dimercaprol.

9.0 PREVENTIVE MEASURES

- Good Improvement in work-process.
- Prompt attention to all cutaneous wounds.
- Workplace hygiene.
- Adequate ventilation.
- Approved Personal Protective equipment. Chemical goggles.
- Appropriate signage.

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NITROBENZENE

1.0 DESCRIPTION

Nitrobenzene is a man-made chemical that is not found naturally in the environment. It is mainly used to produce other chemicals or to dissolve chemicals during manufacturing.

1.1 SYNONYMS

Essence of mirbane, essence of myrbane, mirbane oil, nitrobenzol, oil of mirbane, oil of myrbane.

1.2 OCCUPATIONAL EXPOSURE LIMIT (OEL)

USECHH 2000 (Eight-hour time weighted average limit)	
Nitrobenzene	1 ppm (5 mg/m^3) (TWA)

1.3 PHYSICOCHEMICAL PROPERTIES

- Pale yellow to dark brown oily liquid whose odour resembles bitter almonds.

1.4 MATERIAL USE

- Used in manufacture of aniline, polyurethane, dyes, drugs, pesticides, synthetic rubber and produce lubricating oils.

2.0 TOXICITY

2.1 SOURCES OF POTENTIAL OCCUPATIONAL EXPOSURE

- Nitrobenzene or nitrobenzene-related factories.

2.2 ROUTE OF EXPOSURE

- Inhalation (primary).
- Dermal absorption (primary).
- Ingestion

2.3 TOXICOKINETICS

Absorption	Readily absorbed following inhalation, ingestion, and dermal contact.

Absorption	Limited studies regarding oral and dermal exposure and distribution of nitrobenzene in humans. Studies have shown that 6-hour inhalation exposure of humans to nitrobenzene resulted in extensive pulmonary absorption.
Metabolism	• Reduction to form aniline and subsequent oxidation to form aminophenols which conjugate with glucuronide or sulfate. May also undergo oxidation to form nitrophentols which further conjugate with glucuronide or sulfate. • Primary urinary metabolites are p-aminophenol and p-nitrophenol.
Excretion	Predominantly in the urine and to a lesser extent in the faeces.There are limited studies on the estimated half-life of nitrobenzene in humans.

2.4 HAZARD CLASSIFICATION

Classification code	Hazard Classification	H-code	Signal
Carc.2	Carcinogenicity category 2	H351	Danger
Repr.1B	Reproductive toxicity category 1B	H360F	
Acute Tox. 3 (inh)	Acute toxicity category 3	H331	
Acute Tox. 3 (dermal)	Acute toxicity category 3 dermal	H311	
Acute Tox.3 (oral)	Acute toxicity category 3 oral	H301	
STOT RE 1	Specific target organ toxicity-repeated exposure category 1	H372 (blood)	

Source: ICOP on Chemicals Classification and Hazard Communication (Amendment) 2019.

- Cancer Classification IARC
  - Group 2B

3.0 HEALTH EFFECTS MONITORING

It is a primary skin irritant and can cause burns to the skin as well as ocular burns. Nitrobenzene is efficiently absorbed by the oral exposure route in potentially fatal doses.

Nitrobenzene inhalation causes headache, weakness, dizziness, loss of consciousness, and cyanosis when used as a paint stripper.

3.1 ACUTE EFFECTS

System/Organ	Acute Effects
Cardiovascular	Due to methemoglobinemia: • Rapid heartbeat • Cardiac arrhythmias
Ear, Nose and Throat	Burning of throat
Eye	• Irritation • Corneal damage
Gastrointestinal	Ingestion may result in: • Symptoms similar through inhalation exposure • Abdominal pain • Bloody diarrhoea
Haematological	Inhalation of nitrobenzene induces methemoglobinemia manifested by: • Blue coloration of lips, fingernails, and earlobes • Headaches • Dizziness • Loss of coordination • Laboured breathing • Rapid heartbeat • Low blood pressure • Cardiac arrhythmias • Vomiting • Coma and death Has a delayed effect of up to 4 hours.
Hepatobiliary	Enlarged spleen and liver via ingestion.
Nervous System CNS and PNS	• Headache • Lethargy • Weakness • Vertigo • Severe depression
Renal and Genitourinary	Urine may have a distinct almond odour.

Respiratory	• Inhalation of nitrobenzene at 3-6 ppm resulted in headache and nausea. • Inhalation of nitrobenzene at 40 ppm may result in intoxication. • Respiratory depression and failure. • Due to methemoglobinemia - laboured breathing
Skin	• Irritation • Allergic sensitization

3.2 CHRONIC EFFECTS

System/Organ	Chronic Effects
Ear, Nose and Throat	Thyroid cancer
Eyes	Vision disturbances
Haematological	Exposure to 40 ppm for 6 months has resulted in: • Liver and spleen damage • Liver cancer
Hepatobiliary	• Jaundice • Liver and spleen damage • Liver cancer
Nervous System CNS and PNS	As a result of methemoglobinemia: • Fatigue • Nerve damage
Renal and Genitourinary	Bladder distress
Reproductive	Reproductive toxicant in animal studies
Respiratory	Lung cancer
Skin	Skin allergy

4.0 MEDICAL SURVEILLANCE PROGRAMME

4.1 INDICATIONS

- Any occupation where employees are liable to be exposed to nitrobenzene exceeding 50% of the PEL and/or possibility of absorption.

4.2 CLINICAL EXAMINATION AND RELEVANT INVESTIGATIONS

- Clinical examination and baseline data with particular attention to detecting pre-existing abnormalities of the cardiovascular system, lungs, and blood.
- Urine examination.
- Blood test to detect:
  - Anaemia (Hb, haematocrit).
  - Blood Heinz bodies in severe poisoning.
- Cardiovascular system examination.
- Respiratory system examination.
- Blood methaemoglobin.

4.3 PRE-PLACEMENT MEDICAL EXAMINATION

- Clinical Examination and Relevant Investigations as listed in 4.2.
- Decision for fitness to work:
  - Persons susceptible are those suffering from hereditary haemoglobinopathies, congenital heart disease and chronic alcoholism.

4.4 PERIODIC MEDICAL EXAMINATION

- Clinical Examination and Relevant Investigations as listed in 4.2.
- Frequency of periodic medical examination - annual.
- More frequent assessment when abnormalities are detected.

4.5 BIOLOGICAL MONITORING

Determinant	Sampling time	BEL	Notation
Methaemoglobin in blood	During or end of shift	1.5% of total hemoglobin	B, Ns

- **Laboratory Procedure**
  - Sampling procedures

Example	
Container	EDTA or heparin tube **Note**: *Request container from the laboratory *Own container can be used (provide at least one unit container to the lab for blank test).

Transportation	Specimens should be refrigerated. Do not freeze.
Stability	• The methaemoglobin is not stable. • Specimens are stable for 1 hour at room temperature and up to 4 hours when refrigerated. • The validity of the result will be compromised if the analysis is not performed within 4 hours of sample collection.
Preservation	No specific preservative required.
Volume	Requested volume: 7 mL Minimum volume: 2 mL

- **Analytical equipment/procedure**

Methaemoglobin	CO-oximetry or pulse CO-oximetry. Derivative spectrophotometry via spectrophotometer or haematological analyser.

5.0 MEDICAL REMOVAL PROTECTION

- **Indications for removal**
  - Clinical findings
    - Evidence of health effects that is suspected to be caused by CHTH or may be worsened by CHTH.
  - Target organ function abnormalities.
  - BEL
    - Exceed BEL
  - Pregnancy and breastfeeding.
  - Others:
    - Medical conditions like hereditary haemoglobinopathies, congenital heart disease and chronic alcoholism.

- Duration of the temporary MRP, based on the medical conditions, considering the expected duration of diagnostic procedures and the duration of recovery.
- Duration of temporary MRP based on BEM, is the duration of the repeat BM sample to be available. Return to work when the results are below the BEL.
- All cases recommended for MRP, suspected cases of poisoning and excessive absorption must be notified to the DG of DOSH.
- All employees undergoing MRP should have repeat urine examinations (and relevant biochemical tests where indicated) at 3-monthly intervals

6.0 RETURN TO WORK

- **Return to work is based on:**

  i. Biochemical results
     - Returned to normal.

  ii. Medical condition
     - No longer detected of having a medical condition.

  iii. Workplace management
     - Ensure workplace hygiene is safe and healthy and does not place the employee and increased risk of material impairment to health from exposure to nitrobenzene.

7.0 NOTIFICATION TO DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed in NADOPOD 2004 using the relevant notification forms.

8.0 TREATMENT AND MANAGEMENT

- All nitrobenzene on the body must be removed immediately, remove, and discard all clothing, gloves, and footwear.
- Wash the whole body with soap and water.
- Pay special attention to hair, finger and toenails, nostrils, ear canal.
- All cases of poisoning must be immediately removed from exposure and referred for hospital treatment.
- Irrigate eyes with water.
- Wash contaminated areas of the body with soap and water.

9.0 PREVENTIVE MEASURES

- Good workplace and personal hygiene and safety.
- Approved personal protective equipment.

10.0 REFERENCES

ACGIH. 2022. TLVs® and BEIs® Threshold Limit Values for Chemical Substances and Physical Agents & Biological Exposure Indices. Ohio: American Conference of Governmental Industrial Hygienists.

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NITRO OR AMINO DERIVATIVES OF PHENOL AND DIPHENYL: ANILINE

1.0 DESCRIPTION

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1.1 SYNONYMS

Aminophen, Aminobenzene, Aniline oil, Anyvim, Arylamine, Benzeneamine, Blue oil, Phenylamine.

1.2 OCCUPATIONAL EXPOSURE LIMIT (OEL)

USECHH 2000 (Eight-hour time weighted average limit)	
Aniline and homologous (skin)	2ppm, 7.6 mg/m^3

1.3 PHYSICOCHEMICAL PROPERTIES

- Clear, colourless, oily liquid that darkens on exposure to light with a characteristic amine like odour.

1.4 MATERIAL USE

- Used as anti-knock agents and chemical intermediates for dye, polymer, and rubber production.
- Aniline is widely used as an intermediate in the synthesis of dyestuffs. It is also used in the manufacture of rubber accelerators and antioxidants, pharmaceuticals, marking inks; tetryl, optical whitening agents; photographic developers; resins, varnishes, perfumes shoe polishes, and many organic chemicals.

2.0 TOXICITY

2.1 SOURCES OF POTENTIAL OCCUPATIONAL EXPOSURE

- Rubber, polymer, and general industry.
- Dyes.

2.2 ROUTE OF EXPOSURE

- Inhalation (primary)
- Dermal absorption (primary)
- Ingestion

2.3 TOXICOKINETICS

Aniline	
Absorption	• There is limited information on toxicokinetic of aniline in humans. • Aniline can be absorbed through ingestion, inhalation, and skin absorption. • Dermal absorption in humans was estimated to amount up to 38%.
Metabolism	• Acetylation is required to detoxify the absorbed aniline. Lower activity of N-acetyltransferase will favor the formation of other metabolites (phenyl hydroxylamine, nitrosobenzene and aminophenol) which will lead to the formation of methaemoglobin.
Excretion	• After metabolic transformation the metabolites are predominantly excreted via urine where the major metabolites are glucuronide and sulfate conjugates of 4-hydroxyacetanilide.

2.4 HAZARD CLASSIFICATION

Classification code	Hazard classification	H-Code	Signal
Carc. 2	Carcinogenicity category 2	H351	Danger
Muta. 2	Germ cell mutagenicity category 2	H341	
Acute Tox. 3 (inh)	Acute toxicity, category 3 - inhalation	H331	
Acute Tox. 3 (dermal)	Acute toxicity category 3 - dermal	H311	
Acute Tox. 3 (oral)	Acute toxicity category 3 - oral	H301	
STOT RE 1	Specific target organ toxicity, single exposure category 2	H372 (a)	
Eye Dam. 1	Serious eye damage category 1	H318	
Skin Sens. 1	Sensitization, skin category 1	H317	

*(a) - State the target organ

Source: ICOP on Chemicals Classification and Hazard Communication (Amendment) 2019.

- Cancer Classification IARC
  - Group 2A (Bladder cancer).
  - Animal studies, strong mechanistic evidence

3.0 HEALTH EFFECTS MONITORING

Aniline has been known to cause bladder growths and bladder cancer with chronic exposure however with inadequate human studies. Acute exposure results in irritation of the mucous membranes including the eyes and respiratory tract as well as having a slight to moderate sensitizing effect on the skin.

Systemic acute effects include damage of the hemoglobin resulting in methemoglobinemia.

3.1 ACUTE EFFECTS

System/Organ	Acute Effects
Eyes	• Showed irritative effects on the mucous membranes of eyes.
Haematological	• Causes systemic effects include methemoglobinemia, tachypnea, dyspnea, nausea, vomit, hematuria and hemoglobinuria and anemia.
Renal and Genitourinary	• Urinary signs include painful urination, blood in the urine the presence of hemoglobin in the urine; and diminished amounts of urine.
Respiratory	• Acute inhalation exposure to high levels of aniline in humans has resulted in effects on the lung, such as upper respiratory tract irritation and congestion.
Skin	• Slight to moderate skin sensitizing potential and often induce allergic cross reaction.
Others	• Aniline has been classified as very toxic in humans, with a probable oral lethal dose in humans at 50 to 500 mg/kg body weight.

3.2 CHRONIC EFFECTS

System/Organ	Chronic Effects
Eyes	• Aniline is severely irritating to mucous membranes and affects the eyes. • Can cause systemic effects such as visual impairment.
Gastrointestinal	• Can cause systemic effects such as gastrointestinal problems.
Haematological	• Chronic exposure can cause methemoglobinemia, cyanosis, anemia, and formation of Heinz bodies.

Hepatobiliary	• Jaundice • Liver damage
Nervous System – CNS	• Can cause systemic effects such as headache, weakness, and fatigue.
Renal and Genitourinary	• Malignant bladder growths • Bladder cancer: (Signs and symptoms include blood in the urine, other changes in the appearance of the urine; changes in urinary habits; lumps in the groin and lower abdomen; and pain in the lower abdomen or back).
Respiratory	• Aniline is severely irritating to mucous membranes and affect the upper respiratory tract in humans.
Skin	• Aniline is severely irritating to mucous membranes and affects the skin. • Skin lesions.

4.0 MEDICAL SURVEILLANCE PROGRAMME

4.1 INDICATIONS

- Any work where employees are exposed to levels of airborne levels which are liable to be in excess of half the -in-air standard and/or where there is significant risk of ingesting it.

4.2 CLINICAL EXAMINATION AND RELEVANT INVESTIGATIONS

- Clinical examination with emphasis on the genitourinary, respiratory, and haematological system.
- Urine estimation (early morning specimen corrected to serum creatinine).
- Renal function test
- Liver function test
- Respiratory system examination.
- Full blood count

4.3 PRE-PLACEMENT MEDICAL EXAMINATION

- Clinical examination and relevant investigations as listed in 4.2.
- Decision for fitness to work:
  - Employees with disease of cardiovascular system, respiratory system and haematological system should not work in areas where there is significant aniline exposure.

4.4 PERIODIC MEDICAL EXAMINATION

- Clinical Examination and Relevant Investigations as listed in 4.2.
- Frequency of periodic medical examination - annual

4.5 BIOLOGICAL MONITORING

Determinant	Sampling time	BEL	Notation
Aniline in urine	End of shift	0.5 mg/L	-

- **Laboratory Method**
  - Sampling procedures

	Aniline in Urine
Container	Sealed plastic urine container without preservative (opaque type for p-aminophenol measurement). Note: *Request container from the laboratory. *Own container can be used (provide at least one unit container to the lab for blank test).
Transportation	Urine specimens should be refrigerated.
Stability	• Specimens are stable at room temperature for 24 hours. • Specimens are stable for 7 days when refrigerated. • Specimens are stable for 6 months when frozen.
Preservation	No specific preservation mentioned.
Sample volume	Requested volume: 30 mL Minimum volume: 10 mL

  - Analytical equipment/procedure

    Aniline

    - High Performance Liquid Chromatograph (HPLC) with electrochemical detection.

5.0 MEDICAL REMOVAL PROTECTION

- Indications for removal
    - Clinical findings
        - Evidence of health effects that is suspected to be caused by CHTH or may be worsened by CHTH.
    - Target organ function abnormalities.
    - BEL
        - Exceed BEL
    - Pregnancy and breastfeeding.
    - Others:
        - Medical conditions of cardiovascular system, respiratory system, and haematological system.

- Temporary MRP
    - MRP based on medical condition.
        - The determination of the temporary MRP duration should consider the following aspects:
            - expected duration of diagnostic procedures.
            - duration of recovery.
    - MRP based on BEL.
        - The determination of the temporary MRP duration should consider the following aspects:
            - Availability of the repeat BM sample results

6.0 RETURN TO WORK

- **Return to work based on:**

i. BEL
    - Return to work when the results are below the BEL.

ii. Medical condition
    - Signs and symptoms have resolved and abnormal biochemical results have normalised after repeat of the urine examinations (and relevant biochemical tests where indicated) at **3-monthly intervals.**

iii. Workplace management
    - Ensure workplace hygiene is safe and healthy and does not place the employee at increased risk of material impairment to health from exposure to

7.0 NOTIFICATION TO DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed in NADOPOD 2004 using the relevant notification forms.

8.0 TREATMENT AND MANAGEMENT

- All aniline on the body must be removed immediately, remove, and discard all clothing, gloves, and footwear.
- Wash the whole body with soap and water.
- Pay special attention to hair, finger and toenails, nostrils, ear canal.
- Determine methaemoglobin level every 3-6 hours for 18-24 hrs.
- Ascorbic acid (IV) and methylene blue have been used in severe cases.
- All cases of poisoning must be immediately removed from exposure and referred for hospital treatment.
- Irrigate eyes with water.
- Wash contaminated areas of the body with soap and water.
- Gastric lavage, if ingested, followed by catharsis.

9.0 PREVENTIVE MEASURES

- Adequate ventilation to control vapour.
- All employees should know how to recognise early signs of cyanosis.
- Skin contact must be avoided by use of impervious boot & gloves.
- Chemical goggles, mechanical filter respirator, rubber gloves.
- Appropriate signage.

10.0 REFERENCES

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NITRO OR AMINO DERIVATIVES OF PHENOL AND DIPHENYL: 4-NITRODIPHENYL

1.0 DESCRIPTION

1.1 SYNONYMS

p-Nitrodiphenyl; p-Nitrobiphenyl; 4-Nitrobiphenyl; 4-Phenylnitrobenzene; p-Phenylnitrobenzene, PNB.

1.2 OCCUPATIONAL EXPOSURE LIMIT (OEL)

USECHH 2000 (Eight-hour time weighted average limit)	
4-Nitrodiphenyl	Not listed

1.3 PHYSICOCHEMICAL PROPERTIES

- Exists as yellow plates or needles with a sweetish odour.

1.4 MATERIAL USE

- Used in the synthesis of 4-aminodiphenyl.
- Used only for research purposes. There are no commercial uses.
- Used as plasticizer, fungicide, wood preservative and dye intermediate.

2.0 TOXICITY

2.1 SOURCES OF POTENTIAL OCCUPATIONAL EXPOSURE

- Laboratory

2.2 ROUTE OF EXPOSURE

- Inhalation (primary)
- Ingestion
- Skin and/or eye contact
- Percutaneous absorption

2.3 TOXICOKINETICS

- Limited information on toxicokinetics in human and animal study.

2.4 HAZARD CLASSIFICATION

Classification code	Hazard Classification	H-Code	Signal
Carc. 1B	Carcinogenicity category 1B	H350	Danger

Source: European Union, Commission Regulations (EU) 2018/669.

- Cancer Classification IARC
  - Group 3 (Bladder cancer)

3.0 HEALTH EFFECTS MONITORING

3.1 ACUTE EFFECTS

System/Organ	Acute Effects
Haematological	• Methemoglobinemia
Hepatobiliary	• Liver damage
Nervous system - CNS	• Headache • Lethargy • Dizziness • Ataxia • Weakness • Drowsiness
Renal & Genitourinary	• Painful urination • Blood or pus in the urine • Acute hemorrhagic cystitis • Urinary burning

3.2 CHRONIC EFFECTS

System/Organ	Acute Effects
Hepatobiliary	• May cause liver damage.
Renal & Genitourinary	• Bladder cancer (in animal studies)
Respiratory	• Dyspnoea

4.0 MEDICAL SURVEILLANCE PROGRAMME

4.1 INDICATIONS

- Any occupational exposure to 4-Nitrodiphenyl exceeding 50% of the PEL and/or significant risk of airborne exposure and/or significant risk of ingestion.

4.2 CLINICAL EXAMINATION AND RELEVANT INVESTIGATIONS

- Clinical examination with emphasis on genitourinary system, kidneys, liver, and respiratory system.
- Urine cytology
- Renal function test
- Liver function test

4.3 PRE-PLACEMENT MEDICAL EXAMINATION

- Clinical Examination and Relevant Investigations as listed in 4.2.
- Decision for fitness to work:
  - Employees with disease of kidney, nervous system and respiratory system should not work in areas where there is significant 4-Nitrodiphenyl exposure.

4.4 PERIODIC MEDICAL EXAMINATION

- Clinical Examination and Relevant Investigations as listed in 4.2.
- Frequency of periodic medical examination - annual

4.5 BIOLOGICAL MONITORING

- No biological exposure indices determinant based on ACGIH 2022 TLVs and BEIs.

5.0 MEDICAL REMOVAL PROTECTION

- Indications for removal
  - Clinical findings
    - Evidence of health effects that is suspected to be caused by CHTH or may be worsened by CHTH.
  - Target organ function abnormalities that is significant.
  - Others:
    - Medical conditions of the kidney, nervous system, and respiratory system.

- Temporary MRP
  - MRP based on medical condition.
    - The determination of the temporary MRP duration should consider the following aspects:

- expected duration of diagnostic procedures.
- duration of recovery.

- All cases recommended for MRP, suspected cases of poisoning and excessive absorption must be notified to the DG of DOSH.

6.0 RETURN TO WORK

- Return to work based on:

i. Medical condition
   - Signs and symptoms have resolved and abnormal biochemical results have normalised after repeating the urine examinations (and relevant biochemical tests where indicated).

ii. Workplace management
   - Ensure workplace hygiene is safe and healthy and does not place the employee at increased risk of material impairment to health from exposure to health from exposure to 4-Nitrodiphenyl.

7.0 NOTIFICATION TO DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed in NADOPOD 2004 using the relevant notification forms.

8.0 TREATMENT AND MANAGEMENT

- All cases of poisoning must be immediately removed from exposure, provide appropriate first aid measures, and refer for hospital treatment.
- Wash contaminated areas of the body with soap and water.

9.0 PREVENTIVE MEASURES

- Improvement in work-process & workplace hygiene.
- Adequate ventilation.
- Personal Protective Equipment.
- Chemical goggles.
- Appropriate signage.

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NITRO OR AMINO DERIVATIVES OF PHENOL AND DIPHENYL: 4-AMINODIPHENYL

1.0 DESCRIPTION

1.1 SYNONYMS

4-Aminobiphenyl, p-Aminobiphenyl, p-Aminodiphenyl, 4-Phenylaniline. It is an aromatic amine.

1.2 OCCUPATIONAL EXPOSURE LIMIT (OEL)

USECHH 2000 (Eight-hour time weighted average limit)	
4-Aminodiphenyl	Not listed

1.3 PHYSICOCHEMICAL PROPERTIES

- 4-Aminodiphenyl is an aromatic amine.
- It exists as a colourless to tan crystalline solid.
- May change colour to purple when exposed to air.
- Combustible solid and has floral odour.
- Upon combustion, toxic gases are formed.

1.4 MATERIAL USE

Commonly used for research purposes and no longer used commercially (Sittig 2019).

- Organic chemical synthesis including solvents, perfume manufacture.
- Dyes and colorants.
- Manufacture of rubber chemicals.
- Contaminant in 2-aminobiphenyl.

2.0 TOXICITY

2.1 SOURCES OF POTENTIAL OCCUPATIONAL EXPOSURE

- Rubber chemical manufacturing.

2.2 ROUTE OF EXPOSURE

- Inhalation
- Percutaneous absorption

2.3 TOXICOKINETIC

Absorption	4-Aminodiphenyl is inhaled as dust and/or absorbed through the skin.
Distribution	Target organs are the bladder, skin, and blood
Metabolism	• Undergoes metabolism (N-oxidation) in the liver and bladder (O-acetylation) to active metabolite, N-hydroxy-4-aminobiphenyl and N-glucuronide-4-aminobiphenyl. • Can also be directly activated metabolically via peroxidative activation in the mammary glands and other organs. • Once it has transformed into its active metabolite, toxic effects occur to cellular DNA which contributes to its carcinogenicity. • N-glucuronide-4-aminobiphenyl metabolites are activated in acidic conditions when collected in urine.
Excretion	4-Aminodiphenyl is eliminated in the urine.

2.4 HAZARD CLASSIFICATION

Classification code	Hazard Classification	H-code	Signal
Carc. 1A	Carcinogenicity Category 1A	H350	Danger
Acute Tox. 4 *	Acute toxicity, oral Category 4	H302	

Source: European Union, Commission Regulations 2018/669.

- **Cancer Classification IARC**
  - Group 1 (Bladder cancer)

3.0 HEALTH EFFECTS MONITORING

Acute exposure to 4-aminodiphenyl may cause headaches, lethargy, cyanosis, urinary burning, and haematuria. 4-aminodiphenyl has been known to cause bladder cancer.

3.1 ACUTE EFFECTS

System/Organ	Acute Effects
Cardiovascular	Methemoglobinemia which inhibits oxygen transport in red blood cells. Symptoms include fast heart rate
Haematological	Methemoglobinemia which inhibits oxygen transport in red blood cells. Symptoms include cyanosis.

Nervous System CNS and PNS	Methemoglobinemia which inhibits oxygen transport in red blood cells. Symptoms include: ● Headaches ● Dizziness ● Lethargy or fatigue
Renal and Genitourinary	● Urinary burning ● Haematuria ● Acute haemorrhagic cystitis
Respiratory	● Difficulty breathing ● Collapse and death
Skin	Skin irritation

3.2 CHRONIC EFFECTS

System/Organ	Chronic Effects
Haematological	● Hyperaemia ● Edema
Hepatobiliary	● Liver effects
Nervous system CNS & PNS	● Nerve damage
Renal and Genitourinary	Bladder cancer Has a latent period of 15 to 35 years ● Exposure of only 133 days may cause bladder inflammation and tissue lesions.

4.0 MEDICAL SURVEILLANCE PROGRAMME

4.1 INDICATIONS

Employees exposed to 4-Aminodiphenyl or potential for significant irritation to the airway system and/or significant risk of skin absorption.

4.2 CLINICAL EXAMINATION AND RELEVANT INVESTIGATIONS

- Clinical examination and baseline data with particular emphasis on the renal and genitourinary system.
- Renal function

- Urine cytology
- Full blood picture

4.3 PRE-PLACEMENT MEDICAL EXAMINATION

- Clinical examination and relevant investigations as listed in 4.2.
- Decision for fitness to work:
  - Employees with disease of the renal and genitourinary, should not work in areas where there is significant 4-aminodiphenyl exposure.

4.4 PERIODIC MEDICAL EXAMINATION

- Clinical examination and relevant Investigations as listed in 4.2.
- Urine cytology: if there are red cells or positive smears, cystoscopy must be performed immediately.
- Frequency of periodic medical examination - annual but more frequent if exposure is high.

Note:
Potential exposure to other carcinogens, family history as well as tobacco, alcohol and medication use should be included in pre-placement and periodic examinations.

4.5 BIOLOGICAL MONITORING

- No biological exposure indices determinant based on ACGIH 2022 TLV & BEI.

5.0 MEDICAL REMOVAL PROTECTION

- Indications for removal

Categories	Level
Temporary MRP due to medical determination	
Cases of definite or suspected poisoning and excessive absorption.	All cases
Temporary MRP and referral to cytologist	
Urine sediment	Presence of red cells or positive smears

- Temporary MRP
  - MRP based on medical condition
    - The determination of the temporary MRP duration should consider the following aspects:
      - expected duration of diagnostic procedures
      - duration of recovery.
- All employees undergoing MRP should have repeat urine investigations and relevant biochemical tests within one month.

6.0 RETURN TO WORK

- **Return to work based on:**
  - i. Urine and biochemical test
    - Abnormal cytology and biochemical results have returned to normal.
  - ii. Medical condition
    - No longer detected of having a medical condition.
  - iii. Workplace management
    - Ensure workplace hygiene is safe and healthy and does not place the employee at increased risk of material impairment to health from exposure 4-aminodiphenyl.

7.0 NOTIFICATION TO DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed in NADOPOD 2004 using the relevant notification forms.

8.0 TREATMENT AND MANAGEMENT

- Immediate medical attention for eye exposure and inhalation. Removal from exposure and begin rescue breathing if required. Induce vomiting by providing employee who has ingested the chemical large amounts of water (do not induce vomiting for unconscious person).
- Immediately transfer to medical facility.

9.0 PREVENTIVE MEASURES

- Upon exit, employees should not carry clothing worn during handling with them. Shower and clean change of clothes.
- Employees known to be exposed should be supplied with appropriate personal protective equipment:
  - Protective gloves and clothing.
  - Splash or dust proof chemical goggles and face shield (unless full face-piece respiratory protection is worn).
  - Full-face supplied-air respirators of continuous-flow or pressure-demand type used for employees in handling operations.
- Engineering controls
  - Prohibit open vessel operations.

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ORGANOPHOSPHATES

1.0 DESCRIPTION

Organophosphates (OPs) are chemical substances originally produced by the reaction of alcohols and phosphoric acid. In the 1930s, organophosphates were used as insecticides, but the German military developed these substances as neurotoxins in World War II. They function as cholinesterase inhibitors, thereby affecting neuromuscular transmission.

Organophosphate insecticides, such as diazinon, chlorpyrifos, disulfoton, azinphos-methyl, and fonofos, have been used widely in agriculture and in household applications as pesticides.
Some examples of insecticides – malathion, parathion, diazinon, fenthion, dichlorvos, chlorpyrifos, ethion, methamidophos. monocrotophos.

Depending on the formulation, the Pesticide classifications could be Class 1a to the lesser toxicity Class 3. So, the acute toxicity through the oral route may differ from CLASS Hazard Cat 1 to 3.
All the organophosphates will affect the cholinesterase enzymes.

1.1 SYNONYMS

Examples are from 2 groups which have PELs

Dichlorvos
2,2-Dichlorovinyl dimethyl phosphate (DDVP), 2,2-Dichlorobinyldimethyl phosphate, 2,2-dichlorovinyl dimethyl ester phosphoric acid, 2,2-Dichloroethenol, dimethyl phosphate; dimethyl dichlorovinyl phosphate, dimethyl 2,2-dichloroethenyl phosphate; dimethyl 2,2-dichlorovinyl phosphate, O,O-dimethyl 2,2-dichlorovinyl phosphate, 2,2-dichloroethenyl dimethyl phosphate, phosphoric acid, 2,2-dichlorovinyl dimethyl ester.

Malathion
Butanedioic acid, (dimethoxyphosphinothioyl)thio-diethyl ester, Succinic acid, O,O-Dimethyldithiophosphate diethylmercaptosuccinate; O,O-Dimethyl S-(1,2-bis(ethoxycarbonyl) ethyl) dithiophosphate; S-1,2-bis(ethoxycarbonyl)ethyl-O,O-dimethyl thiophosphate.

1.2 OCCUPATIONAL EXPOSURE LIMIT (OEL)

USECHH 2000 (Eight-hour time weighted average limit)	
Dichlorvos	0.1 ppm, 0.90 mg/m^3 (TWA)
Malathion	10 mg/m^3 (TWA)

The list is for the groups. Other groups do not have a PEL yet. Care should be taken when conducting chemical exposure monitoring so that a proper method is selected for the chemical concerns, since there are many types of organophosphates in the market

1.3 PHYSICOCHEMICAL PROPERTIES

Dichlorvos

- Colourless to amber liquid with a mild aromatic odour.

Malathion

- Deep-brown to yellow liquid with a garlic-like odour.

1.4 MATERIAL USE

- Used as an agricultural insecticide.
- Used as an antihelmintic drugs (Dichlorvos).

2.0 TOXICITY

2.1 SOURCES OF POTENTIAL OCCUPATIONAL EXPOSURE

- Agricultural industry (as pesticides and antihelmintic agents).
- Public heath sectors (pest strips or sprays for insect control).
- General industry (manufacture, formulation and packing of pesticides).

2.2 ROUTE OF EXPOSURE

- Inhalation (primary)
- Skin absorption (primary)
- Ingestion
- Eye absorption

2.3 TOXICOKINETICS

- **Dichlorvos**

Absorption	• Readily absorbed through the skin, lungs, and GI tract. • Absorption through the skin contributes to systemic toxicity.
Distribution	In the human bloodstream, ester hydrolysis occurs rapidly (Half-Life = 7 to 11 minutes).
Metabolism	Once dichlorvos enters the body, it will be metabolized through ester hydrolysis and oxidative O-demethylation.

Excretion and Half-life	Dichlorvos will be excreted as urinary metabolites dimethylphosphate, demethyldichlorvos, urea and hippuric acid. The estimated half life of dichlorvos found in animal studies is 13.5 minutes in the kidney (WHO EHC 79., 1989)

Insufficient data on the toxicokinetic of malathion in human study. All organophosphate insecticides are able to be absorbed through the skin.

2.4 HAZARD CLASSIFICATION

	Classification code	Hazard Classification	H-code	Signal
Dichlorvos	Acute Tox. 2 (inh) (dermal)	Acute toxicity category 2 (inhalation) (dermal)	H330	Danger
	Acute Tox. 3	Acute toxicity category 3	H311	
	Acute Tox.3 (oral)	Acute toxicity category 3 (oral)	H301	
	Skin Sens. 1	Skin sensitization category 1	H317	
Malathion	Acute Tox. 4 (oral)	Acute toxicity category 4 (oral)	H302	Warning
	Skin Sens. 1	Skin sensitization category 1	H317	

Source: ICOP on Chemicals Classification and Hazard Communication 2019.
*Refer to SDS for other type of organophosphates.

- Cancer Classification IARC
  - Dichlorvos: Group 2B
  - Malathion: Group 3

3.0 HEALTH EFFECTS MONITORING

Health effects are mainly due to inhibition of the cholinesterase enzymes. So, the main effects are the nervous system depressions. Examples below are for Dichlorvos and Malathion only.

3.1 ACUTE EFFECTS

- **Dichlorvos**

System/Organ	Acute Effects
Cardiovascular	• Low blood pressure • High levels of exposure may result in cardiac dysfunction.

Eyes	• Irritation of the eyes manifested as: o Miosis o Aching eyes o Rhinorrhoea • Lacrimation • Impaired vision
Gastrointestinal	• Salivation • Anorexia • Nausea • Vomiting • Diarrhoea
Haematological	• Cyanosis • Act as irreversible acetylcholinesterase inhibitor and exerts haematotoxic effects.
Musculoskeletal	• Muscle fasciculation • Ataxia
Nervous System CNS and PNS	• Headache • Giddiness • Convulsions • Drowsiness • Fatigue • Act as irreversible acetylcholinesterase inhibitor and exerts neurotoxic effects which may be fatal. • High levels of exposure may result in: o Weakness o Tremor o Paralysis o Confusion o Slurred speech o Convulsions o Coma
Respiratory	• Chest tightness • Wheezing • Laryngeal spasm High levels of exposure may result in an inability to breath.
Skin	Irritation of the skin
Others	Perspiration

- **Malathion**

System/Organ	Acute Effects
Cardiovascular	High level of exposure may cause low blood pressure.
Eyes	Irritation and discomfort of the eyes. Impaired vision
Gastrointestinal	• High level of exposure may cause: o Nausea o Vomiting o Abdominal pain o Diarrhoea
Haematological	Acetylcholinesterase inhibitor resulting in haematotoxic effects.
Nervous System CNS and PNS	• High level of exposure may cause: o Headache o Paralysis o Muscle spasms o Loss of reflexes o Convulsions o Coma
Respiratory	Cholinergic hyperstimulation resulting in respiratory disorders such as wheezing.
Skin	Irritation of the skin

3.2 CHRONIC EFFECTS

- **Dichlorvos**

System/Organ	Chronic Effects
Haematological	Animal studies indicate exposure may cause mononuclear cell leukemia.
Hepatobiliary	Animal studies indicate exposure may cause cancer of pancreas.
Nervous System - CNS and PNS	Cholinesterase inhibitor causing similar symptoms as acute effects.
Reproductive	In animal studies, dichlorvos demonstrated neurodevelopmental effects (decreased brain weight and altered nerve electrophysiology during prenatal and/or lactational exposure).
Respiratory	Animal studies indicate exposure may cause esophageal squamous cell carcinoma.
Skin	• Skin sensitization • Dermatitis

- **Malathion**

System/Organ	Chronic Effects
Nervous System - CNS and PNS	• Nerve damage resulting in: ○ Weakness ○ Dizziness ○ Poor coordination in arms and legs • Personality changes: ○ Depression ○ Anxiety or ○ Irritability
Skin	Skin irritation Skin sensitization
Others	Genetic effects

Recognizing acute poisoning (for all types of OP)

- Increased salivation, lacrimation, perspiration. Miosis (pinpoint and non-reactive) ptosis, blurring of vision, conjunctival injection, 'bloody tears'.
- Nausea, vomiting, abdominal tightness, swelling and cramps, diarrhoea, tenesmus, faecal incontinence.
- Excessive bronchial secretions, rhinorrhoea, wheezing, oedema, tightness in chest, bronchospasms, bronchoconstriction, cough, bradypnoea, dyspnoea.
- Bradycardia, decrease in blood pressure.
- Urinary frequency and incontinence.
- Tachycardia, pallor, increase in blood pressure.
- Muscle fasciculations (eyelids, fine facial muscles), cramps, diminished tendon reflexes, generalised muscle weakness in peripheral and respiratory muscles, paralysis, flaccid or rigid tone.
- Restlessness, generalised motor activity,
- Reaction to acoustic stimuli, tremulousness, emotional lability, ataxia.
- Drowsiness, lethargy, fatigue, mental confusion, inability to concentrate, headache, pressure in head, generalised weakness.
- Coma with absence of reflexes, tremors, Cheyne-Stokes respiration, dyspnoea, convulsions, depression of respiratory centres, cyanosis.

4.0 MEDICAL SURVEILLANCE PROGRAMME

4.1 INDICATIONS

Any occupation where employees are liable to be exposed to dichlorvos & malathion exceeding 50% of the PEL and/or possibility of skin absorption. All agricultural usage posed a significant risk of skin absorptions and recommended for Medical Surveillance Program.

4.2 CLINICAL EXAMINATION AND RELEVANT INVESTIGATIONS

4.2.1. EMPLOYEE SELECTION

All employees directly exposed to the chemical should be selected. Training on the chemical safety, safe procedures, the equipment used for the handling and the use of PPE is the main control measures. To ensure a good control and good surveillance, job rotation is not recommended, unless it is among the employees included in the medical surveillance program.

The program relies mainly on the BEM. Plasma samples may not give a true indication of the cholinesterase level if sample collection is delayed after the last exposure has occurred.
In the case of minor poisoning, if there is a delay in sample collection of more than 48 hours, then the subject's serum cholinesterase may increase to its normal level. However, the erythrocyte cholinesterase activity would still be inhibited, and this is the parameter that should be measured.

4.2.2. TYPE OF TESTS

- **Clinical examination with emphasis on the central and autonomic nervous system and skin.**

  - BEM
    - **Baseline and periodic monitoring of OP depends on its pesticide classification:**
      - **OP Pesticide Class 1 and 2**
      - **OP Pesticide class 3**
    - Involves monitoring of plasma cholinesterase and/or erythrocyte cholinesterase (RBC AChE).
    - Plasma cholinesterase estimation reflects recent exposure preceding two to three weeks while RBC AChE estimation reflects exposure within the 120 days which is the life of the red blood cell.
  - Baseline data
    - Baselines require both RBC AChE and Plasma AChE.
  - Periodic
    - Periodic samples may proceed with Plasma AChE.
    - Retest with RBC AChE, whenever there are symptoms and signs of poisoning or MRP is required based on the Plasma level.
    - Comparison of RBC samples should be done with RBC baseline samples. Similar rule applies to plasma samples.

- **Determination of baseline and periodic monitoring**

	Pre-placement Baseline	Working Baseline (when 30 days free of exposure is not possible)	Frequency of periodic monitoring
OP Class 1 and 2	• Average of two baseline taken three days apart. • Free of exposure for 30 days.	• Two samples taken one week apart. • Result should not be different by 10%. If more, a third sample must be taken. • Must be free from exposure between the samples. • The highest value should be used as the baseline.	A frequency of monthly examination and testing is recommended.
OP Class 3	• One baseline • Free of exposure for 30 days		Annually

*If pre-placement level is not available, use lower limit of the laboratory reference range as baseline for comparison or previous results (whichever is higher).

4.5 BIOLOGICAL MONITORING

- **Biological effect monitoring**

Determinants	Sampling time	BEL
Plasma AChE (screening)	End of shift	60% of individual's baseline activity*
RBC AChE		70% of individual's baseline activity*

Source: ACGIH TLV and BEI 2019

- **Laboratory Methods**
  - Method reference: DHHS (NIOSH) Publication Number 77-106
  - Sampling procedure

Blood for Acetylcholinesterase	
Container	EDTA tube for plasma AChE. Heparin tube for RBC AChE.
Transportation	Specimens should be refrigerated.
Stability	Specimens are stable for 1 week either at room temperature, refrigerated or frozen.
Preservation	No specific preservative mentioned.
Volume	Requested volume: 7ml Minimum volume: 2ml

  - Analytical equipment/procedure

AChE activity via thiocholine production.	• Spectrophotometry • Ellman colorimetric assay

5.0 MEDICAL REMOVAL PROTECTION

- **In the presence of any of the following.**
- **Indications for MRP are similar for all classes of OP.**

Temporary MRP based on AChE levels	
Plasma AChE	≤ 60% of the baseline
RBC AChE	≤ 70% of the baseline
Temporary MRP due to medical determination	
Cases of definite or suspected poisoning and excessive absorption.	All cases

- All employees undergoing MRP should have repeat plasma AChE measurement at monthly intervals.
- They should not return to work until the AChE level has returned to within the return level (see below) and any related signs and symptoms have disappeared.
- Temporary removal includes removal from work with carbamates.

6.0 RETURN TO WORK

- Indications for RTW are similar for all classes of OP.
- Return to work based on:

i. AChE levels

Category	Level
Plasma AChE levels	>80% of individual's baseline level
RBC AChE levels	Above individual's baseline level

ii. Medical condition
   - No longer detected having any medical conditions.

iii. Workplace management
   - Ensure workplace hygiene is safe and healthy and does not place the employee at increased risk of material impairment to health from exposure to organophosphate.

7.0 NOTIFICATION TO DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed in NADO POD 2004 using the relevant notification forms.

8.0 TREATMENT AND MANAGEMENT

- All cases of acute poisoning must be immediately removed from exposure, provided appropriate first aid measures, and refer for hospital treatment.
- Treatment based on specific medical conditions arising from exposure to the chemical.

9.0 PREVENTIVE MEASURES

- Good workplace and personal hygiene and safety.
- Approved personal protective equipment.
- Pesticide application course for all the pesticide applicators

10.0 REFERENCES

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ORTHOTOLIDINE

1.0 DESCRIPTION

1.1 SYNONYMS

O-Tolidine, 3,3-Dimethylbenzidine, 4,4-Bianisidine, 2-tolidine, Diaminoditolyl, Diaminotolyl.

1.2 OCCUPATIONAL EXPOSURE LIMIT (OEL)

USECHH Regulation 2000 (Eight-hour time weighted average limit)	
Orthotolidine	Not listed

Note:

- Not listed in USECHH Regulations 2000 or ACGIH TLV-TWA 2022.
- No numerical OELs have been established.
- No safe level of exposure for potential carcinogen.

1.3 PHYSICOCHEMICAL PROPERTIES

- White to reddish crystalline solid.

1.4 MATERIAL USE

- Used as chemical intermediate for azo dyes, pigments, rubber products and pesticide production.

2.0 TOXICITY

2.1 SOURCES OF POTENTIAL OCCUPATIONAL EXPOSURE

- Pigments and dyes manufacturing (azo dyes and pigments).
- Intermediate in rubber products and pesticides.
- Medical laboratories work where orthotolidine is used to detect occult blood and blood in urine.

- Production of polyurethane-based high-strength elastomers, coatings, and rigid plastics.
- Analytical industry (gold detection).
- Used as pool test kits to measure total chlorine.

2.2 ROUTE OF EXPOSURE

- Skin absorption (primary)
- Inhalation
- Ingestion
- Eye absorption

2.3 TOXICOKINETICS

Absorption	• Enters the body via dermal absorption.
Distribution	• Orthotolidine does not accumulate extensively in the tissues.
Metabolism	• Evidence of azo reduction by azoreductase in intestinal bacteria to release benzidine congeners and several other metabolites like monoacetyl- and diacetylbenzidine and their congeners.
Excretion	• Rapid metabolism and excretion in urine and feces. Studies on the half-life in humans is limited.

2.4 HAZARD CLASSIFICATION

Classification Code	Hazard Classification	H-Code
Carc. 1B	Carcinogenicity Category 1B	H350
Acute Tox. 4 *	Acute toxicity, oral Category 4	H302

Source: European Union, Commission Regulations (EU) 2018/669.

- **Cancer Classification IARC**

  Group 1 (Urinary bladder cancer)

3.0 HEALTH EFFECTS MONITORING

Chronic exposure to orthotolidine has been associated with increased incidence of bladder cancer. Orthotolidine may be absorbed through the skin.

3.1 ACUTE EFFECTS

System/Organ	Acute Effects
Ear, Nose and Throat	• Nasal irritation • Inhalation of small amounts can cause sneezing spasms followed by upper respiratory tract irritation.
Eye	• Eye irritation
Haematological	• High level of exposure causes haematuria.
Renal and Genitourinary	• Blood effects
Respiratory	• Inhalation of small amounts can cause sneezing spasms followed by upper respiratory tract irritation.
Skin	• Skin irritation and redness • Blue skin
Others	• Blue lips or fingernails.

3.2 CHRONIC EFFECTS

System/Organ	Acute Effects
Hepatobiliary	• Exposed animals developed liver cancer
Renal and Genitourinary	• Increased risk of bladder cancer • Exposed animals developed kidney damage and kidney cancer
Reproductive	• Animal studies indicate exposure associated with mammary gland tumours • Teratogenic and genotoxic potential • Trigger formation of transplacental tumours
Skin	• Dermatitis

4.0 MEDICAL SURVEILLANCE PROGRAMME

4.1 INDICATIONS

- Any occupation where employees are liable to be exposed to orthotolidine exceeding 50% of the PEL and/or possibility of absorption.

4.2 CLINICAL EXAMINATION AND RELEVANT INVESTIGATION

- Clinical examination with particular attention to the skin, renal and genitourinary, respiratory system.
- Renal function test
- Full blood count
- Urine cytology

4.3 PRE-PLACEMENT MEDICAL EXAMINATION

- Clinical examination and relevant investigations as listed in 4.2.
- Decision for fitness to work:
    - Employee with disease of the skin, renal and genitourinary and respiratory system should not work in areas where there is significant orthotolidine exposure.

4.4 PERIODIC MEDICAL EXAMINATION

- Clinical examination and relevant investigations as listed in 4.2.
- Frequency of periodic medical examination - annual
- More frequent assessment when abnormalities are detected.

4.5 BIOLOGICAL MONITORING

- No established biological monitoring is available based on ACGIH 2022 TLVs® and BEIs®.

5.0 MEDICAL REMOVAL PROTECTION

- Indications for removal
    - Clinical findings:
        - Evidence of health effects that is suspected to be caused by CHTH or may be worsened by CHTH.
    - Target organ function abnormalities.
    - Pregnancy and breastfeeding.
    - Others:
        - Medical conditions of skin, renal and genitourinary and respiratory system.
- Duration of the temporary MRP, based on the medical conditions, considering the expected duration of diagnostic procedures and the duration of recovery.
- All cases recommended for MRP, suspected cases of poisoning and excessive absorption must be notified to the DG of DOSH.
- All employees undergoing MRP should have repeat urine examinations (and relevant biochemical tests where indicated) at 3-monthly intervals.

6.0 RETURN TO WORK

- Return to work based on:
  - i. Urine examination and other relevant biochemical test
    - ○ Abnormal results have returned to normal.
  - ii. Medical condition
    - ○ No longer detected of having a medical condition.
  - iii. Workplace management
    - ○ Ensure workplace hygiene is safe and healthy and does not place the employee at increased risk of material impairment to health from exposure to orthotolidine and its salts.

7.0 NOTIFICATION TO DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed in NADOPOD 2004 using the relevant notification forms.

8.0 TREATMENT AND MANAGEMENT

- All cases of acute poisoning must be immediately removed from exposure, provide appropriate first aid measures, and refer for hospital treatment.
- Treatment based on specific medical conditions arising from exposure to the chemical.

9.0 PREVENTIVE MEASURES

- Good workplace and personal hygiene and safety.
- Approved personal protective equipment.

10.0 REFERENCES

ACGIH. 2022. TLVs® and BEIs® Threshold Limit Values for Chemical Substances and Physical Agents & Biological Exposure Indices. Ohio: American Conference of Governmental Industrial Hygienists.

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PITCH, TAR, BITUMEN & CREOSOTE

1.0 DESCRIPTION

The bitumen is the binding material that is present in asphalt. It is also sometimes called mineral tar. It is obtained by partial distillation of crude petroleum.

It is chemically a hydrocarbon. It is insoluble in water, but it completely dissolves in carbon disulfide chloroform, alkalies, alkaline, carbonates, petroleum spirit, and oil of turpentine.

Tar is a viscous black liquid made of hydrocarbons that can form in multiple ways. Because of this, the chemical composition of tar varies, though it is always made of organic matter of some sort.

It has many uses as a waterproofing and sealing agent. It is also used for many medicinal purposes.

Pitch is a viscoelastic polymer which can be natural or manufactured, derived from petroleum, coal tar, or plants. Various forms of pitch may also be called tar, bitumen, or asphalt.

Creosote is a category of carbonaceous chemicals formed by the distillation of various tars and pyrolysis of plant-derived material, such as wood or fossil fuel. They are typically used as preservatives or antiseptics.

1.1 SYNONYMS

- Coal tar pitch, black oil

1.2 OCCUPATIONAL EXPOSURE LIMIT (OEL)

USECHH 2000 (Eight-hour time weighted average limit)	
Coal tar pitch volatiles, as benzene solubles.	0.2 mg/m^3 (TWA)

1.3 PHYSICOCHEMICAL PROPERTIES

- Thick dark bituminous mixture and have a tarry odour.

1.4 MATERIAL USE

- Manufacture of pitch, tar bitumen and creosote.

- Waterproofing of wood, making of roofing and insulating materials.
- Lining irrigation canals and reservoirs.
- Road surfacing.
- Lubricant for die moulds.
- Manufacture of dyestuff.
- Base for coating, paints.
- Chemical feedstock for the production of benzene, toluene, xylene, phenol.
- Sealing agents e.g., in battery manufacture.

2.0 TOXICITY

2.1 SOURCES OF POTENTIAL OCCUPATIONAL EXPOSURE

- Construction
- Railway
- Briquette manufacturing
- Paint industry

2.2 ROUTE OF EXPOSURE

- Inhalation (primary)
- Skin
- Ingestion

2.3 TOXICOKINETICS

Pitch, Tar, Bitumen & Creosote	
Absorption	• Coal-tar pitch can enter the body through lungs, intestine, skin.
Distribution	• Coal tar products are composed of hydrocarbons. Thus, they are likely to distribute to lipid-rich tissues. • Coal tar creosote is also likely to distribute to the liver as evidenced by the presence of metabolites in the urine, indicating microsomal enzyme induction.
Metabolism	• Coal tar components may be metabolised, and some may store in body fat and breast milk.
Excretion	• Most of the coal-tar pitch will be excreted in faeces with small amounts found in urine.

2.4 HAZARD CLASSIFICATION

Classification Code	Hazard Classification	H-Code	Signal
Carc. 1B	Carcinogenicity category 1B	H350	Danger

Source: European Union, Commission Regulations (EU) 2018/669.

- **Cancer Classification IARC**
  - Group 1

3.0 HEALTH EFFECTS MONITORING

Symptoms of acute health effects are signs of over exposure conditions. Main target organs are the respiratory system and the skin.

3.1 ACUTE EFFECTS

System/Organ	Acute Effects
Ear, Nose and Throat	• Eyes - blepharoconjunctivitis, keratitis, eye irritation.
Respiratory	• Respiratory tract irritation
Skin	• Skin burns • Skin irritation • Photosensitive reactions of previously exposed areas of skin.

3.2 CHRONIC EFFECTS

System/Organ	Chronic Effects
Gastrointestinal	• Burning pain • Diarrhoea
Respiratory	• Irritation - congestion, pneumonitis. • Mild to moderate pulmonary restriction and obstruction. • Squamous cell and/or oat cell carcinoma (evidence still uncertain). Due to residues of coal tar distillation, 3-4-benzpyrene, 1,2,5,6-dibenzanthracene.

Skin	• Irritation erythema, burning, itching, followed by desquamation (aggravated by sunlight) • Pigmentation changes – hyperpigmentation (primarily forearms, wrists, hands, scrotum) • Follicular dermatitis (comedones, acne, sebaceous cysts) • Benign neoplasms - coarsening and hardening (shagreen appearance), kerato-acanthoma, tar warts or papillomata (tar warts may be premalignant). • Malignant neoplasms - epithelioma (usually after 20 years of exposure. Common sites are head, neck, scrotum, and upper limbs).

4.0 MEDICAL SURVEILLANCE PROGRAMME

4.1 INDICATIONS

- Any occupation where employees are exposed to pitch, tar, bitumen, and creosote above 50% of PEL and/or possibility of skin absorption.

4.2 CLINICAL EXAMINATION AND RELEVANT INVESTIGATIONS

- Clinical examination with emphasis on skin, lungs, gastrointestinal system and bladder (within 3 months of exposure).
- Skin biopsy when indicated (specialist referral).
- Chest X-ray.

4.3 PRE-PLACEMENT MEDICAL EXAMINATION

- Clinical examination and relevant investigations as listed in 4.2.
- Decision for fitness to work and frequency of periodic medical examination:
    - Employees with disease of skin, lungs, gastrointestinal system, and bladder should not work in areas where there is significant pitch, tar, bitumen, and creosote.

4.4 PERIODIC MEDICAL EXAMINATION

- Clinical examination and relevant investigations as listed in 4.2.
- Chest X-ray should be less than once a year. E.g., once in 2 years.
- Frequency of periodic medical examination – annual.
- Regular skin examination of the skin annually ensures early detection of pre-cancerous lesions and their treatment before cancer can develop. But frequency will depend on exposure levels and symptoms and signs.

4.5 BIOLOGICAL MONITORING

- No biological exposure indices determinant based on ACGIH 2022 TLVs and BEIs.

5.0 MEDICAL REMOVAL PROTECTION

- Indications for removal
  - Clinical findings
    - Evidence of health effects that is suspected to be caused by CHTH or may be worsened by CHTH.
    - Any medical conditions of the skin, lungs, GIT, and bladder.
  - Target organ: abnormalities of chest X-ray and skin biopsies suspected or confirmed are due to the chemical.
- Temporary MRP
  - MRP based on medical condition.
    - The determination of the temporary MRP duration should consider the following aspects:
      - expected duration of diagnostic procedures.
      - duration of recovery.
- All cases recommended for MRP, suspected cases of poisoning and excessive absorption must be notified to the DG of DOSH.
- All medically removed employees should have repeat investigations and relevant biochemical tests within one month.

6.0 RETURN TO WORK

- Return to work based on:
  - i. Medical condition
    - No longer detected of signs and symptoms and abnormal cytology/ biochemical results have returned to normal.
  - ii. Workplace management
    - Ensure workplace hygiene is safe and healthy and does not place the employee at increased risk of material impairment to health from exposure to pitch, tar, bitumen & creosote.

7.0 NOTIFICATIONS TO DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed in NADOPOD 2004 using the relevant notification forms.

8.0 TREATMENT AND MANAGEMENT

- All cases of acute poisoning must be immediately removed from exposure, provide appropriate first aid measures, and refer for hospital treatment.
- All cases with cancer must be referred to the appropriate specialist for treatment immediately.
- Treatment based on specific medical conditions arising from exposure to the chemical.

9.0 PREVENTIVE MEASURES

- Good workplace and personal hygiene and safety.
- Approved personal protective equipment.
- Inform all women that they are to inform the supervisor as soon as they are found to be pregnant. All pregnant women are to be suspended from exposure to coal tar, coal tar pitch, and coal tar creosote.
- Employees at risk should be made aware of the dangers of the substances they are handling.
- Employees should be encouraged to examine their skin regularly and report any suspicious lesions to the OHD.
- Change the underclothes and working clothes often.
- Avoid putting any tools or other materials contaminated with pitch and tar in trouser pockets.
- Wash hands before going to the toilet.
- Make sure to have a bath or shower after work.

10.0 REFERENCES

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POTASSIUM OR SODIUM CHROMATE OR DICHROMATE

1.0 DESCRIPTION

These are inorganic compounds and chromate salts. It belongs to hexavalent chromium VI compounds.

Chromium and Chromate are different compounds. Chromium is represented as Cr and Chromate is represented as Cr04.

1.1 SYNONYMS

- Sodium chromate cas: 7775-11-3
- Sodium Dichromate cas: 10588-01-9
- Potassium Chromate cas: 7789-00-6
- Potassium Dichromate, cas: 7778-50-9

Potassium chromate

- Bipotassium chromate, chromate of potass, dipotassium chromate, neutral potassium chromate, potassium chromate (VI).

Sodium chromate

- Chromate of soda, chromium disodium oxide, chromium sodium oxide, disodium chromate, neutral sodium chromate, sodium chromate (VI).

Potassium dichromate

- Dichromic acid, dipotassium salt, potassium bichromate; dipotassium dichromate (VI).

Sodium dichromate

- Disodium dichromate (VI), dichromic acid, disodium salt, disodium dichromium heptaoxide.

1.2 OCCUPATIONAL EXPOSURE LIMITS (OEL)

USECHH 2000 (Eight-hour time weighted average limit)	
Chromium and inorganic compounds, as Cr	0.5 mg/m^3
Water soluble Cr VI compounds, NOC	0.05 mg/m^3

1.3 PHYSICOCHEMICAL PROPERTIES

Potassium chromate

- Yellow odourless crystal.

Sodium chromate

- Yellow odourless crystal.

Potassium dichromate

- Orange-red odourless solid.

Sodium dichromate

- Red to bright orange odourless crystal.

1.4 MATERIAL USE

- Used in photography, chrome plating, leather tanning and pigmentation for textile, paints, inks, and plastics.
- Used in water samples analysis in laboratories.

2.0 TOXICITY

2.1 SOURCES OF POTENTIAL OCCUPATIONAL EXPOSURE

- Photography printing.
- Construction (improve the texture of cement).
- Used in water samples analysis in laboratories.

2.2 ROUTE OF EXPOSURE

- Inhalation (primary)
- Ingestion
- Skin and eye absorption

2.3 TOXICOKINETIC

Potassium or Sodium Chromate or Dichromate	
Absorption	• Chromium (VI) is efficiently absorbed through the skin.
Distribution	• Once deposited in the lungs, chromium (VI) is generally transferred to the systemic circulation. • Chromium compounds are widely distributed in the body, with a greater distribution reported there is greater tendency of chromium (VI) to cross plasma membranes.
Metabolism	• Chromium (VI) is unstable in the body and is reduced to chromium (V), chromium (IV), and ultimately to chromium (III) by endogenous substances such as ascorbate and glutathione and it is believed that the toxicity of chromium may result from damage to cellular components during this process (e.g., through the generation of free radicals).
Excretion & Half-life	• In humans, absorbed chromium is excreted primarily via urine. • The half-life for elimination of chromium when given as potassium chromate (0.05 mg chromium (VI) kg-1 in drinking water) is estimated to be approximately 35-40 hours.

2.4 HAZARD CLASSIFICATION

Potassium chromate			
Classification Code	**Hazard Classification**	**H-Code**	**Signal**
Carc. 1B	Carcinogenicity category 1B	H350i	Danger
Muta. 1B	Germ cell mutagenicity category 1B	H340	
Eye Irrit. 2	Eye irritation category 2	H319	
STOT SE 3	Specific target organ toxicity, single exposure category 3; Respiratory tract irritation	H335	

Skin Irrit. 2	Skin irritation category 2	H315	Danger
Skin Sens. 1	Skin sensitization category 1	H317	

Source: ICOP on Chemicals Classification and Hazard Communication 2019.

Sodium chromate			
Classification Code	**Hazard Classification**	**H-Code**	**Signal**
Carc. 1B	Carcinogenicity category 1B	H350	Danger
Muta. 1B	Germ cell mutagenicity category 1B	H340	
Acute Tox. 2 (inh)	Acute toxicity category 2 - inhalation	H330	
Acute Tox. 3 (oral)	Acute toxicity category 3 - oral	H301	
Acute Tox. 4 (dermal)	Acute toxicity category 4 - dermal	H312	
STOT RE 1	Specific target organ toxicity, repeated exposure category 1	H372 (resp. system, kidney)	
Skin Corr. 1B	Skin corrosion category 1B	H314	
Eye Dam. 1	Serious eye damage or eye irritation category 1	H318	
Resp. Sens. 1	Sensitization, respiratory category 1	H334	
Skin Sens. 1	Sensitization, skin category 1	H317	

Source: ICOP on Chemicals Classification and Hazard Communication 2019.

Potassium or sodium dichromate			
Classification code	**Hazard classification**	**H-Code**	**Signal**
Ox. Sol. 2	Oxidizing solids category 2	H272	Danger
Carc. 1B	Carcinogenicity category 1B	H350	
Muta. 1B	Germ cell mutagenicity category 1B	H340	
Repr. 1B	Reproductive toxicity category 1B	H360FD	
Acute Tox. 2 (inh)	Acute toxicity category 2 - inhalation	H330	
Acute Tox. 3 (oral)	Acute toxicity category 3 - oral	H301	
STOT RE 1	Specific target organ toxicity, repeated exposure category 1	H372**	
Acute Tox. 4 (dermal)	Acute toxicity category 4 - dermal	H312	
Skin Corr. 1B	Skin corrosion category 1B	H314	

Eye Dam. 1	Serious eye damage or eye irritation category 1	H318	
Resp. Sens. 1	Sensitization, respiratory category 1	H334	
Skin Sens. 1	Sensitization, skin category 1	H317	

Source: ICOP on Chemicals Classification and Hazard Communication 2019.

- **Cancer Classification IARC**
  - Group 1 (Lung cancer)

3.0 HEALTH EFFECTS MONITORING

The main target organ is the nasopharynx and the respiratory system.

3.1 ACUTE EFFECTS

System/Organ	Acute Effects
Cardiovascular	• Chest pain
Ear, Nose and Throat	• Chronic rhinitis • Cough
Eye	• Blepharospasm • Lacrimation • Conjunctivitis • Palpebral oedema • Photophobia
Gastrointestinal	• Abdominal pain • Haematemesis • Bloody diarrhoea with circulatory collapse.

Respiratory	● Irritation to the respiratory tract ● Pharyngitis ● Laryngitis
Skin	● Dermal ulcers ● Dermatitis

3.2 CHRONIC EFFECTS

System/Organ	Acute Effects
Ear, Nose and Throat	● Nasal septal ulceration and perforation.
Gastrointestinal	● Occupational exposure may cause gastrointestinal effects.
Haematological	● Occupational exposure may cause haematological effects.
Hepatobiliary	● Occupational exposure may cause hepatic effects.
Renal and Genitourinary	● Occupational exposure may cause renal effects.
Respiratory	● Respiratory irritation and inflammation ● Dyspnoea ● Cyanosis ● Occupational exposure increased the risk of lung cancers and sinonasal cancer in lesser extent.
Skin	● Dermal contact can lead to allergic dermatitis and chronic dermal exposure can result in deeply penetrating skin ulcers if left untreated.

4.0 MEDICAL SURVEILLANCE PROGRAMME

4.1 INDICATIONS

- Employers must provide medical surveillance for the employees who are:
    - Exposed or may be exposed to Chromium (VI) at concentrations at or above/ 50% of the PEL or evidence of health effects.

- Exposed or may be exposed to Chromium (VI) at concentrations at or above/ 50% of the PEL or evidence of health effects.

4.2 CLINICAL EXAMINATION AND RELEVANT INVESTIGATIONS

- Clinical examination with emphasis on respiratory system and skin.
- Detect pre-existing allergies.
- Examination of nose and skin.
- Full blood count.

4.3 PRE-PLACEMENT MEDICAL EXAMINATION

- Clinical examination and relevant investigations as listed in 4.2.
- Decision for fitness to work and frequency of periodic medical examination:
  - Employees with disease of skin and respiratory system should not work in areas where there is significant potassium chromate/sodium chromate/potassium dichromate/sodium dichromate exposure.

4.4 PERIODIC MEDICAL EXAMINATION

- Clinical examination and relevant investigations as listed in 4.2.
- Frequency of periodic medical examination when normal findings: annual.

4.5 BIOLOGICAL MONITORING

Determinants	Sampling time	BEL	Notation
Total chromium in urine	End of shift at the end of workweek	0.7 µg/L	Pop

Note:
Determinant being tested is the total chromium in urine. Total chromium in urine consists of organic chromium (III) and inorganic chromium (VI). Interpretation without unexpose baseline will be a challenge in assessing chromium (VI).

- Laboratory Method
  - Method reference: NMAM 8310
  - Sampling procedure

Chromium in Urine	
Container	Polyethylene or plastic container. Note: *Request container from the laboratory. *Own container can be used (provide at least one unit container to the lab for blank tests).
Transportation	Specimens should be transported at 2°C - 8°C.
Stability	● Urine specimens are stable at room temperature for 5 days. ● Urine specimens are stable for 1 month when refrigerated or frozen
Preservation	No specific preservative mentioned.
Sample volume	Requested volume: 30 mL Minimum volume: 10 mL

○ Analytical equipment/procedure

Chromium
- Atomic absorption spectrometry.
- Inductively Coupled Plasma Mass Spectrometry (ICP-MS).

5.0 MEDICAL REMOVAL PROTECTION

● **Indications for removal**

o Clinical findings:
- Evidence of health effects that is suspected to be caused by CHTH or may be worsened by CHTH.

o Target organ function abnormalities

o BEL
- Exceed BEL

o Pregnancy and breastfeeding.

● Duration of the temporary MRP, based on the medical conditions, considering the expected duration of diagnostic procedures and the duration of recovery.

- Duration of temporary MRP based on BM, is the duration of the repeat BM sample to be available. Return to work when the result is below the BEL.

- All cases recommended for MRP, suspected cases of poisoning and excessive absorption must be notified to the DG of DOSH.

- All suspended cases should have repeat urine examinations (and relevant biochemical tests where indicated) at 3-monthly intervals.

6.0 RETURN TO WORK

- Return to work based on:
  - i. BEL
    - ○ Return to work when the results are below the BEL.
  - ii. Medical condition
    - o Level of urine for total chromium does not exceed the normal reading.
  - iii. Workplace management
    - ○ Ensure workplace hygiene is safe and healthy and does not place the employee at increased risk of material impairment to health from exposure to potassium chromate/sodium chromate/ potassium dichromate/sodium dichromate.

7.0 NOTIFICATION TO DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed in NADOPOD 2004 using the relevant notification forms.

8.0 TREATMENT AND MANAGEMENT

- All cases of acute poisoning must be immediately removed from exposure, provide appropriate first aid measures, and refer for hospital treatment.
- Treatment based on specific medical conditions arising from exposure to the chemical.

9.0 PREVENTIVE MEASURES

- Good workplace and personal hygiene and safety.
- Approved personal protective equipment.

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SILICA (CRYSTALLINE)

1.0 DESCRIPTION

Workers are exposed to respirable crystalline silica, also known as silica dust, in a variety of industries, including construction, mining, oil and gas extraction, stone countertop fabrication, foundries and other manufacturing settings. Silica dust is made up of small particles, more importantly the respirable form, that become airborne during various work activities including cutting, drilling, chipping, sanding, or grinding materials that contain crystalline silica. These materials can include sand, concrete, brick, block, stone, and mortar.

Crystalline silica is found in soil, sand, concrete, mortar, granite, other minerals, and artificial stone. The most common form of crystalline silica is quartz; however, it can also occur in the form of cristobalite and tridymite. Exposure to cristobalite typically occurs in foundries where the intense heat of molten metal causes cristobalite to be formed in clay molds.

Silicosis, an irreversible but preventable lung disease, is caused by inhalation of respirable silica dust. Work exposures to silica dust also cause other serious diseases, including lung cancer.

1.1 SYNONYMS

- **Cristobalite**: calcined diatomite, silica, cristobalite, silica, crystalline-cristobalite.
- **Quartz**: Agate, Amethyst, Chalcedony, Cherts, Flint, Onyx, Pure quartz, Quartz, Rose quartz, Sand, Silica flour (powdered crystalline silica), Silicic anhydride.
- **Tridymite**: Christensenite, Silica, crystalline-tridymite, α-Tridymite, Tridymite 118.

1.2 OCCUPATIONAL EXPOSURE LIMITS (OEL)

USECHH 2000 (Eight-hour time weighted average limit)		
Silica - Crystalline	Cristobalite	0.05 mg/m³ (TWA)
	Quartz	0.1 mg/m³ (TWA)
	Tridymite	0.05 mg/m³ (TWA)
Silica - Amorphous	Inhalable particulate	0.05 mg/m³ (TWA)
	Respirable particulate	10 mg/m³ (TWA)

	Precipitated silica	10 mg/m³ (TWA)
	Silica, fume	2 mg/m³ (TWA))
	Silica, fused	0.1 mg/m³ (TWA)
	Silica gel	10 mg/m³ (TWA)

1.3 PHYSICOCHEMICAL PROPERTIES

- Silicon dioxide/crystalline silica is a component of many mineral dusts and materials which melt into glass at very high temperatures.

1.4 MATERIAL USE

- When workers cut, drill, chip, sand, or grind materials that contain crystalline silica, hazardous levels of respirable crystalline silica dust can be released into the air that workers breathe.

2.0 TOXICITY

2.1 SOURCES OF POTENTIAL OCCUPATIONAL EXPOSURE

- Construction (concrete-grinding) and mining.
- Granite quarrying, cutting, and processing industry (stone crushing).
- Glass and clay manufacturing.
- Foundry industry (moulding and grinding).
- Sand blasting.

2.2 ROUTE OF EXPOSURE:

- Inhalation (primary)
- Skin absorption
- Ingestion

2.3 TOXICOKINETICS

Silica	
Absorption	• Inhaled silica particles that deposit in the respiratory tract are subject to three general distribution processes:

	1. bronchial and tracheal mucociliary transport to the gastrointestinal tract; 2. transport to thoracic lymph nodes (e.g., lung, tracheobronchial, mediastinal); or 3. absorption by blood and/or lymph and transfer to other tissues (e.g., peripheral lymph tissues, kidney). • The above processes apply to all forms of deposited silica, although the relative contributions of each pathway and rates associated with each pathway vary with the physical characteristics (e.g., particle size) and biological reactivity (e.g., macrophage recruitment, activation, and cytotoxicity).
Distribution	• When silica dusts are inhaled, most of them are expelled, but some can become lodged in the lungs and remain there throughout life. • Part of them is cleared by mucociliary action and swallowed and remaining silica in the lungs will be cleared by inflammatory cells. This will cause localized inflammation, tissue injury and epithelial proliferation. • Silica particles may distribute to the kidney tissue following inhalation exposure. • Inhalation exposure of rats to silica shows distribution primarily to mediastinal lymph nodes and thymus. Silica particles were detected in in negligible amounts in the blood, kidney, liver, and spleen.
Metabolism	• Absorbed silica is not metabolized. Although c-silica particles are highly insoluble, in vitro studies have found that silica particles dissolved from slate dust can bind to serum albumin.
Excretion	• Urine is an excretory pathway for silica absorbed from the respiratory tract. • Ingested silica is excreted in the faeces.

	• Studies of excretion of silica following dermal exposures have not been reported.

2.4 HAZARD CLASSIFICATION

Best to refer to the SDS of respective compounds.

- **Cancer Classification IARC**
  - Group 1

3.0 HEALTH EFFECTS MONITORING

It is important to be aware that silica exposure can also cause lung cancer, chronic kidney disease, various autoimmune diseases, and can predispose exposed individuals to pulmonary tuberculosis (TB).

Irritation effects of the eye and the respiratory symptoms can be significant and a sign of overexposures. Progressive signs of inflammation of the respiratory system causes coughing, phlegm and breathing difficulties that can be detected by careful history taking.

As lung tissue turns into scar tissue with silicosis, reduced lung function occurs and gets progressively worse, even after dust exposure has ended. Symptoms typically include shortness of breath, cough, wheezing, and exercise intolerance. The time from initial exposure to when symptoms first appear (the latency period) with silicosis depends on duration and intensity of exposure. Higher exposures tend to result in shorter latency periods and faster disease development. The table below describes different types of silicosis. The type with the quickest onset is acute silicosis, which can occur after only a few weeks or months following exposure to very high levels of respirable crystalline silica. In acute silicosis, affected parts of the lung fill with fluid, typically causing severe illness or death. Accelerated silicosis occurs after high levels of exposure and typically presents after 5 to 10 years. Chronic silicosis is the most common type and occurs after 10 or more years of exposure to lower levels of silica than those that cause accelerated silicosis.

Accelerated and chronic silicosis have the same radiographic appearance and are differentiated based on their different latency periods.

Silicosis			
Type of silicosis	**Exposure level**	**Latency period**	**Severity of illness**
Acute	Very high	Weeks to months	Severe morbidity and mortality are common.

Accelerated	High	5 to 10 years	Variable, often severe
Chronic	Lower than accelerated	More than 10 years	Variable, mild to severe

Chronic Silicosis

- Chronic silicosis is the most common presentation of silicosis and usually occurs after at least 10 years of exposure to respirable crystalline silica. The clinical presentation of chronic silicosis is:
    - Symptoms - shortness of breath and cough, although employees may not notice any symptoms early in the disease. Constitutional symptoms, such as fever, loss of appetite and fatigue, may indicate other diseases associated with silica exposure, such as TB infection or lung cancer. Employees with these symptoms should immediately receive further evaluation and treatment.
    - Physical examination - may be normal or disclose dry rales or rhonchi on lung auscultation.
    - Spirometry - may be normal or may show only a mild restrictive or obstructive pattern.
    - Chest X-ray - classic findings are small, rounded opacities in the upper lung fields bilaterally. However, small irregular opacities and opacities in other lung areas can also occur. Rarely, “eggshell calcifications” in the hilar and mediastinal lymph nodes are seen.
    - Clinical course - chronic silicosis in most cases is a slowly progressive disease.

Accelerated Silicosis

- Accelerated silicosis generally occurs within 5-10 years of exposure and results from high levels of exposure to respirable crystalline silica. The clinical presentation of accelerated silicosis is:
    - Symptoms - shortness of breath, cough, and sometimes sputum production. Employees with exposure to respirable crystalline silica, and especially those with accelerated silicosis, are at high risk for activation of TB infections, atypical mycobacterial infections, and fungal superinfections. Constitutional symptoms, such as fever, weight loss, hemoptysis (coughing up blood), and fatigue may herald one of these infections or the onset of lung cancer.
    - Physical examination - rales, rhonchi, or other abnormal lung findings in relation to illnesses present. Clubbing of the digits, signs of heart failure, and cor pulmonale may be present in severe lung disease.
      Spirometry - restrictive or mixed restrictive/obstructive pattern.
    - Chest X-ray - small rounded and/or irregular opacities bilaterally. Large opacities and lung abscesses may indicate infections, lung cancer, or progression to complicated silicosis, also termed progressive massive fibrosis.
    - Clinical course - accelerated silicosis has a rapid, severe course.

Acute Silicosis

- Acute silicosis is a rare disease caused by inhalation of extremely high levels of respirable crystalline silica particles. The pathology is similar to alveolar proteinosis with lipoproteinaceous material accumulating in the alveoli. Acute silicosis develops rapidly, often, within a few months to less than 2 years of exposure and is almost always fatal. The clinical presentation of acute silicosis is as follows:
    - Symptoms - sudden, progressive, and severe shortness of breath. Constitutional symptoms are frequently present and include fever, weight loss, fatigue, productive cough, hemoptysis (coughing up blood), and pleuritic chest pain.
    - Physical examination - dyspnea at rest, cyanosis, decreased breath sounds, inspiratory rales, clubbing of the digits, and fever.
    - Spirometry - restrictive or mixed restrictive/obstructive pattern.
    - Chest X-ray - diffuse haziness of the lungs bilaterally early in the disease. As the disease progresses, the “ground glass” appearance of interstitial fibrosis will appear.
    - Clinical course - employees with acute silicosis are at especially high risk of TB activation, nontuberculous mycobacterial infections, and fungal superinfections. Acute silicosis is immediately life-threatening.

Kidney and Autoimmune disease

- Exposure to silica has been associated with tubulointerstitial disease, immune-mediated multisystem disease, chronic kidney disease and end-stage renal disease. A rare syndrome of painful, nodular skin lesions has been described in dialysis patients with excessive levels of silicon. Balkan endemic nephropathy is postulated to be due to chronic intoxication with drinking water polluted by silicates released during soil erosion. The mechanism of silica nephrotoxicity is thought to be through direct nephrotoxicity, as well as silica-induced autoimmune diseases such as scleroderma and systemic lupus erythematosus. The renal histopathology varies from focal to crescentic and necrotizing glomerulonephritis with aneurysm formation suggestive of polyarteritis nodosa.

4.0 MEDICAL SURVEILLANCE PROGRAMME

Careful medical surveillance programs can detect early health effects characterized by irritation, inflammation, and lung fibrosis. Unfortunately, it can only suspect overexposures and monitor health effects, but cannot determine conditions of excessive absorption (biomarker of exposure is not available).

4.1 INDICATIONS

- Any occupation where workers are liable to be exposed to airborne silica above 50% of PEL or above the MEL. History of such exposure may also warrant a medical surveillance program.

4.2 CLINICAL EXAMINATION AND RELEVANT INVESTIGATIONS

- To detect pre-existing respiratory, kidney and autoimmune disease, which may worsen when exposed to crystalline silica.
- Symptoms of overexposures, as silica dust is usually associated with high exposure to dust such as sand blasting, dust from quarry activities, and dust from construction activities involving material that contain crystalline silica.
- Physical examination with emphasis on the respiratory system.
- Fitness to wear respirators.
- Spirometry.
- Chest X-ray is critical for early detection and monitoring of progression of silicosis. The OHD should be familiar with expected findings of silicosis, and referral to chest physicians is recommended if silicosis is suspected.
- Look out for tuberculosis.
- Kidney function test.

4.3 PRE-PLACEMENT MEDICAL EXAMINATION

- Clinical examination and relevant investigations as listed in 4.2.
- Decision for fitness to work and frequency of periodic medical examination:
    - Workers with disease of the respiratory system should not work in areas where there is significant silica exposure.
    - Decision for fitness to work for workers with kidney impairments, should be judged individually based on the exposure conditions. The advanced stage of kidney impairment may render the workers not fit to work with silica dust.
    - Pre-existing autoimmune disease should be considered unfit to work with silica dust.

4.4 PERIODIC MEDICAL EXAMINATION

- Clinical examination and relevant investigations as listed in 4.2.
- Frequency of periodic medical examination – annual
- Any work related to positive findings of the medical examination may need more frequent medical examination.
- Care should be taken for radiation risk of chest X-ray, and generally recommended, once in 1 to 3 years.
- **Exit medical examination -** the OHD should arrange for further medical follow up of cases with associated positive medical findings, due to progressive nature of silicosis.
- **Surveillance Case Definition for Silicosis**

A. History of occupational exposure to airborne silica dust.[1]
AND EITHER OR BOTH OF THE FOLLOWING:
B1. Chest radiograph or other imaging technique interpreted as consistent with silicosis.[2]
B2. Pathologic findings characteristic of silicosis.[3]

Footnotes

1. Exposure settings associated with silicosis are well characterized and have been summarized in several reviews. The induction period between initial silica exposure and development of radiographically detectable nodular silicosis is usually >10 years. Shorter induction periods are associated with heavy exposures, and acute silicosis may develop within months following massive silica exposure.

2. Cases can be classified as nodular or acute. Common radiographic findings of nodular silicosis include multiple, bilateral, and rounded opacities in the upper lung zones; other patterns have been described. Since patients may have mixed dust exposure, irregular opacities may be present or even predominant. To be considered consistent with silicosis, radiographs of nodular silicosis classified by NIOSH-certified “B” readers should have small opacity profusion categories of 1/0 or greater by the International Labour Organization classification system. If the largest opacity is >1 cm in diameter, progressive massive fibrosis [PMF] (also known as ‘complicated’ silicosis) is present. A bilateral alveolar filling pattern is characteristic of acute silicosis and may be followed by rapid development of bilateral small or large opacities.

3. Characteristic lung tissue pathology in nodular silicosis consists of fibrotic nodules with concentric “onion-skinned” arrangement of collagen fibers, central hyalinization, and a cellular peripheral zone, with lightly birefringent particles seen under polarized light. In acute silicosis, microscopic pathology shows a periodic acid-Schiff positive alveolar exudate (alveolar lipoproteinosis) and a cellular infiltrate in the alveolar walls.

4.5 BIOLOGICAL MONITORING

- No established biological exposure indices determinant based on ACGIH 2022 TLVs and BEIs.

5.0 MEDICAL SURVEILLANCE PROTECTION

- Indications for removal

Temporary MRP due to medical determination	
a. Abnormal clinical findings of the respiratory system.	Any level
b. Abnormal chest X-ray, suggestive of silicosis.	

c. If suspected lung cancer.	Any level
Permanent MRP due to medical determination	
a. All cases of pulmonary tuberculosis, active or inactive (aged below 35 years).	Any level
b. All symptomatic cases (pulmonary tuberculosis, chronic bronchitis, or cardiac failure).	
c. All cases diagnosed with lung cancer.	

- Duration of the temporary medical removal protection should be based on the medical conditions, considering the expected duration of diagnostic procedures and the duration of recovery.
- Return to work assessment is necessary to determine fitness to work.
- All cases recommended for MRP, suspected cases of poisoning and excessive absorption must be notified to the DG of DOSH.

6.0 RETURN TO WORK

- Generally, confirmed silica associated medical conditions, will worsen, when exposed further to silica, and thus, not fit to return to work with silica.
- Ensure workplace hygiene is safe and healthy and does not place the worker at increased risk of material impairment to health from exposure to silica.

7.0 NOTIFICATION TO DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed in NADOPOD 2004 using the relevant notification forms.

6.0 RETURN TO WORK

- Treatment based on specific medical conditions arising from exposure to the chemical.
- There is no definite treatment for silicosis. All pulmonary tuberculosis cases should be referred for treatment and further management in a chest hospital/ clinic. Symptomatic silicosis may require treatment as and when indicated.

9.0 PREVENTIVE MEASURES

- Good workplace and personal hygiene and safety.
- Approved personal protective equipment.
- Fit testing the respiratory protection.

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TOLUENE

1.0 DESCRIPTION

1.1 SYNONYM

Methacide, methylbenzene, methylbenzol, phenylmethane, toluol, tolu-sol.

1.2 OCCUPATIONAL EXPOSURE LIMITS (OEL)

USECHH 2000	
Toluene	
Eight-hour time weighted average limit	15 min – Short-term exposure limit
50 ppm (188 mg/m^3)	100 ppm (376 mg/m^3)

1.3 PHYSICOCHEMICAL PROPERTIES

- A clear, colourless, noncorrosive liquid with a sweet, pungent, and benzene-like odour.

1.4 MATERIAL USE

- Used to improve octane ratings in automobile.
- As important industrial solvent.
- In production of benzene, polymers, and organic chemicals.

2.0 TOXICITY

2.1 SOURCES OF POTENTIAL OCCUPATIONAL EXPOSURE

- Toluene and benzene production.
- As solvent in shoemaking, painting, and printing industry.
- Polymer and chemical industry.

2.2 ROUTE OF EXPOSURE

- Inhalation of vapor (primary)
- Percutaneous absorption of liquid (primary)
- Ingestion
- Eye contact

2.3 TOXICOKINETIC

Absorption	• Readily absorbed from the respiratory (40-60%) and gastrointestinal tracts and to some degree through the skin.
Distribution	• Rapidly distributed throughout the body. • Target organ of toluene are the eyes, skin, respiratory system, CNS, liver, kidneys. • May affect the reproductive system.
Metabolism	• Metabolised to benzoic acid and subsequently conjugated with glycine to form hippuric acid or conjugated with UDP-glucuronate to form acyl-glucuronide. • Minor metabolic pathway via ring hydroxylation to form metabolite cresol.

Excretion and Half Life	• Mainly excreted in the urine as hippuric acid and less frequently as cresol • Approximately 7–20% of absorbed toluene is eliminated in air unchanged. • Following a single acute exposure, toluene and its metabolites are almost completely eliminated within 24 hours.

2.4 TOXICOKINETIC

Toluene; methylbenzene

Toluene			
Classification Code	**Hazard classification**	**H-Code**	**Signal**
Flam. Liq. 2	Flammable liquids category 2	H225	Danger
Repr. 2	Reproductive toxicity category 2	H361d	
Asp. Haz.	Aspiration hazard category 1	H304	
STOT RE 2	Specific Target Organ Toxicity-Repeated Exposure Category 2	H373[(a)]	
Skin Irrit.2	Skin corrosion or irritation category 2	H315	
STOT SE 3	Specific Target Organ Toxicity-Single Exposure Category 3	H336	

*(a) - State the organ
Source: ICOP on Chemicals Classification and Hazard Communication 2019.

- **Cancer Classification IARC**
  Group 3. Not classifiable as to its carcinogenicity to humans.

3.0 HEALTH EFFECT MONITORING

Main target organ is the central nervous system. Organic solvents toxicity syndrome is anticipated following chronic over exposures.

3.1 ACUTE EFFECTS

System/Organ	Acute Effects
Cardiovascular	Cardiac dysrhythmia
Eyes	• Irritation of the eyes High exposure levels may cause: • Dilated pupils • Lacrimation
Gastrointestinal	• Burning sensation in mouth, stomach, and GIT. • Nausea and abdominal pain. • Higher exposure levels may cause loss of appetite.
Nervous System CNS and PNS	Inhalation of 100 ppm can cause: • Headache • Dizziness • Drowsiness • Hallucinations Inhalation of 100 to 200 ppm can cause CNS depression. Higher exposure levels: • Headache • Loss of energy • Loss of coordination • Confusion • Euphoria • Insomnia • Paraesthesia • Coma or death • Fatigue • Weakness

Respiratory	• Irritates the respiratory tract. • Cause cough • Liquid toluene that reaches the airways can cause serious lung damage and pneumonia with bleeding and necrosis.
Skin	• Skin dryness • Irritation

3.2 CHRONIC EFFECTS

System/Organ	Acute Effects
Cardiovascular	Heart palpitations
Ear, Nose and Throat	Sore throat Impaired hearing
Eyes	Irritation of eyes Nystagmus
Haematological	Possible blood effects and anaemia (most likely associated with benzene exposure).
Hepatobiliary	Mild effects on the liver
Nervous System CNS and PNS	• Dizziness • Headache • Insomnia • CNS damage and depression • Drowsiness • Impaired speech • Ataxia • Tremors • Cerebral atrophy • Decreased learning ability • Psychological disorders • Memory loss • Loss of coordination

Renal and Genitourinary	Mild effects on the kidneys.

Reproductive	• Evidence that exposure is associated with spontaneous abortion.
	• Effects to children of pregnant women occupationally exposed to toluene or mixed solvents:
	  ○ CNS dysfunction.
	  ○ Attention deficits.
	  ○ Minor craniofacial and limb anomalies.

Respiratory	Irritation of upper respiratory tract
Skin	• Degreasing of skin • Fissures • Inflammation • Dermatitis • Skin rash

4.0 MEDICAL SURVEILLANCE PROGRAMME

4.1 INDICATIONS

- Any occupation where workers are liable to be exposed to toluene exceeding 50% of the PEL and/or possibility of absorption.

4.2 CLINICAL EXAMINATION AND RELEVANT INVESTIGATIONS

- Clinical examination and baseline data with particular emphasis on the nervous system, liver, kidney, and skin (examined for evidence of chronic disorder).
- Urinalysis which includes specific gravity, albumin, glucose, and microscopic exami nation.
- Liver function test
- Renal function test
- Full blood count

4.3 PRE-PLACEMENT MEDICAL EXAMINATION

- Clinical examination and relevant investigation as listed in 4.2.
- Decision for fitness to work:
  - Workers with chronic diseases of the central nervous system, hepatic and renal function impairments have increased susceptibility to toluene.

4.4 PERIODIC MEDICAL EXAMINATION

- Clinical examination and relevant investigation as listed in 4.2.
- Frequency of periodic medical examination when findings are normal: annual.

4.5 BIOLOGICAL MONITORING

Determinant	Sampling time	BEL	Notation
Toluene in urine	End of shift	0.03 mg/L	-

- **Laboratory Method**
  - o No standard method reference currently established for toluene.
  - o Sampling procedures

Toluene in Urine	
Container	Glass container without preservative Note: *Request container from the laboratory. *Own container can be used (provide at least one unit container to the lab for blank test).
Transportation	Urine specimens should be refrigerated.
Stability	Urine specimens are stable for 30 days when refrigerated and frozen.
Preservation	Fill up the container, zero headspace.
Volume	Requested volume: 30 mL Minimum volume: 10 mL

  - o Analytical equipment/procedure
    Toluene
    - Headspace Gas Chromatography with Flame Ionization Detection (FID)
    - Mass Spectrometry Detection (MSD)

5.0 MEDICAL REMOVAL PROTECTION

- Indications for removal
    - Clinical findings and target organ function abnormalities:
        - Evidence of health effects suspected to be caused by CHTH or may be worsened by CHTH.
        - Duration of removal is the duration expected for normalization.
    - BEL
        - Exceed BEL
        - MRP and repeat sample immediately, return to work assessment when the result is available.
    - Pregnancy and breastfeeding.
    - Others:
        - Medical conditions of central nervous system, hepatic system.
        - Renal function impairments.
- Temporary MRP
    - MRP based on medical condition.
        - The determination of the temporary MRP duration should consider the following aspects:
            - expected duration of diagnostic procedures.
            - duration of recovery.
    - MRP based on BEL.
        - The determination of the temporary MRP duration should consider the following aspect:
            - Availability of the repeat BM sample results.
- All cases recommended for MRP and suspected cases of poisoning and excessive absorption must be notified to the DG of DOSH.
- All employees undergoing MRP should have repeat urine examinations (and relevant biochemical tests where indicated) at 3 monthly intervals.

6.0 RETURN TO WORK

- **Return to Work based on:**

  i. BEL
  - Return to work when the results are below the BEL.

  ii. Biochemical results which include urine biomarkers
  - Abnormal results have returned to normal.

  iii. Medical condition
  - No longer detected of having a medical condition.

  iv. Workplace management
  - Ensure workplace hygiene is safe and healthy and does not place the worker at increased risk of material impairment to health from exposure to toluene.

7.0 NOTIFICATION TO DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed in NADOPOD 2004 using the relevant notification forms.

8.0 TREATMENT AND MANAGEMENT

- All cases of poisoning must be immediately removed from exposure and referred for hospital treatment.
- Irrigate eyes with water.
- Wash contaminated areas of the body with soap and water.

9.0 PREVENTINE MEASURES

- Good workplace and personal hygiene and safety.
- Approved personal protective equipment.

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TRICHLOROETHYLENE

1.0 DESCRIPTION

1.1 SYNONYMS

- Acetylene trichloride, Ethinyl trichloride; Ethylene trichloride, Trichloran, Trichloren, Trichloroethene, 1,1,2-Trichloroethylene, Trichlororan; 1,1,2-Trichloro-1,2,2-trifluoroethan.

1.2 OCCUPATIONAL EXPOSURE LIMITS (OEL)

USECHH 2000	
Trichloroethylene	
Eight-hour time weighted average limit	**15 min – Short-term exposure limit**
100 ppm (550 mg/m^3)	150 ppm (820 mg/m^3)

1.3 PHYSICOCHEMICAL PROPERTIES

- Non-flammable, colourless liquid with a somewhat sweet odour and a sweet, burning taste.

1.4 MATERIAL USE

- Used mainly as a solvent to remove grease from metal parts.
- As an ingredient in adhesives, paint removers, typewriter correction fluids, and spot removers.
- Used for cleaning lenses in the optical industry.
- Used as solvent for extraction of waxes, fats, resins, and oils.
- Used as a solvent or chemical intermediate in printing inks, varnishes, adhesives, paints, lacquers, rug cleaners and disinfectants.
- Used in the textile industry to scour cotton, wool, and other fabrics (removes oils and lubricants) and in waterless dyeing and finishing.
- Chemical intermediate in production of other chemicals, such as refrigerants.

2.0 TOXICITY

2.1 SOURCES OF POTENTIAL OCCUPATIONAL EXPOSURE

- Manufacture of Trichloroethylene (TCE) .
- General industry (Using trichloroethylene as solvent or intermediate chemical).
- Optical industry.
- Printing and the production of printing ink.
- Paint production.
- Industrial dry cleaning.
- Textile industry.

2.2 ROUTE OF EXPOSURE

- Inhalation (primary)
- Ingestion
- Skin absorption

2.3 TOXICOKINETICS:

Trichloroethylene	
Absorption	• TCE is readily absorbed following exposure by inhalation or ingestion and to some extent following skin contact. • Approximately 37 - 64% of inhaled TCE is absorbed from the lungs.
Distribution	• Following absorption TCE is distributed throughout the body.
Metabolism	• TCE undergoes metabolism via an oxidative pathway in the liver and lungs. • The main metabolites are trichloroethanol (major), trichloroacetic acid (major) and trichloroethanol-glucuronide.
Excretion	• TCE is excreted unchanged via the lungs or as metabolites in the urine. • Half life of 8 hours.

2.4 HAZARD CLASSIFICATION

Classification code	Hazard classification	H-Code	Signal
Carc. 1B	Carcinogenicity category 1B	H350	Danger
Muta. 2	Germ cell mutagenicity category 2	H341	
Eye Irrit. 2	Serious eye damage or eye irritation category 2	H319	

Skin Irrit. 2	Skin corrosion or irritation category 2	H315	
STOT SE 3	Specific target organ toxicity – single exposure category 3	H336	

Source: ICOP on Chemicals Classification and Hazard Communication 2019.

- Suspected of causing genetic defects (Sittigs., 2019)
- **Cancer Classification IARC**

  Group 1 (GHS system as Cat 1a)

3.0 HEALTH EFFECT MONITORING

The ACGIH basis for the TLV is CNS impairment, cognitive decrements, and renal toxicity.

- The OHD should look out for CNS effects and irritation effects to the respiratory tract and skin, in the history taking, as those are signs of overexposures.
- Short-Term Exposure: Exposure to the vapour irritates the eyes, skin, and respiratory tract. High exposures can cause pulmonary edema, a medical emergency that can be delayed for several hours. This can cause death. The symptoms of pulmonary edema are aggravated by physical effort. Inhalation at levels of around 100 ppm causing headache, sleepiness, nausea, vomiting, dizziness, and coughing have been documented.
- Unconsciousness can result with exposure at 3,000 ppm. Exposure to 8,000 ppm can cause death. Can be absorbed through skin. Can cause skin irritation, burning or redness; blistering can occur. Can cause eye irritation; burning sensation; and/or watering and can cause permanent damage.
- Ingestion can cause chemical pneumonitis and diminish kidney action. It can cause drunkenness, vomiting, diarrhoea, or abdominal pain. Unconsciousness, liver or kidney damage, vision distortion and death have been reported at large doses. Exposure to TCE may affect the CNS causing light-headedness, dizziness, visual disturbances; feeling of excitement; nausea, and vomiting. High levels can cause irregular heartbeat, unconsciousness, and death.
- Long-Term Exposure: Liver problems; increased risk of cancer. May affect kidneys. Contact with vapour levels near 100 ppm can cause giddiness, nervous exhaustion, increased sensitivity to alcohol including redness in the face (trichloroethylene blush); the ability to become addicted to the vapour; as well as effects of acute exposure listed above. Higher levels can cause irregular heartbeat.
- Repeated contact with hands can cause excessive dryness, cracking, burning, loss of sense of touch or temporary paralysis of fingers. Most of these effects seem to go away after exposure has stopped.

3.1 ACUTE EFFECTS

System/Organ	Acute Effects
Cardiovascular	• High exposure levels can sensitise myocardium and cause cardiac arrhythmia. • Death from cardiac failure.
Nervous System - CNS	• Narcosis • Lack of coordination • Mood changes (addictive potential) • Massive exposure can cause excitation, dizziness, and euphoria initially. This is followed by a depressive phase of headache, nausea, sleepiness, and coma.
Ear, Nose and Throat	• Nose and throat irritation
Respiratory	• Respiratory tract irritation • Chemical pneumonitis • Death from respiratory failure can occur.
Skin	• Irritations effects

3.2 CHRONIC EFFECTS

System/Organ	Acute Effects
Hepatobiliary	• Few cases of hepatitis-like syndromes and steatosis (fatty liver) have been reported from chronic exposure to Trichloroethylene.
Nervous System - CNS	• Non-specific complaints like headache, irritability, fatigue, and insomnia. • Psychological disorders. • Mood changes. • Poor memory impairment in psychomotor and behavioural tests have been reported. • Alcohol intolerance characterised by skin vasodila -tation especially in the face can occur. • Neuropathy – loss of function of nerves.
Renal and Genitourinary	• Altered renal function such as proteinuria. • Increase of blood urea may occur from chronic exposures to high levels of TCE. • Prolonged exposure to high concentrations of TCE (hundreds to thousands ppm) increases incidence of renal cancer.

Skin	• Prolonged or repeated skin contact with liquid TCE can cause irritation and dermatitis.
Renal and Genitourinary	• Severe systemic allergic reaction - presents with triad of generalized rash (Steven Johnson Syndrome or Toxic Epidermal Necrolysis), fever and jaundice; seen within 2 – 3 weeks after starting exposure in sensitive individuals with minimal TCE exposure.

4.0 MEDICAL SURVEILLANCE PROGRAMME

4.1 INDICATIONS

- Any work where workers are exposed to air levels of trichloroethylene which are liable to exceed 50% of the permissible exposure level and/or where there is a routine risk of skin contact.

4.2 CLINICAL EXAMINATION AND RELEVANT INVESTIGATIONS

- Clinical examination on history of health effects related to exposure to the central nervous system, liver, skin, and kidney.
- Target organ markers (non-specific): Q16 questionnaire, LFT (periodic), RFT (periodic), Chest X-ray (baseline and later when clinically indicated).
- Biological monitoring: Trichloroacetic acid in urine (end of shift or at end of work week), TCA in urine in case of high exposure incidents.

4.3 PRE-PLACEMENT MEDICAL EXAMINATION

- Clinical examination and relevant investigations as listed in 4.2.
- Baseline medical examinations and target organ markers.
- Baseline BM marker (due to Ns notation in the ACGIH TLVs).
- Decision for fitness to work:
  - Workers with liver diseases, solvent abuse or who are alcoholics should not work in areas where there is significant TCE exposure.

4.4 PERIODIC MEDICAL EXAMINATION.:

- Clinical examination and relevant investigations as listed in 4.2.
- Frequency of periodic medical examination.
  - Annually when there are no work-related health effects medical findings and negative BM markers.
  - More frequent program is required if there are complaints of work-related health effects and any abnormal findings in the medical examinations and the BM marker. The OHD have to decide on the practicable frequency, which is less than once in 12 months.

4.5 BIOLOGICAL MONITORING

Determinant	Sampling time	BEL	Notation
Trichloroacetic acid in urine.	End of shift at end of workweek.	15 mg/L	Ns

Note: Trichloroacetic acid and trichloroethanol levels are not specific only to TCE exposure. May interfere by other chemicals such as perchloroethylene, ethanol and phenobarbital exposure.

- **Laboratory Method**
  - o Sampling procedure

Trichloroacetic Acid in Urine	
Container	Polyethylene container. Note: *Request container from the laboratory. *Own container can be used (provide at least one unit container to the lab for blank test).
Transportation	Specimens should be refrigerated.
Stability	Specimens are stable for 14 days either at room temperature, refrigerated or frozen.
Preservation	No specific preservative mentioned.
Volume	Requested volume: 30 mL Minimum volume: 10 mL

  - o **Analytical equipment/procedure**

    Trichloroacetic acid
    - Gas chromatography-mass spectrometry

5.0 MEDICAL REMOVAL PROTECTION

- **Indications for removal**

Categories	Level	Duration of removal
Temporary MRP due to medical determination		
All workers of definite or suspected TCE poisoning and	Any level	The duration required for confirmations and recovery and corrective actions at the workplace.
Excessive absorption of TCE	Exceed BEL	Repeat the test as soon as possible. The duration required for the marker to fall below BEL.
Permanent MRP due to medical determination		
a. Workers presenting with work related fever, severe skin rash and/or jaundice. *They should be immediately removed and investigated to exclude TCE allergy. These workers may need immediate hospitalisation.	Any level	Not applicable
b. Workers with diagnosed liver disease.		
c. Alcoholics and cases of solvent abuse.		

- Pregnancy and breastfeeding.
- All cases recommended for MRP, suspected cases of poisoning and excessive absorption must be notified to the DG of DOSH.

6.0 RETURN TO WORK

- Return to work based on:
  i. BEL
    - Return to work when the results are below the BEL.

  ii. Medical condition
    - Normal liver function test results.
    - No symptoms of TCE poisoning.

  iii. Workplace management
    - Ensure workplace hygiene is safe and healthy and does not place the employee at increased risk of material impairment to health from exposure to TCE.

7.0 NOTIFICATION TO DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed in NADOPOD 2004 using the relevant notification forms.

8.0 TREATMENT AND MANAGEMENT

- All cases of acute poisoning must be immediately removed from exposure, provide appro priate first aid measures, and refer for hospital treatment.
- Treatment based on specific medical conditions arising from exposure to the chemical.
- All cases of severe allergic reaction must be immediately referred for treatment in hospital. They should be advised to ensure that they are never exposed to TCE in future as re-exposure may result in fatality.

9.0 PREVENTINE MEASURES

- Good workplace and personal hygiene and safety.
- Approved personal protective equipment.
- Appropriate signage.

10.0 REFERENCES

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VINYL CHLORIDE MONOMER

1.0 DESCRIPTION

1.1 SYNONYMS

- Chloroethene; Chloroethylene; Ethylene monochloride; Monochloroethene; Monochloroethylene; VC; VCM.

1.2 OCCUPATIONAL EXPOSURE LIMITS (OEL)

USECHH 2000 (Eight-hour time weighted average limit)	
Vinyl chloride	1 ppm, 2.6 mg/m^3 (TWA)

1.3 PHYSICOCHEMICAL PROPERTIES

- Flammable gas at room temperature (encountered as a cooled liquid).
- The colorless liquid forms vapors which have a pleasant, ethereal odor.

1.4 MATERIAL USE

- Used as a vinyl monomer in the manufacture of polyvinyl chloride (vinyl chloride homopolymer) and other copolymer resins.
- Used as a chemical intermediate and as a solvent.

2.0 TOXICITY

2.1 SOURCES OF POTENTIAL OCCUPATIONAL EXPOSURE

- PVC-processing plants (manufacture of PVC).
- Storage of VCM
- Sampling and analysis of VCM
- Chemical industry (vinyl chloride and related derivatives production).
- Construction and general industry (usage of vinyl chloride as solvent or intermediate).

2.2 ROUTE OF EXPOSURE

- Inhalation (primary)
- Ingestion
- Skin absorption
- Eye contact

2.3 TOXICOKINETICS:

Vinyl Chloride Monomer	
Absorption	• Vinyl chloride is readily and rapidly absorbed via inhalation, ingestion and through the skin. • At room temperature vinyl chloride is a gas, so inhalation is the major route exposure.
Distribution	• Following exposure, it is distributed through the body, with the highest concentrations found in the liver and kidneys, followed by the lungs and spleen.
Metabolism	• Vinyl chloride is mainly metabolised in the liver into reactive metabolites (chloroethylene oxide) which acts as ultimate toxicant.
Excretion	• It is mainly excreted in the urine as thiodiglycolic acid. In higher doses of exposure, majority vinyl chloride will be excreted through exhalation. • Half life is about 18 hours.

2.4 HAZARD CLASSIFICATION

Classification code	Hazard classification	H-Code	Signal
Carc. 1A	Carcinogenicity category 1A	H350	Danger
Flam. Gas 1	Flammable gases category 1	H220	
Press. Gas(c)	Gases under pressure	H280/281(d)	

*(d) For gases under pressure, state the relevant H-code based on its hazard category.

Source: ICOP on Chemicals Classification and Hazard Communication (Amendment) 2019.

- Cancer Classification IARC

  Group 1
  - There is sufficient evidence in humans for the carcinogenicity of vinyl chloride. Vinyl chloride causes angiosarcoma of the liver, and hepatocelular carcinoma.

  - There is no strong evidence for other type of cancer, although there are studies, suggesting that VCM can cause lung, brain, hematopoietic system, and the lymphatic system.

3.0 HEALTH EFFECT MONITORING

ACGIH TLVs set the TLV determinant as Lung cancer and liver damage. No BM marker listed in the ACGIH TLVs. The PEL is at 1 ppm.

The smell of VCM is an important way to determine exposure to VCM. The odour threshold is 10 ppm to 25,000 ppm. After a 5-min exposure to VCM at 16,000 ppm, volunteers experienced dizziness, lightheadedness, nausea, and visual and auditory dulling (Lester et al. 1963). Symptomatic exposures occur much far higher than the PEL.

Any health effects related to acute exposure to VCM is a significant exposure and signifying significant loss of control.

It is important to document the frequency and the duration of employee smells VCM during work, in the MS program. Any such reports from the employee, indicate that the control measures need to be improved. Otherwise, symptoms of exposure only occur in high exposure incidence.

3.1 ACUTE EFFECTS

System/Organ	Acute Effects
Eyes	• Compressed gas or liquid can cause irritation of the eyes. • Frostbite injury of eyes (corneal/conjunctival burns or irritation) from contact with escaping compressed vinyl chloride gas or liquid vinyl chloride.
Nervous System – CNS	• Non-specific manifestations e.g., headache, giddiness, disorientation. • May progress to loss of consciousness. • Acute heavy inhalation of vinyl chloride may cause nausea, headache, dizziness and drowsiness, central nervous system depression, cardiac arrhythmias, unconsciousness, and possible death. • Depression of the central nervous system (CNS) i. Dizziness ii. Confusion, headache, dizziness

	iii. Drowsiness, loss of coordination, visual and auditory abnormalities, disorientation, nausea, headache, burning or tingling of extremities (~20,000 ppm); and iv. Death (>20,000 ppm) likely due to CNS and respiratory depression.
Respiratory	• Lung irritation • Asphyxiation in poorly ventilated or enclosed spaces as it is heavier than air. • Mild respiratory irritation (wheezing and bronchitis) – transient.
Skin	• Compressed gas or liquid can cause frostbite or irritation of the skin. • Mucous membrane irritation. • Frostbite injury of skin (redness, blistering, scaling) from contact with escaping compressed vinyl chloride gas or liquid vinyl chloride.

3.2 CHRONIC EFFECTS

System/Organ	Chronic Effects
Hepatobiliary	• Liver and/or spleen fibrosis. • Chronic exposure can permanently damage the liver and spleen. • Liver cancers (angiosarcoma of liver and hepatocellular carcinoma). • Cirrhosis • Portal hypertension
Nervous System	• Chronic exposure can permanently damage the nervous system. • Sensory motor polyneuropathy • Pyramidal, extrapyramidal, and cerebellar abnormalities.
Renal and Genitourinary	Chronic exposure can permanently damage the kidneys.
Respiratory	Lung fibrosis and lung cancer.

Skin	Scleroderma-like lesions
Others	• Raynaud's disease • Acro osteolysis (especially of the hands) • Pancytopenia • Chronic exposure of vinyl chloride can permanently damage the blood cells. • Vinyl chloride may damage the developing foetus, increased incidence of birth defects & pre-eclampsia and spontaneous abortions. • Purpura (immunopathological phenomena) • Thrombocytopenia (immunopathological phenomena)

4.0 MEDICAL SURVEILLANCE PROGRAMME

4.1 INDICATIONS

- Any occupation where workers are liable to be exposed to vinyl chloride above 50% of PEL or when exceeding the MEL.
- However, it is best to proceed with MS program, as it is not possible to secure all possible exposures, routine, or non-routine, in a production where VCM is used.

4.2 CLINICAL EXAMINATION AND RELEVANT INVESTIGATIONS

- Clinical examination with emphasis on liver (ALT and GGT are the first enzymes raised), central nervous system, lungs, skin, kidney, and blood system.
- Required for initial and periodic medical examination; includes alcohol intake, history of hepatitis, exposure to hepatotoxic agents, blood transfusions, hospitalizations, and work history.
- Target organ markers (non-specific): LFT (periodic), RFT (periodic), Full blood pictures, spirometry, Chest X-ray (baseline and thereafter at least once in two years).
- Annual ultrasound of the liver may be done to detect early health effects to the liver. Features to look out for is hepatomegaly, steatosis and periportal fibrosis.
- Biological monitoring: urine Thiodiglycolic Acid

4.3 PRE-PLACEMENT MEDICAL EXAMINATION

- Clinical examination and relevant investigations as listed in 4.2.
- Baseline medical examinations, target organ markers and BM.
- Decision of fitness to work:
- Workers with abnormal liver function test results, heavy alcohol ingestion, Hepatitis B carrier, Hepatitis C serology positive and have clinical evidence of liver disease, e.g., enlarged spleen and liver, spider naevi, etc. should not work in areas where there is significant vinyl chloride exposure.

4.4 PERIODIC MEDICAL EXAMINATION

- Clinical examination and relevant investigations as listed in 4.2.
- Frequency of periodic medical examination - Annually when there are no work-related health effects medical findings and negative BM marker.
- More frequent program is required if there are complaints of work-related health effects and any abnormal findings in the medical examinations and the BM marker. The OHD have to decide on the practicable frequency, which is less than once in 12 months.
- Monitoring frequency:
- Every 6 months for each employee who has been employed in vinyl chloride or polyvinyl chloride manufacturing for 10 years or longer.
- After exposure during emergency situations.

4.5 BIOLOGICAL MONITORING

No biological exposure indices determinant based on ACGIH 2022 TLVs and BEIs.

5.0 MEDICAL REMOVAL PROTECTION

Indications for removal

Temporary MRP due to medical determination	
a. All cases of suspected VCM disease b. Abnormal liver function test result with high risk of VCM Any level poisoning. c. Abnormal ultrasound of liver	Any level

Temporary MRP due to pregnancy	
All pregnant women	Any level
Permanent MRP due to medical determination	
a. All cases of definite VCM disease	Any level
b. All cases of liver cancer	
c. Workers with persistently abnormal liver function test results (one or more abnormal result on at least 2 occasions within a 3-month period).	
d. Has clinical evidence of liver disease, e.g., enlarged spleen and liver, spider naevi, etc.	
e. Hepatitis B carrier; and/or Hepatitis C serology positive.	
Permanent MRP due to alcohol	
Heavy drinkers and solvent abusers.	Any level

- **Duration of temporary MRP**
  - o MRP based on medical condition.
    The determination of the temporary MRP duration should consider the following aspects:
    - expected duration of diagnostic procedures
    - duration of recovery.

- All cases recommended for MRP, suspected cases of poisoning and excessive absorption must be notified to the DG of DOSH.

6.0 RETURN TO WORK

- Assessment due date will depend on the duration required to confirm the diagnosis or the duration required for normalisation of the medical conditions.
- Return to work based on:
  i. Medical condition
     - Normal liver function test results and no symptoms of VCM poisoning.
     - After returning to work, continue to monitor the worker's liver function test every 3-monthly for the next 6 months.

  ii. Workplace management
     - Ensure workplace hygiene is safe and healthy and does not place the worker at increased risk of material impairment to health from exposure to vinyl chloride.

7.0 NOTIFICATION TO DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed in NADOPOD 2004 using the relevant notification forms.

8.0 TREATMENT AND MANAGEMENT

- Care should be taken when conducting EM, for possible false negative results especially when mobilising integrated sampling trains. Passive sampling offers a reliable alternative.
- All cases of acute poisoning must be immediately removed from exposure,provide appropriate first aid measures, and refer for hospital treatment.
- Treatment based on specific medical conditions arising from exposure to the chemical.
- Cases with liver cancer and liver angiosarcoma should be referred to the appropriate clinical specialist for treatment.

9.0 PREVENTIVE MEASURES

- Good workplace and personal hygiene and safety.
- Approved personal protective equipment.
- Inform all women that they are to inform the supervisor as soon as they are found to be pregnant.

10.0 REFERENCES

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XYLENE

1.0 DESCRIPTION

1.1 SYNONYMS

- **m-isomer**

m-Dimethylbenzene, 1,3-Dimethylbenzene, m-Methyltoluene, m-Xylene; 1,3-Xylene, m-Xylol.

- **o-isomer**

Dimethylbenzene, 1,2-Dimethylbenzene, o-Methyltoluene, 1,2-Methyltoluene, o-Xylene, 1,2-Xylene, o-Xylol.

- **p-isomer**

Chromar, p-Dimethylbenzene, 1,4-Dimethylbenzene, p-Methyltoluene, 4-Methyltoluene, Scintillar, p-Xylene; 1,4-Xylene, p-Xylol.

1.2 OCCUPATIONAL EXPOSURE LIMITS (OEL)

USECHH 2000 (Eight-hour time weighted average limit	
Xylene (o-, m-, p-isomers)	100 ppm (434 mg/m^3) (TWA)
	150 ppm (651 mg/m^3) (STEL)

1.3 PHYSICOCHEMICAL PROPERTIES

- Colourless, flammable liquids that are practically insoluble in water and have a sweet odour.

1.4 MATERIAL USE

- As important industrial solvent and intermediate in chemical synthesis.

2.0 TOXICITY

2.1 SOURCES OF POTENTIAL OCCUPATIONAL EXPOSURE

- Xylene production or petrochemical industry

- Shoemaking, painting, and printing industry that need toluene as solvent.
- As important industrial solvent and intermediate in chemical synthesis.

2.2 ROUTE OF EXPOSURE

- Inhalation of vapor (primary)
- Percutaneous absorption of liquid/ingestion
- Skin and/or eye contact

2.3 TOXICOKINETIC

Absorption	Xylene can be absorbed from the respiratory and gastrointestinal tracts and to some degree through the skin.
Distribution	• Rapidly distributed to the tissues through the systemic circulation. • Bound to serum proteins as it is distributed through the blood. • Mainly accumulates in the adipose tissues.
Metabolism	• Metabolised to methylbenzyl alcohol. • Followed by further oxidation to corresponding methylbenzoic acid. • Methylbenzoic acid is subsequently conjugated with glycine to form methylhippuric acid or conjugated with UDP-glucuronate to form acyl glucuronides.
Excretion and Half Life	• Elimination process is rapid. More than 70% of absorbed xylene was excreted in the urine as metabolites. Minor portion was exhaled unchanged. • The estimated half-life of xylene in the venous blood is 30 minutes to 1 hour which is followed by a slower phase with an estimated half-life of 20 to 30 hours (EPA., 2013)

2.4 HAZARD CLASSIFICATION

Hazard Classification

	Classification Code	Hazard Classification	H-code	Signal
Xylene o-xylene m-xylene p-xylene	Flam. Liq. 3	Flammable Liquids Category 3	H226	Warning
	Acute Tox. 4 (inh)	Acute toxicity category 4 (inhalation)	H332	

	Acute Tox. 4 (dermal)	Acute toxicity category 4 (dermal)	H312	
	Skin Irrit. 2	Skin corrosion or irritation category 2	H315	

Source: ICOP on Chemicals Classification and Hazard Communication 2019.

- **Suspected of causing genetic defects (Sittigs., 2019)**
- **Cancer Classification IARC**

Group 3. Not classifiable as to its carcinogenicity to humans

3.0 HEALTH EFFECTS MONITORING

Acute over exposures mainly cause central nervous system depression and irritation effects to the respiratory systems. Other organs are affected by chronic over exposures.

3.1 ACUTE EFFECTS

System/Organ	Accute Effects
Eyes	Irritates eye
Gastrointestinal	• Burning sensation in mouth, stomach, and GIT • Abdominal pain
Hepatobiliary	Reversible liver damage
Hepatobiliary	• Xylene intoxication* which causes: o Headache o Dizziness o Nausea and vomit • Further exposure can cause CNS depression manifested by: o Shallow breathing o Weak pulse • Exposure to extreme high concentrations (10,000 ppm or above) can lead to strong narcotic effects characterized by: o Slurred speech o Stupor fatigue

	o Confusion o Unconsciousness o Coma o Possible death
Renal and Genitourinary	Reversible kidney damage
Respiratory	• Irritates the respiratory tract. • Sudden high exposure can cause lung congestion.
Skin	• Skin dryness • Irritation • Cracking

*Xylene intoxication may occur at 3 minutes of 250 ppm inhalation exposure.

3.2 CHRONIC EFFECTS

System/Organ	Chronic Effects
Eyes	Damage to surface of the eyes
Gastrointestinal	Intestinal tract disturbances
Hepatobiliary	Liver damage
Nervous System CNS and PNS	• Headache • Dizziness • Fatigue • Anxiety • Impaired short-term memory • Difficulty concentrating • CNS depression
Renal and Genitourinary	Kidney damage
Skin	• Irritation • Dryness • Cracking

4.0 MEDICAL SURVEILLANCE PROGRAMME

4.1 INDICATIONS

Any occupation where workers are liable to be exposed to xylene exceeding 50% of the PEL and/or possibility of absorption.

4.2 CLINICAL EXAMINATION AND RELEVANT INVESTIGATIONS

- Clinical examination with emphasis on neurological, respiratory system, the kidneys, and skin.
- Complete blood count as baseline (xylene has been shown to cause reversible hematopoietic depression in animals).
- Liver function test (liver damage has been observed in human exposed to xylene).
- Urinalysis (kidney damage has been observed in human exposed to xylene).
- Neurological, psychiatric, and psychological examination.
- Respiratory system examination.
- Kidney function test and urine examination.
- Full blood count.

4.3 PRE-PLACEMENT MEDICAL EXAMINATION

- Clinical examination and relevant investigations as listed in 4.2.
- Decision for fitness to work.
    - Workers suffering from chronic disease of the central nervous system, diseases impairing hepatic and/or renal functions as well as pregnant women are susceptible groups.

4.4 PERIODIC MEDICAL EXAMINATION

- Clinical examination and relevant investigations as listed in 4.2.
- Frequency of periodic medical examination with normal findings: Annually

4.5 BIOLOGICAL MONITORING

Determinant	Sampling time	BEL	Notation
Methylhippuric acids in urine	End of shift	1.5 g/g creatinine	-

- Laboratory Method
    - Sampling procedures: NMAM 8301

Methylhippuric Acids in Urine	
Container	Plastic urine container without preservative. Note: *Request container from the laboratory. *Own container can be used (provide at least one unit container to the lab for blank test).
Transportation	Urine specimens should be refrigerated.
Stability	Urine specimens are stable for 30 days when refrigerated and frozen.
Preservation	● No specific preservative is required. ● Acidify with 1.0 mL acetic acid per 100 mL urine. ● Sample must be creatinine corrected.
Volume	Requested volume: 30 mL Minimum volume: 10 ml

Note: The creatinine in the urine should be measured within 24 hours of sample collection.

- Analytical equipment/procedure
    - Methylhippuric acid
        - Colorimetry
        - High Performance Liquid Chromatography Ultra-violet Detection.

5.0 MEDICAL REMOVAL PROTECTION

- Indications for removal
    - Clinical findings
      Evidence of health effects that is suspected to be caused by CHTH or may be worsened by CHTH.
    - Target organ function abnormalities
    - BEL
      Exceed BEL
    - Pregnancy and breastfeeding
    - Others:
      Medical conditions of the central nervous system, diseases impairing hepatic and/or renal functions.

- MRP duration depends on the abnormalities found:
    - Exceed BEL: MRP and repeat the sample as soon as possible. Next assessment when the result is available.
    - Clinical findings and target organ functions: assessment date depends on expected recovery duration.
- All cases recommended for MRP and suspected cases of poisoning and excessive absorption must be notified to the DG of DOSH.
- All employees undergoing MRP should have repeat urine examinations (and relevant biochemical tests where indicated) at 3 monthly intervals.

6.0 RETURN TO WORK

Return to work based on;

i. BEL

Return to work when the results are below BEL.

ii. Medical condition

No longer detected of having a medical condition.

ii. Workplace management

Ensure workplace hygiene is safe and healthy and does not place the worker and increased risk of material impairment to health from exposure to xylene.

7.0 NOTIFICATION TO DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed in NADOPOD 2004 using the relevant notification forms.

8.0 TREATMENT AND MANAGEMENT

- All cases of acute poisoning must be immediately removed from exposure, provide appropriate first aid measures, and refer for hospital treatment.
- Treatment based on specific medical conditions arising from exposure to the chemical.

9.0 PREVENTIVE MEASURES

- Good workplace and personal hygiene and safety.
- Approved personal protective equipment.

10.0 REFERENCES

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ALUMINIUM AND ITS COMPOUND

1.0 DESCRIPTION

Aluminium is a silvery-white metal is the most widespread metal on Earth, making up more than 8% of the Earth's core mass. It's also the third most common chemical element on our planet after oxygen and silicon. At the same time, because it easily binds with other elements, pure aluminium does not occur in nature. This is the reason that people learned about it relatively recently. Formally aluminium was produced for the first time in 1824 and it took people another fifty years to learn to produce it on an industrial scale.

The most common form of aluminium found in nature is aluminium sulphates. Today we know about almost 300 various aluminium compounds and minerals containing aluminium, from feldspar, a key source mineral on Earth, to ruby, sapphire and emerald, which are far less common.

Another rather common mineral, bauxite, is used today as the primary raw material in aluminium production.

Several chemical compounds with Al are in extensive use in various products and processes associated with human activities. These compounds are Al chloride, Al hydroxide (alumina trihyrate), Al nitrate, Al phosphate, Al sulfate (alum), Al potassium (potash alum), Al ammonium sulfate (ammonium alum) and Al silicate.

Once mineral-bound aluminum is recovered from ores, it forms metal compounds, complexes, or chelates. Examples of the different forms of aluminum include aluminum oxide, aluminum chlorhydrate, aluminum hydroxide, aluminum chloride, aluminum lactate, aluminum phosphate, and aluminum nitrate. The metal itself is also used.

1.1 SYNONYM

Aluminium sulfate: Alum; Aluminum alum; Aluminum sulfate; Aluminum trisulfate; Cake alum; Diaaluminum trisulfate; Dialuminum sulfate; Paper maker’s alum; Sulfuric acid, aluminum salt; Sulfuric acid, aluminum salt.

Aluminium chloride: Aluminum chloride anhydrous; Aluminum chloride solution; Aluminum trichloride; Anhydrol forte; Anhydrous aluminum chloride.

Aluminium as Al: Aluminum flake; Aluminum 27; Aluminum dehydrated; Aluminum metallic powder; Aluminum powder; AO A1; AR2; AV00; AV000; C.I. 77000; Emanay atomized

aluminum powder; JISC 3108; JISC 3110; L16; Metana; Metana aluminum paste; Noral aluminum; Noral extra fine lining grade; Noral nonleafing grade; PAP-1.

Aluminum oxide, Aluminium nitrate, Aluminum phosphate, Alumnium potassium, Aluminum ammonium sulfate, aluminum silicate.

1.2 OCCUPATIONAL EXPOSURE LIMITS (OEL)

USECHH 2000 8 hr TWA Airborne Concentration (mg/m^3)	
Aluminum, as metal dust	10
As pyro powders	5
Welding fumes as AL	5
Soluble salts, as AL	2
Alkyl (NOC) as AL	2
Aluminum oxide (1344-28-1	10
Aluminum metals (7429-90-5) and soluble compounds	

1.3 PHYSICOCHEMICAL PROPERTIES

Aluminium offers a rare combination of valuable properties. It is one of the lightest metals in the world: it's almost three times lighter than iron but it's also very strong, extremely flexible and corrosion resistant because its surface is always covered in an extremely thin and yet very strong layer of oxide film. It doesn't magnetise, it's a great electricity conductor and forms alloys with practically all other metals.

Even a very small amount of admixtures can drastically change the properties of the metal, making it possible to use it in new areas. For example, in ordinary life you can find aluminium mixed with silicon and magnesium literally on the road, i.e., in the aluminium alloy wheels, in the engines, chassis and other parts of modern automobiles.

1.4 MATERIAL USE

The compounds are used in crude oil refining and cracking of petroleum; manufacturing of cooking utensils and foils, parchment paper, printing ink, glass, ceramics, pottery, incandescent filaments, fireworks, explosives, photographic flashlight, electric insulators, cement, paints and varnishes, fumigants and pesticides, lubricants, detergents, cosmetics,

pharmaceuticals (drugs), vaccines, as well as in water treatment and purification, treating sewage and fur, tanning leather, waterproofing clothes and concretes, industrial filtration, hemodialysis, measuring radiation exposure, in products as flame retardant and fireproofing, anticorrosion agent, food additives to prevent caking as well as components of baking powders and colorants.

2.0 TOXICITY

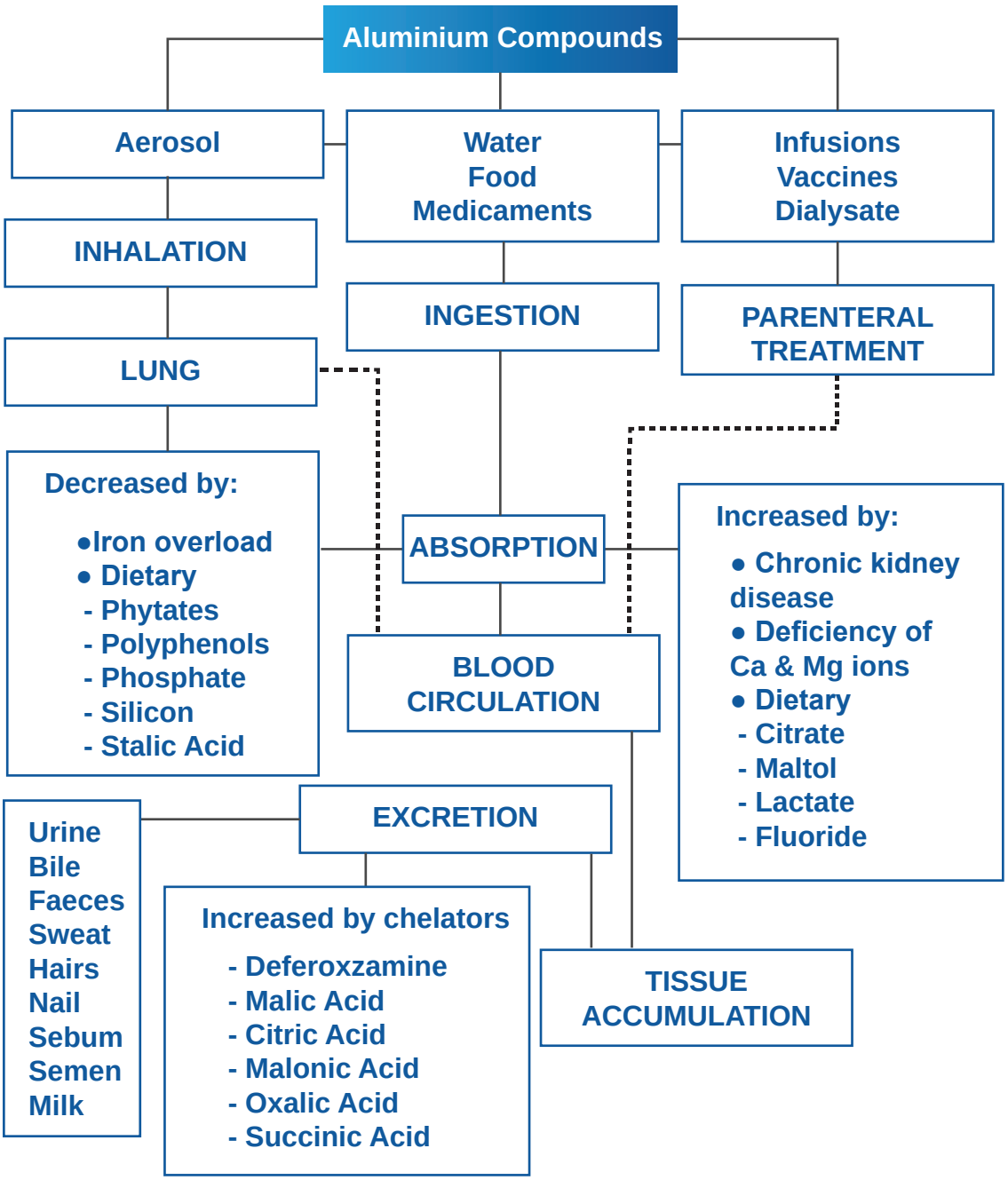
- Toxicosis associated with Al exposure is the pathological condition or disease caused by the toxic actions of Al and its compounds.
- Aluminum exposure include non-occupational exposure from the environment, contamination in food, medicine, and water.

2.2 ROUTE OF EXPOSURE

Main route of exposure is inhalation and oral route.

2.3 TOXICOKINETIC

The total body burden of Al in healthy humans has been reported to be approximately 30–50 mg/kg body weight and normal levels of Al in serum are approximately 1–3 µg/L (Krewski et al., 2007). The mean serum Al level in 44 non-exposed persons who did not use antacids was reported to be 1.6 µg/L (Valkonen and Aitio, 1997) and Chen et al. (2010) reported that values in hemodialysis patients were ten-fold higher than the values in unexposed individuals. About one-half of the total body Al is in the skeleton, and the levels in human bone tissue range from 5 to 10 mg/kg (Anon, 2008c). Al has also been found in human skin, lower gastrointestinal tract, lymph nodes, adrenals, parathyroid glands, and in most soft tissue organs (Anon, 2008b).



The Al ion in blood circulation is eliminated primarily by the kidneys (about 95%) in the urine, presumably as Al citrate (Shirley and Lote, 2005; Krewski et al., 2007; Anon, 2008c).

Toxic effects of Al arise mainly from its pro-oxidant activity which results in oxidative stress, free radical attack and oxidation of cellular proteins and lipids (Exley, 2013).

2.4 HAZARD CLASSIFICATION

- GHS CLASSIFICATION

  Depends on the compound. please refer to respective SDS.

- Carcinogenic Effects

IARC has stated (IARC 1984) that “the available epidemiological studies provide limited evidence that certain exposures in the aluminum production industry are carcinogenic to humans, giving rise to cancer of the lung and bladder. A possible causative agent is pitch fume.” It is important to emphasize that the potential risk of cancer in the aluminum production industry is probably due to the presence of known carcinogens (e.g., PAHs) in the workplace and is not due to aluminum or its compounds.

3.0 Health Effects Monitoring

The health effects listed below are a result of accumulation in the body through various sources, not necessarily occupational exposure alone.

3.1 Acute effects

Occupational acute health effects are normally associated with the irritation effects to the airway system. It's also an eye and skin irritant.

Metal fume fever is also an acute effect.

3.2 Chronic effects

- **Pulmonary effect**

  Pulmonary lesions in humans linked to Al exposure during production of Al products include granulomatous pneumonia, pulmonary granulomatosis, pulmonary fibrosis, pulmonary alveolar proteinosis and desquamative interstitial pneumonia (Chen et al., 1978; Herbert et al., 1982; Miller et al., 1984; De Vuyst et al., 1987; Jederlinic et al., 1990; Taiwo, 2014; Iijima et al., 2017). Asthma may be caused by Al exposure (Burge et al., 2000), though the asthma among Al workers may be due to other chemical factors like gases and smoke (Taiwo et al., 2006). Reactive airways dysfunction syndrome was rarely reported among Al smelter workers (Wesdock and Arnold, 2014).

- **Cardiovascular effect**

  Toxic myocarditis, myocardial hypokinesia, left ventricular thrombosis and myocardial dysfunction were reported in a case of Al phosphide intoxication (Hangouche et al., 2017).
  Ischemic stroke due to thrombosis in the right middle cerebral artery was reported as the delayed complication of Al phosphide poisoning (Abedini et al., 2014). However, other Al compounds may not cause cardiovascular lesions.

No studies regarding cardiovascular effects of various forms of aluminum following acute- or intermediate-duration inhalation exposure in humans were identified.

- **Gastrointestinal effect**

In individuals that are genetically susceptible to Crohn's disease, Al is linked to the induction and persistence of the chronic relapsing intestinal inflammation (Lerner, 2007). Inflammatory bowl diseases, consisting of disease entities like Crohn's disease and ulcerative colitis, are characterized by excessive intestinal inflammation and experimental evidence in mice indicates that Al promotes intestinal inflammation, thereby implicating Al in the pathogenesis of inflammatory bowl diseases (de Chambrun et al., 2014).

No studies regarding gastrointestinal effects of various forms of aluminum following acute-, intermediate-, or chronic-duration inhalation exposure in humans were identified.

- **Haematological effect**

Most evidence are in animal studies. Causing anaemia.

- **Neurologic effects**

In humans, Al accumulation in the brain and scalp hairs has been associated with neurodegenerative diseases such as dialysis-associated encephalopathy, Alzheimer's disease, Parkinson's disease (dementia), amyotropic lateral sclerosis, multiple sclerosis, and autism (King et al., 1981; Savory et al., 1996; Kawahara and Kato-Negishi, 2011; Arain et al., 2015; Jones et al., 2017; Mirza et al., 2017; Mold et al., 2018).

- **Musculoskeletal effect**

The major myopathy induced by Al exposure is macrophagic myofasciitis (aluminic granuloma) associated with chronic arthromyalgia or myalgia and chronic fatigue syndrome (Exley et al., 2009; Gherardi and Authier, 2012; Rigolet et al., 2014; Gherardi et al., 2016; Miller, 2016).

The bone diseases associated with Al exposure are osteoporosis, osteomalacia, rickets, exostosis, osteodystrophy, and osteitis fibrosa (Sherrard et al., 1985; Chappard et al., 2016; Rodríguez and Mandalunis, 2018; Klein, 2019).

- **Reproductive and developmental effects**

  Human reproduction may be affected negatively by Al exposure (Klein et al., 2014; Mouro et al., 2017). Human semen and spermatozoa contain Al and patients with oligospermia had higher Al concentration than healthy individuals (Klein et al., 2014).

- **Hepato-renal and pancreatic effects**

  No studies were identified regarding hepatic effects in humans following acute- or chronic-duration inhalation exposure to various forms of aluminum. Intermediate occupational inhalation exposure to aluminum fumes, dusts, or powders did not affect liver function or hepatic microanatomy in a group of seven workers as determined from biopsy samples (Mussi et al. 1984).

  No studies regarding renal effects in humans following acute-duration inhalation exposure to various forms of aluminum were identified.

- **Endocrine, Dermal and Ocular effects**

  No studies were identified regarding endocrine, dermal, or ocular effects in humans following acute- or intermediate-duration inhalation exposure to various forms of aluminum.

4.0 Medical Surveillance Programme

4.1 Indications

Workplace personal exposures exceed 50% of PEL.

4.2 Clinical Examination and Relevant Investigations

- Target organs assessment focusing on the respiratory system.
- Spirometry
- Chest X-ray when indicated.

4.3 Pre-placement Medical Examination

Clinical examination and relevant investigation as listed in 4.2.

4.4 Periodic Medical Examination

- Clinical examination and relevant investigation as listed in 4.2.
- Frequency of periodic medical examination with no abnormal findings – annual.

4.5 Biological Monitoring

- Methods available to determine the level of aluminum in the blood, but the correlation with occupational exposure is yet to be established, thus still not usable.
- There are no known biomarkers of effects yet.

5.0 Medical Removal Protection

- **Indications for removal**
  - Clinical findings
    - Evidence of health effects that is suspected to be caused by CHTH or may be worsened by CHTH.
  - Target organ function abnormalities.
  - Pregnancy and breastfeeding.
- **Temporary MRP**
  - MRP based on medical condition.
    - The determination of the temporary MRP duration should consider the following aspects:
      - expected duration of diagnostic procedures.
      - duration of recovery.
- All cases recommended for MRP, suspected cases of poisoning and excessive absorption must be notified to the DG of DOSH.

6.0 Return to Work

- Weekly assessment until medical condition normalises.
- Return to work based on:

i. Medical condition:
   - Signs and symptoms and abnormal biochemical results have disappeared.

ii. Workplace management:
   - Ensure workplace hygiene is safe and healthy and does not place the worker at increased risk of material impairment to health from exposure to aluminum.

7.0 Notification to DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed in NADOPOD 2004 using the relevant notification forms.

8.0 Treatment and Management

- All cases of poisoning must be immediately removed from exposure and referred for hospital treatment.

9.0 Preventive Measures

- Good workplace hygiene
- Adequate ventilation
- Approved personal protective equipment including appropriate respirator for workers working in in areas of aluminium exposure.
- Appropriate signage

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DIESEL

1.0 Description

Diesel is a complex mixture of chemicals mainly obtained from the distillation of crude oil.

It is refined mineral oil where most of the hazardous substance in the crude oil was already removed through the distillation process. Diesel is produced by mixing fractions of crude oil distillates (petrochemicals) with various, brand-specific additives.

Diesel is not considered to be particularly toxic and accidental poisoning is very rare. However, if diesel is swallowed, medical advice should be obtained immediately as there is a small risk of short-term lung damage if vomiting occurs or if droplets of diesel are inhaled. Long-term skin exposure to diesel may result in eczema (dermatitis) and should be avoided.

Diesel is manufactured, thus reference to the SDS of the respective manufacturer is required. Additives in the diesel may pose a unique hazard to the mixture.

1.1 Synonym

Diesel Fuel

1.2 Occupational Exposure Limits (OEL)

	USECHH 2000 (Eight-hour time weighted average limit)
Diesel fuel	Not available

Note:

Diesel airborne release is regulated as mineral oil mist when there is mist formation.

1.3 Physicochemical Properties

- It is a sweet-smelling liquid.
- Possess low volatility with a vapour pressure <1 mm Hg and specific gravity between 0.82 and 0.95 at 15°C (water = 1)
- Flammable with lower explosive limit 0.6% and upper explosive limit 6.5%.
- Water solubility 0.5 mg 100 mL-1
- Vapours may be violently reactive with air.

- Reaction or degradation products may liberate irritating or toxic fumes during combustion.

1.4 Material Use

- Mainly used as fuel.

2.0 Toxicity

2.1 Source of occupational exposure

- Fuelling vehicles and machineries
- As a cleaning substance, however this practice is not allowed by best practicesand CHRA, due to significant skin exposure.
- Agriculture use by mixing with certain herbicides.

2.2 Route of exposure

- Skin
- Inhalation (uncommon to be exposed to oil mist).

2.3 Toxicokinetic

- As diesel is a mixture of chemicals, there is no definitive ADME data.
- The onset of local or systemic effects following dermal, oral, or pulmonary exposure indicates that these are all potential routes of absorption for diesel.

2.4 Hazard Classification

Diesel

GHS Classification
H304 May be fatal if swallowed and enters airways.
H315 Causes skin irritation.
H332 Harmful if inhaled.
H351 Suspected of causing cancer.
H373 May cause damage to organs (Thymus, Liver, Bone Marrow) through prolonged or repeated exposure.

	Classification Code	H-Code	Signal Word
Fuels, diesel, coal solvent extn., hydrocracked hydrogenated;	Carc. 2	H351	Warning

Source: European Union, Commission Regulations (EC) 1272/2008.

- **Cancer Classification IARC**
  - Group 3. Not classifiable. There is "inadequate evidence" to classify diesel as a human carcinogen and "limited evidence" for the carcinogenicity of diesel to experimental animals.

3.0 Health Effects Monitoring

3.1 Acute Effects and Chronic Effects

- **Delayed effects following an acute exposure**

The available literature pertaining to the long-term effects of diesel following acute exposure is mainly concerned with jet fuels and thus its relevance is uncertain due to the presence of chemical additives and different hydrocarbon profile. In general, single (acute) oral, dermal, and ocular exposures do not appear to result in persistent effects.

- **General toxicity**

Under normal industrial or domestic use, dermal contamination is the most likely exposure scenario. Chronic or repeat exposure to diesel may result in dermatitis although there is some evidence to suggest that hyperkeratosis may be a common feature of regular contact with diesel.

- **Inhalation**

There are currently no unequivocal studies to relate chronic or repeated diesel exposures to long-term pulmonary dysfunction (other than that associated with aspiration of contaminated water or vomit). There is limited evidence to suggest that chronic exposure to long-chain hydrocarbon mixtures may be associated with a tightness of chest and breathing difficulties, although a review of the duration and extent of exposure in such circumstances was not reported.

- **Ingestion**

  Chronic, oral exposure to diesel is unlikely to arise under normal circumstances and there is currently no data available on the chronic effects of diesel ingestion in humans.

- **Dermal/Ocular exposure**

  There are no reports on the effects of chronic ocular exposure to diesel in humans. Acne and folliculitis have been reported in one subject who may have received chronic (occupational) exposure to diesel. Hyperkeratosis was also reported to be associated with prolonged skin contact with diesel.

- **Genotoxicity**

  There was no adequate evidence to support genotoxicity.

4.0 Medical Surveillance Programme

4.1 Indications

Exposed to mineral oil mist that exceeds 50% of PEL and/ or prolonged skin contact with diesel.

4.2 Clinical Examination and Relevant Investigations

Target organs are the respiratory system if exposed to oil mist, and the skin if there is significant and prolonged skin contact.

- History taking and physical examination.
- Spirometry
- Chest X-ray if indicated.

4.3 Pre-placement medical examination

- Non-specific.

4.4 Periodic Medical Examination

- Clinical examination and relevant investigations as listed in 4.2.
- Frequency of periodic medical examination - annual

4.5 Biological Monitoring

- There are currently no BEI determinants established.

5.0 Medical Removal Protection

- **Indications for removal**
  - Clinical findings
    - Evidence of health effects
  - Target organ function abnormalities
  - Others
    - Medical conditions of the skin
- **Temporary MRP**
  - MRP based on medical condition.
    - The determination of the temporary MRP duration should consider the following aspects:
      - expected duration of diagnostic procedures.
      - duration of recovery.
- All cases recommended for MRP, suspected cases of poisoning and excessive absorption must be notified to the DG of DOSH.

6.0 Return to Work

- Weekly assessment until medical conditions return to normal.
- Return to work based on:

i. Medical condition
   - Signs and symptoms have resolved and abnormal biochemical results have normalised.

ii. Workplace management
   - Ensure workplace hygiene is safe and healthy and does not place the worker at increased risk of material impairment to health from exposure to diesel.

7.0 Notification to DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed in NADOPOD 2004 using the relevant notification forms.

8.0 Treatment and Management

- All cases of poisoning must be immediately removed from exposure and referred for hospital treatment.

9.0 Preventive measures

- Good workplace hygiene
- Adequate ventilation
- Approved personal protective equipment including appropriate respirator for workers working in areas of diesel exposure.
- Appropriate signage

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FORMALDEHYDE

1.0 DESCRIPTION

Formaldehyde is a naturally occurring organic compound with the formula CH2O (H−CHO). The pure compound is a pungent-smelling colorless gas that polymerises spontaneously into paraformaldehyde (refer to section Forms below), hence it is stored as an aqueous solution (formalin). It is the simplest of the aldehydes (R−CHO). The common name of this substance comes from its similarity and relation to formic acid. Formalin may contain methanol. Formaldehyde will be significantly more released to the work environment compared to methanol.

Formaldehyde resins may also release a certain amount of formaldehyde to the work environment when heated.

1.1 Synonym

Methanal, methyl aldehyde, methylene oxide, formalin, oxymethylene, formic aldehyde, Lysoform, methylene glycol, oxomethane, oxymethylene, polyoxymethylene glycols.

1.2 Occupational Exposure Limit (OEL)

	USECHH 2000 (Ceiling Limit)
Formaldehyde	0.3 ppm, 0.37 mg/m^3

1.3 Physicochemical Properties

- Colourless aqueous solution with a pungent odour.
- Flammable gas at room temperature

1.4 Material Use

- Wide industrial usage in fungicide, germicide, disinfectants, and embalming fluids.
- Resins in manufacturing of composite wood products.
- Building materials and insulation.
- Preservatives in some medicines, cosmetics, dishwashing liquids and fabric softeners
- Used in manufacture of artificial silk and textiles, latex, phenol, urea, thiourea and melamine resins, dyes, inks, cellulose esters and other organic molecules, mirrors, and explosives.

- Used in the manufacture of paper, photographic and furniture industries.
- Intermediate in drug manufacture and pesticides.

2.0 TOXICITY

2.1 Source of occupational exposure

- Free release from the resins. Manufacture of composite wood products, paper,furniture, artificial silk and textiles, latex, phenol, urea, thiourea and melamine resins, dyes, and inks. Usually, significant formaldehyde released occur during heating of the resins. Free release from formaldehyde resins is also possible.

- Formalin use: embalming industry
- Formalin for tissue preservative. Healthcare professionals and laboratory staff.
- Use of fungicide, germicide, and disinfectant fluids.

2.2 Route of exposure

- Inhalation
- Ingestion
- Skin and/or eye contact

2.3 Toxicokinetic

Absorption	• Readily absorbed via inhalation into the upper respiratory tract. • Absorbed well by the gastrointestinal tract. • May be absorbed through the skin but at a lesser extent. • Less than a third is absorbed into the blood following breakdown by cells lining the mouth, nose, throat, and airways.
Distribution	Removed from mucosal blood supply and distributed throughout the body.
Metabolism	• Reacts immediately with primary and secondary amines, thiols, hydroxyls, and amides to form methylol derivatives. • Enzymatic processes can oxidise formaldehyde to formic acid or to carbon dioxide by formaldehyde dehydrogenase (FDH), aldehyde dehydrogenase and in rare instances, catalase. • Converted and excreted as carbon dioxide in the air and as formic acid in the urine.

	• Formed endogenously in the body as well as during oxidative demethylation of xenobiotics leading to deposition in the liver
Excretion	• Excreted mostly in metabolized form. • Animal studies show predominant excretion in the air as carbon dioxide. • Primary excretion is via exhalation. • Smaller amounts are excreted in faeces and urine as formate salts and other metabolites. • Metabolized and excreted from the plasma with a half-life of 1 to 1.5 minutes.

- Readily reacts with macromolecules such as DNA, RNA, and protein in the body.
- Rapid metabolism of formaldehyde following skin contact makes systemic effects unlikely to occur.

2.4 Hazard Classification

Classification Code	Hazard Classification	H-Code	Signal
Carc. 1B	Carcinogenicity category 1B	H350	Danger
Muta. 2	Germ cell mutagenicity category 2	H341	
Acute Tox. 3 (inh)	Acute toxicity category 3 (inhalation)	H331	
Acute Tox. 3 (dermal)	Acute toxicity category 3 (dermal)	H311	
Acute Tox. 3 (oral)	Acute toxicity category 3 (oral)	H301	

Skin Corr. 1B	Skin corrosion or irritation category 1B	H314	
Eye Dam. 1	Serious eye damage or eye irritation category 1	H318	
Skin Sens. 1	Skin sensitization category 1	H317	
STOT SE 3	Specific target organ toxicity - single exposure category 3	H335	

Source: ICOP on Chemicals Classification and Hazard Communication 2019

- Possible risk of gene damage/ impaired fertility (Sittigs, 2017)
- **Cancer classification IARC**

  Group 1. Carcinogenic to humans.

3.0 HEALTH EFFECTS MONITORING

Formaldehyde possesses irritating properties to the nose and pharynx. Under controlled exposure conditions, symptoms of irritation were noted by healthy individuals exposed to formaldehyde concentrations of 2-3 ppm for a duration between 40 minutes and three hours (for details, see Table 30 in IARC Monograph Volume 88 2006).

Contact dermatitis is a known effect of formaldehyde exposure. Chronic exposure has been known to cause chronic inflammation, loss of cilia, mild dysplasia, hyperplasia, and squamous metaplasia. Confounding factors such as exposure to wood dust may influence the latter.

There is strong but insufficient evidence to conclude that formaldehyde may cause leukemia. There is however sufficient evidence for the carcinogenicity of formaldehyde to the nasopharynx in humans.

3.1 Acute Effects

System / Organ	Acute Effects
Cardiovascular	• Hypotension • Cardiovascular collapse

Eyes	• Corrosive to the eyes • Burns • Lacrimation • Irritation at levels between 0.05 and 2.0 ppm • Corneal damage
Ear, Nose, Throat	Irritation of nose and throat at exposures of 0.25 – 0.45 ppm.
Gastrointestinal	• Damage to stomach and intestine causing: o Nausea o Vomiting o Abdominal Pain o Diarrhea
Nervous System CNS and PNS	• Lethargy • Dizziness • Convulsions • Coma
Renal and Genitourinary	• Kidney damage • Nephritis • Hematuria
Respiratory	• Corrosive to respiratory tract. • Difficulty breathing. • Coughing and wheezing at levels between 0.4 ppm and 0.8 ppm. • Pneumonia • Chest tightness and shortness of breath at levels between 0.4 ppm and 0.8 ppm. • Pulmonary edema. • Irritation of lung at concentrations of 4 ppm which may cause bronchitis and laryngitis. • Impaired breathing at high levels. • Serious lung damage. • Sensitization; asthmatic reactions in sensitized individuals.
Skin	• Irritation • Itching • Burning • Drying • Allergic reaction

3.2 Chronic Effects

Formaldehyde is a potent skin and respiratory sensitization agent. Causes allergic sensitization of the skin and airways.

System / Organ	Acute Effects
Ear, Nose and Throat	Nasopharyngeal cancer.
Reproductive	Could increase chances of having fertility issues or miscarriages.
Respiratory	• Lung cancer • Respiratory congestion • Coughing • Shortness of breath • Subsequent contact causes asthma (symptoms worsen if exposure persists).
Skin	• Repeated skin contact causing drying and scaling. • Allergic reactions in some individuals. • Subsequent contact cause skin rashes (symptoms worsen if exposure persists).

4.0 MEDICAL SURVEILLANCE PROGRAMME

4.1 Indications

Any work where workers are exposed to levels exceeding Ceiling limit and/or where there is significant risk of absorption.

4.2 Clinical Examination and Relevant Investigations

- Clinical examination
- Chest X-ray
- Lung function tests (FEV1 , FVC, FEV1 /FVC, FEF)
- Speculum examination of nasal mucosa for signs of irritation

4.3 Pre-placement Medical Examination

- Clinical examination and relevant investigation as listed in 4.2.
- History of smoking (smoking reduces clearance of deposited materials in respiratory tract and confounding factor in investigation of chronic respiratory disease), skin disease, previous exposure to formaldehyde or other dermal sensitizers and atopic or allergic diseases.

- Decision for fitness to work.
    - Workers with disease of the skin and respiratory system should not work in areas where there is significant formaldehyde exposure.

4.4 Periodic Medical Examination

- Clinical examination and relevant investigation as listed in 4.2.
- Frequency of periodic medical examination with normal findings – annual.

4.5 Biological Monitoring

- No biological exposure indices determinant based on ACGIH 2022 TLVs and BEIs.

5.0 MEDICAL REMOVAL PROTECTION

- Indications for removal
    - Clinical findings
        - Evidence of health effects that is suspected to be caused by CHTH or may be worsened by CHTH.
    - Target organ function abnormalities.
    - Pregnancy and breastfeeding.
- Temporary MRP
    - MRP based on medical condition.
    - The determination of the temporary MRP duration should consider the following aspects:
        - expected duration of diagnostic procedures.
        - duration of recovery.
- All cases recommended for MRP, suspected cases of poisoning and excessive absorption must be notified to the DG of DOSH.
- Follow-up medical examination shall be conducted within six months following medical removal.

6.0 RETURN TO WORK

- Weekly assessment until medical conditions return to normal.
- Return to work based on:

i. Medical condition

Signs and symptoms of excessive exposure to formaldehyde have resolved.

ii. Workplace management

Ensure workplace hygiene is safe and healthy and does not place the worker at increased risk of material impairment to health from exposure to formaldehyde.

7.0 NOTIFICATION TO DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed NADOPOD 2004 using the relevant notification forms.

8.0 TREATMENT AND MANAGEMENT

- All cases of poisoning must be immediately removed from exposure and referred for hospital treatment.
- All contaminated clothing should be removed and washed immediately with soap and water.
- If inhalation or ingestion has occurred, remove immediately and refer for hospital treatment.
- Medical observation is recommended for 24 to 48 hours after excessive inhalation exposure due to delayed effects of pulmonary edema.
- Cigarette smoking may exacerbate pulmonary effects and should be discouraged for at least 72 hours after exposure.

9.0 PREVENTIVE MEASURES

- Adequate ventilation provided in areas of formaldehyde work.
- Workers should be supplied with appropriate protective clothing including eye protection, gloves, and clothing to prevent skin or eye contact.
- Maintain good workplace hygiene.
- Appropriate signage.

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RESPIRATORY IRRITANTS: CALCIUM CARBONATE

1.0 DESCRIPTION

1.1 Synonyms

- Calcium salt of carbonic acid [Note: Occurs in nature as limestone, chalk, marble,dolomite, aragonite, calcite, and oyster shells.]

1.2 Occupational Exposure Limits (OEL)

USECHH 2000 (8 hrs TWA Airborne Concentration)	
Calcium carbonate	
Inhalable dust	10 mg/m^3
Respirable dust	4 mg/m^3

1.3 Physicochemical Properties

- Calcium carbonate is a white, odourless powder, or crystalline solid.
- Calcium carbonate is soluble in concentrated mineral acids.

1.4 Material Use

- Used as a source of lime.
- Used as neutralising agent.
- Used in manufacturing of rubber, plastics, paint, and coatings; sealants, paper, dentifrices, ceramics, putty, polishes and cleaners, insecticides, inks, and cosmetics; whitewash; Portland cement; antacids; analytical chemistry, and others.

2.0 TOXICITY

2.1 Sources of potential occupational exposure

- Used in manufacturing of rubber, plastics, paint, and coatings; sealants, paper, dentifrices, ceramics, putty, polishes and cleaners, insecticides, inks, and cosmetics; whitewash; Portland cement; antacids; analytical chemistry, and others.

2.2 Route of exposure

- Inhalation of dust
- Ingestion

2.3 Toxicokinetics

Calcium carbonate	
Absorption	Mainly through the respiratory system.
Distribution	Localised to the respiratory system.
Metabolism	Not applicable
Excretion & Half-life	Not applicable

2.4 Hazard Classification

Hazard classification	H-Code
May cause skin irritation	H315
May cause eye irritation	H320
May cause respiratory irritation	H335

Source: ICOP on Chemicals Classification and Hazard Communication 2014.

- **Cancer Classification IARC**
  - Not listed

3.0 Health Effects Monitoring

3.1 Acute Effects and Chronic Effects

System/Organ	Acute Effects and chronic effects
Ear, Nose & Throat	Inhalation can cause irritation to nose.

Eye	Eye contact can cause irritation.
Gastrointestinal	• Large amounts can cause irritability, nausea, dehydration, and constipation. Estimated lethal dose is over 2 lb (nearly 1 kg).
Respiratory	• Irritation and inflammatory effects of the airway and the lungs. • Chronic high exposures can lead to neumoconiosis.
Skin	• Acute irritations • Chronic irritation may lead to irritant dermatitis.

4.0 MEDICAL SURVEILLANCE PROGRAMME

4.1 Indications

- Any occupation where workers are liable to be exposed to calcium carbonate above 50% of PEL.

4.2 Clinical Examinations and Relevant Investigations

- Clinical examination with emphasis on the respiratory system and the skin.
- Spirometry
- Chest X-ray when clinically indicated.

4.3 Pre-placement Medical Examination

- Clinical examination and relevant investigations as listed in 4.2.
- Decision for fitness to work.
  - Chronic lung conditions may worsen when exposed to respiratory irritants.

4.4 Periodic Medical Examination

- Clinical examination and relevant investigations as listed in 4.2.
- Frequency of periodic medical examination – annual.

4.5 Biological Monitoring

- There is no BEI determinant monitoring.

5.0 MEDICAL REMOVAL PROTECTION

- **Indications for removal**
  - Clinical findings
    - Evidence of health effects that is suspected to be caused by CHTH or may be worsened by CHTH.
  - Target organ function abnormalities

- **Temporary MRP**
  - MRP based on medical condition.

    The determination of the temporary MRP duration should consider the following aspects:
    - expected duration of diagnostic procedures.
    - duration of recovery.

All cases recommended for MRP, suspected cases of poisoning and excessive absorption must be notified to the DG of DOSH.

6.0 RETURN TO WORK

- Return to work based on:
  i. Medical condition.
     - Signs and symptoms and abnormal biochemical results have resolved.
  ii. Workplace management.
     - Ensure workplace hygiene is safe and healthy and does not place the worker at increased risk of material impairment to health from exposure to calcium carbonate.

7.0 NOTIFICATION TO DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed in NADOPOD 2004 using the relevant notification forms.

8.0 TREATMENT AND MANAGEMENT

- All cases of poisoning must be immediately removed from exposure and referred for hospital treatment.

9.0 PREVENTIVE MEASURES

- Approved personal protective equipment including appropriate respirator for workers working in areas of calcium carbonate exposure.
- Adequate ventilation.
- Good workplace hygiene.
- Appropriate signage.

10.0 REFERENCES

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RESPIRATORY IRRITANTS: CHLORINE AND CHLORINE PRODUCTS

1.0 DESCRIPTION

There are chlorine gas and chlorine products which are used in the industries.

At room temperature, chlorine is a yellow-green gas with a pungent irritating odour. Under increased pressure or at temperatures below -30ºF, it is a clear, amber-coloured liquid. It is generally shipped in steel cylinders as a compressed liquid. Chlorine is only slightly soluble in water, but on contact with moisture it forms hypochlorous acid (HClO) and hydrochloric acid (HCl); the unstable HClO readily decomposes, forming oxygen free radicals. Because of these reactions, water substantially enhances chlorine's oxidizing and corrosive effects.

There are 5 types of Chlorine products: Sodium hypochlorite, Lithium hypochlorite, Calcium hypochlorite, Dichlor, and Trichlor.

Sodium Hypochlorite

Sodium Hypochlorite is a liquid Chlorine and has around 10-12% available Chlorine. Available Chlorine (AC) is the amount of Chlorine released in the water to disinfect. Bleach, which contains Sodium Hypo, only has 5% AC which is why bleach is not a good pool disinfectant. Because of its liquid nature, Sodium Hypo is usually applied to a pool through an automatic chemical feeder. Big water parks and large commercial pools are the common users of Sodium Hypochlorite.

Side note: Salt pools are still Chlorine pool because the salt cell breaks the salt down into Sodium Hypochlorite.

Lithium Hypochlorite

Lithium Hypochlorite is granular Chlorine with a 35% AC. Lithium dissolves very quickly making it great for super chlorinating (shocking) vinyl lined and fiberglass pools and those who have problems with hard water and Calcium levels. But its low active strength and high cost make it home pool disinfectant rather than commercial pool disinfectant.

Calcium Hypochlorite

Calcium Hypochlorite is commonly seen as granular Chlorine but also is in puck/tablet form. Calcium Hypo has an AC of 40-78% and it the most popular of the Chlorines. Calcium Hypo is used not only to shock a pool but is used in erosion feeders as the main way to disinfect a pool. Calcium Hypo is used regularly in both home and commercial pools and is usually what is seen on the local mart shelves.

Trichlor and Dichlor

As stated above, both are stabilised Chlorines making them perfect for outdoor pools and are usually seen as pucks/tablets/sticks though granular forms are available. Their AC level is usually around

80-90% and introduced to the water through chemical feeders or skimmers. Because of their high AC level, the granular form is commonly used to treat pool problems due to algae or a Chlorine demand.

1.2 Occupational Exposure Limits (OEL)

USECHH 2000 (8 hrs TWA Airborne Concentration)	
Chlorine	
Eight-hour time weighted average limit	**15 min – Short-term exposure limit**
0.5 ppm (1.5 mg/m^3)	1 ppm (3 mg/m^3)

Note: There are no Permissible Exposure Limits set for the chlorine products.

1.3 Physicochemical Properties

- Chlorine is a greenish-yellow gas with a pungent, irritating odour.
- It is not flammable, but it is a strong oxidizer, and contact with other materials may cause fire.

1.4 Material Use

- Chlorine is used as a bleach in the manufacture of paper and cloth.
- Used widely as a chemical reagent in the synthesis and manufacture of metal chlorides, chlorinated solvents, pesticides, polymers, synthetic rubbers, and refrigerants.

2.0 TOXICITY

2.1 Sources of Potential Occupational Exposure

- Bleaching
- Formation of chlorinated compounds
- Household cleaners

2.2 Route of Exposure

- Inhalation
- Eye and skin contact
- Ingestion

2.3 Toxicokinetics

Chlorine gas	
Absorption	Mainly through the respiratory system.

2.4 Hazard Classification

Classification code	Hazard classification	H-Code	Signal
Press. Gas[(c)]	Gases under pressure	H280/281[(d)]	Danger
Ox. Gas 1	Oxidizing gases category 1	H270	
Acute Tox. 3 (inh)	Acute toxicity category 3 - inhalation	H331	
Eye Irrit. 2	Eye irritation category 2	H319	
STOT SE 3	Specific target organ toxicity – single exposure category 3	H335	
Skin Irrit. 2	Skin irritation category 2	H315	

*(c) - State the category (compressed gas, liquefied gas, dissolved gas or refrigerated liquefied gas).
*(d) - For gases under pressure, state the relevant H-code based on its hazard category.

Source: ICOP on Chemicals Classification and Hazard Communication (Amendment) 2019.

Cancer Classification IARC

- Not listed

3.0 Health Effects Monitoring

3.1 Acute Effects

The toxic effects of chlorine gas are primarily due to its corrosive properties. The action of chlorine is due to its strong oxidising capability, in which chlorine splits hydrogen from water in moist tissue, causing the release of nascent oxygen and hydrogen chloride which produce major tissue damage. Alternatively, chlorine may be converted to hypochlorous acid which can penetrate cells and react with cytoplasmic proteins to form N-chloro derivatives that destroy cell structure. Symptoms may be apparent immediately or delayed for a few hours.

System/Organ	Acute Effects
Cardiovascular	• Tachycardia and initial hypertension followed by hypotension may occur. • After severe exposure, cardiovascular collapse may occur from lack of oxygen.
Eyes	• Low concentrations in air can cause burning discomfort, spasmodic blinking or involuntary closing of the eyelids, redness, conjunctivitis, and tearing. • Corneal burns may occur at high concentrations.
Respiratory	• Inhalation of higher concentrations of chlorine gas (>15 ppm) can rapidly lead to respiratory distress with airway constriction and accumulation of fluid in the lungs (pulmonary edema). • In symptomatic patients, pulmonary injury may progress over several hours. • Rapid breathing.
Skin	• Blue discoloration of the skin. • Chlorine irritates the skin and can cause burning pain, inflammation, and blisters. Exposure to liquefied chlorine can result in frostbite injury.
Others	• Metabolic acidosis may result from insufficient oxygenation of tissues. An unusual complication of massive chlorine • inhalation is an excess of chloride ions in the blood, causing an acid-base imbalance.

3.2 Chronic Effects

Chronic exposure to chlorine, usually in the workplace, may cause corrosion of the teeth. Multiple exposures to chlorine have produced flu-like symptoms and a high risk of developing reactive airways dysfunction syndrome (RADS).

System/Organ	Chronic Effects
Others	• Chlorine has not been classified for carcinogenic effects. However, the association of cigarette smoking, and chlorine fumes may increase the risk of cancer.

3.3 Acute And Chronic Effects of Chlorine Products

Chlorine products are respiratory and skin irritants. High volume, in powder form, it can produce severe corrosive effects when in contact with the respiratory system moisture. Irritation, inflammation, muscular spasm, and necrosis are possible effects to the respiratory system. High volume exposures may be life threatening.

Chronic irritations of the respiratory system may lead to lung conditions such as pneumoconiosis.

4.0 MEDICAL SURVEILLANCE PROGRAMME

4.1 Indications

- Any occupation where workers are liable to be exposed to chlorine above 50% of PEL.

4.2 Clinical Examinations and Relevant Investigations

- Clinical examination with emphasis on respiratory system and skin and eyes
- diseases.
  Lung function tests.
- Chest X-ray when clinically indicated.

4.3 Pre-placement Medical Examination

- Clinical examination and relevant investigations as listed in 4.2.
- Decision for fitness to work.
  - Workers with disease of the respiratory system, skin and eyes should not work in areas where there is significant chlorine exposure.
  - Chronic lung conditions may worsen when exposed to respiratory irritants over long period.

4.4 Periodic Medical Examination

- Clinical examination and relevant investigations as listed in 4.2.
- Consider chest X-ray after acute overexposure to chlorine gas.
- Frequency of periodic medical examination – annual.

4.5 Biological Monitoring

There are no biological exposure determinants for chlorine gas or chlorine products.

5.0 MEDICAL REMOVAL PROTECTION

- **Indications for removal**
  - Clinical findings
    - Evidence of health effects that is suspected to be caused by CHTH or may be worsened by CHTH.
  - Target organ function abnormalities

- **Temporary MRP**
  - MRP based on medical condition.

    The determination of the temporary MRP duration should consider the following aspects:
    - expected duration of diagnostic procedures.
    - duration of recovery.

All cases recommended for MRP, suspected cases of poisoning and excessive absorption must be notified to the DG of DOSH.

6.0 RETURN TO WORK

- Return to work based on:
  i. Medical condition.
     - Signs and symptoms and abnormal biochemical results have disappeared.

  ii. Workplace management.
     - Ensure workplace hygiene is safe and healthy and does not place the worker at increased risk of material impairment to health from exposure to chlorine and chlorine products.

7.0 NOTIFICATION TO DOSH

DOSH shall be notified within 7 days of confirmed occupational disease as listed in NADOPOD 2004 using the relevant notification forms.

8.0 TREATMENT AND MANAGEMENT

- All cases of poisoning must be immediately removed from exposure and referred for hospital treatment.

9.0 PREVENTIVE MEASURES

- Approved personal protective equipment including appropriate respirator for workers working in areas of chlorine exposure.
- Adequate ventilation.
- Good workplace hygiene.
- Appropriate signage.
- Using local exhaust ventilation for chemicals that may be harmful with a single exposure.
- Using general ventilation to control exposures to skin and eye irritants.

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Appendix 1: Case Study (Benzene)

Benzene

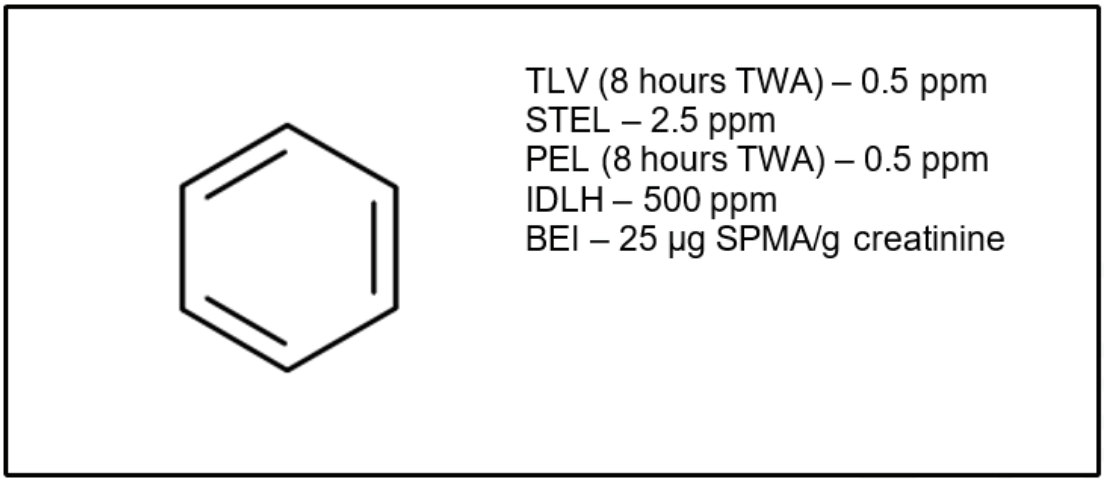


Figure 13. Exposure Limits for Benzene

Benzene is a chemical that is a colourless or light-yellow liquid at room temperature. It has a sweet odour and is highly flammable. Benzene evaporates into the air very quickly. Its vapor is heavier than air and may sink into low-lying areas. Benzene dissolves only slightly in water and will float on top of water. The PEL of benzene is as listed in Figure 13. The PEL of benzene is 0.5 ppm and this value is derived from human data based on Non-observable Effect Concentration (NOEC) for blood disorder upon safety adjustment.

a) Exposure

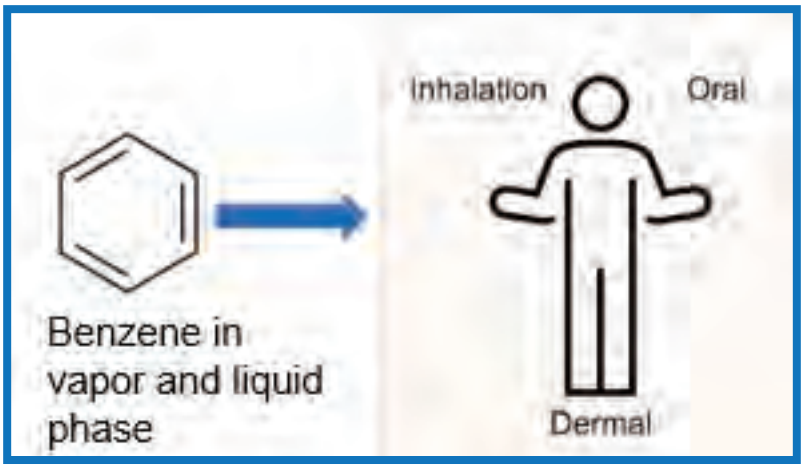


Figure 14. Exposure Routes for Benzene

Exposure to benzene can either be in vapor or liquid form. Inhalation represents the main route for benzene vapor. Nevertheless, benzene exposure can be via ingestion and transdermal (Figure 14). The amount of benzene absorbed through the skin over a long period can be significant, depending on exposure time and exposed skin surface areas.

b) Absorption

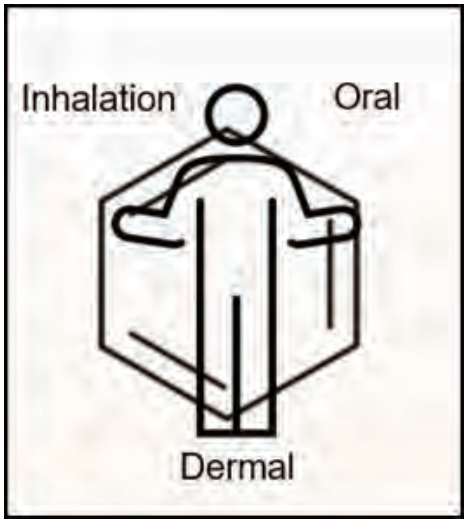


Figure 15. Absorption of Benzene

Benzene is readily absorbed by both test animals and humans from inhalation, oral, and dermal exposure. Absorption of benzene from inhalation and oral route is rapid and extensive. Although dermal absorption is less extensive, this exposure pathway can contribute to total body burden. (Figure 15)

c) Distribution

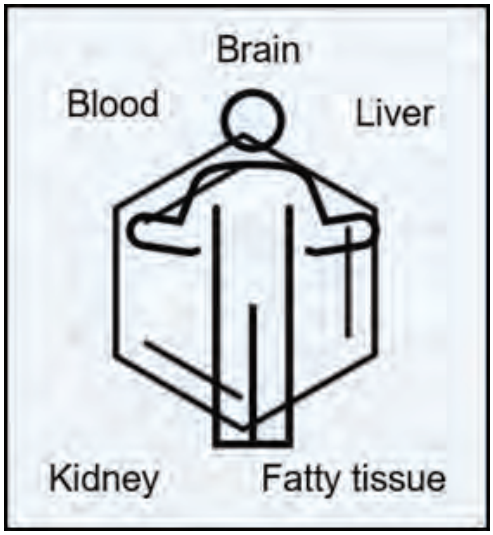


Figure 16. Distribution of Benzene

Absorbed benzene is rapidly distributed throughout the body and tends to accumulate in fatty tissues. Circulating benzene is preferentially taken up by lipid-rich tissues such as adipose and nervous tissue including brain. Benzene has also been detected in the lungs, liver, and kidneys. (Figure 16)

d) Metabolism

Benzene is metabolized, primarily in the liver, to a variety of hydroxylated and ring-opened products that are transported to the bone marrow where subsequent secondary metabolism occurs.[59] The first step in the metabolism of benzene is oxidation to the intermediate benzene oxide, which is catalyzed by cytochrome P450 (CYP) 2E1.[3] Benzene oxide is in equilibrium with the intermediate benzene oxepin. Benzene oxide and oxepin can undergo non-enzymatic rearrangement to phenol, hydrolysis to a dihydrodiol, ring opening to trans,trans-muconic acid (ttMA), or react with glutathione to form a pre-mercapturic acid conjugate. Phenol can undergo an additional oxidation catalyzed by CYP2E1 to hydroquinone and catechol. Hydroquinone and catechol can undergo further oxidation catalyzed by peroxidases to their respective quinones. These quinones, which are reactive species, can be reduced back to hydroquinone and catechol by NAD(P)H:quinone oxidoreductase 1 (NQO1). The schematic representation of benzene metabolism is shown in Figure 17.

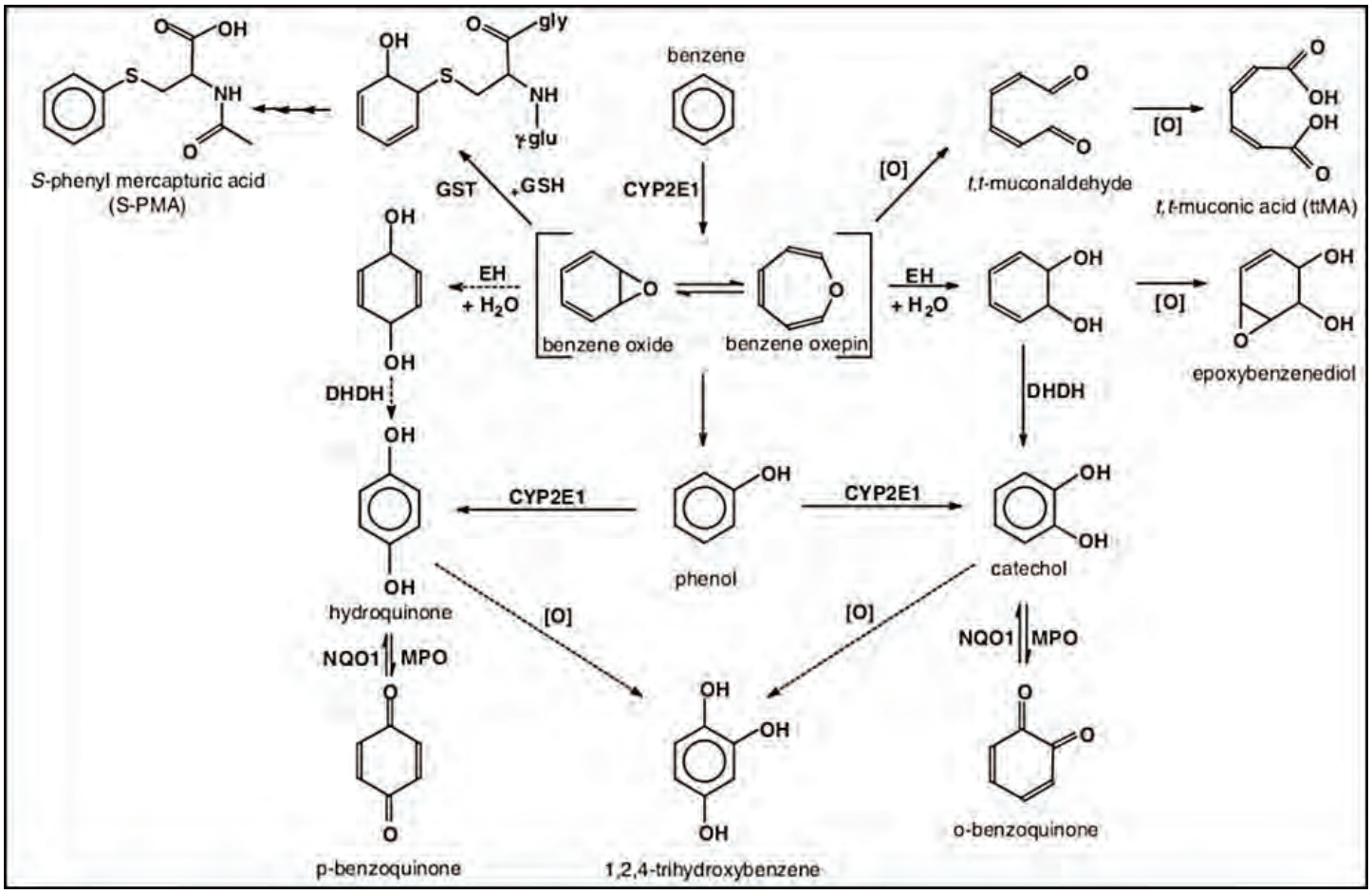


Figure 17. Metabolism of Benzene

Source: Arnold et al., 2013

e) Elimination/Excretion

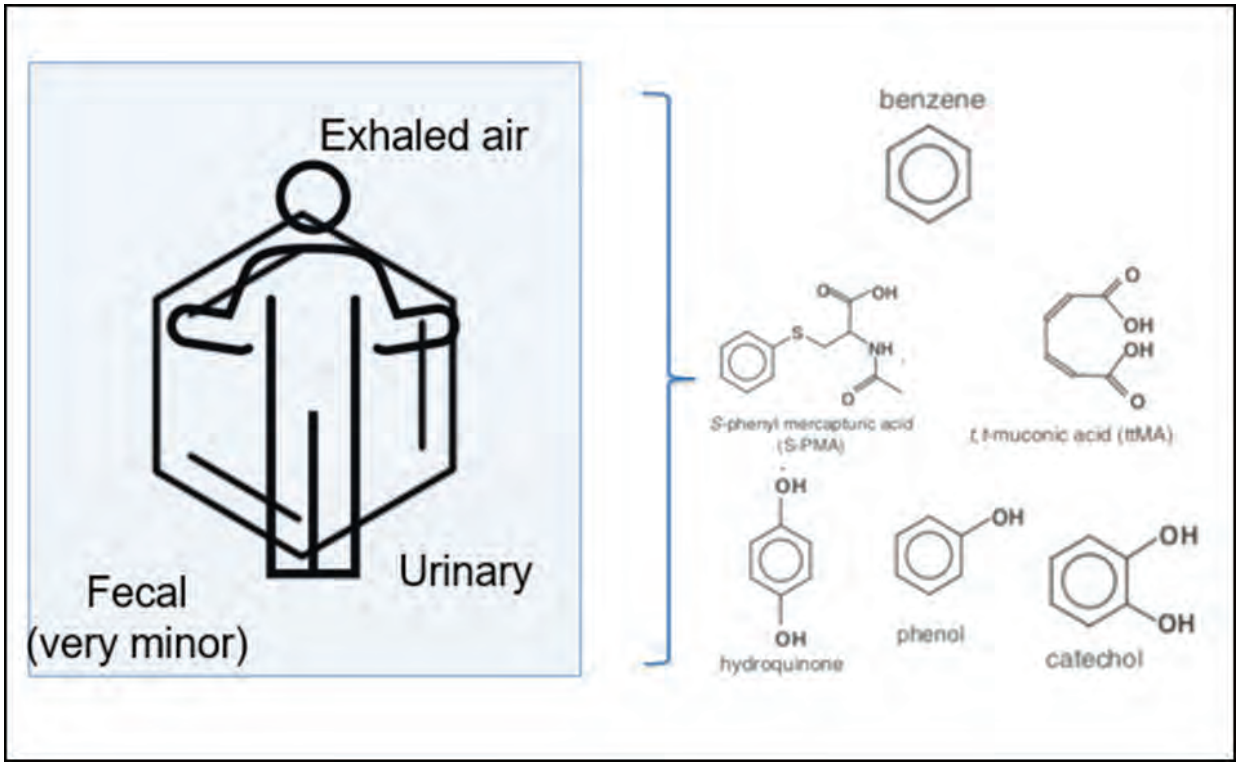


Figure 18. Elimination of Benzene

Unmetabolized absorbed benzene can be eliminated via exhalation and much lower amount excreted in the urine while fecal route is being an insignificant route. Several benzene metabolites such as SPMA, ttMA, phenol, hydroquinone, and catechol are excreted in urine (Figure 18). Some of the metabolites are being used as biomarker of exposure for benzene. SPMA is the most suitable biomarker for benzene exposure because there are not any known non-benzene sources for this metabolite as compared to other. Please refer to the table below for non-benzene sources of benzene urinary metabolites.

Non-benzene sources of benzene urinary metabolites.

Table 27. Metabolites of Benzene

Chemical	Source	Amount
SPMA	No known endogenous or exogenous sources	-

tMA	Diet (sorbitol) Europe	6-30 mg/d
	Diet (sorbitol) USA	25 mg/d
	Percentage of ttMA in smokers attributed dietary sorbic acid	10-50%
	Percentage of ttMA in non-smokers attributed to dietary sorbic acid	5-25%
Phenol	Diet and endogenous sources	0.2 mg/kg-bw/d
	Mainstream cigarette smoke	60-140 µg/cig
	Sidestream cigarette smoke	1.6-3.0 x mainstream smoke
	Over the counter medicines	Not quantified
Catechol	Diet and endogenous sources	0.3 mg/kg-bw/d
	Mainstream cigarette smoke	100-350 µg/cig
	Sidestream cigarette smoke	0.6-0.9 x mainstream smoke
Phenol	Diet and endogenous sources	0.1 mg/kg-bw/d
	Black and white photographic processing	Not quantified
	Mainstream cigarette smoke	110-300 µg/cig
	Sidestream cigarette smoke	0.7-0.9 x mainstream smoke

Source: Arnold et al., 2013.

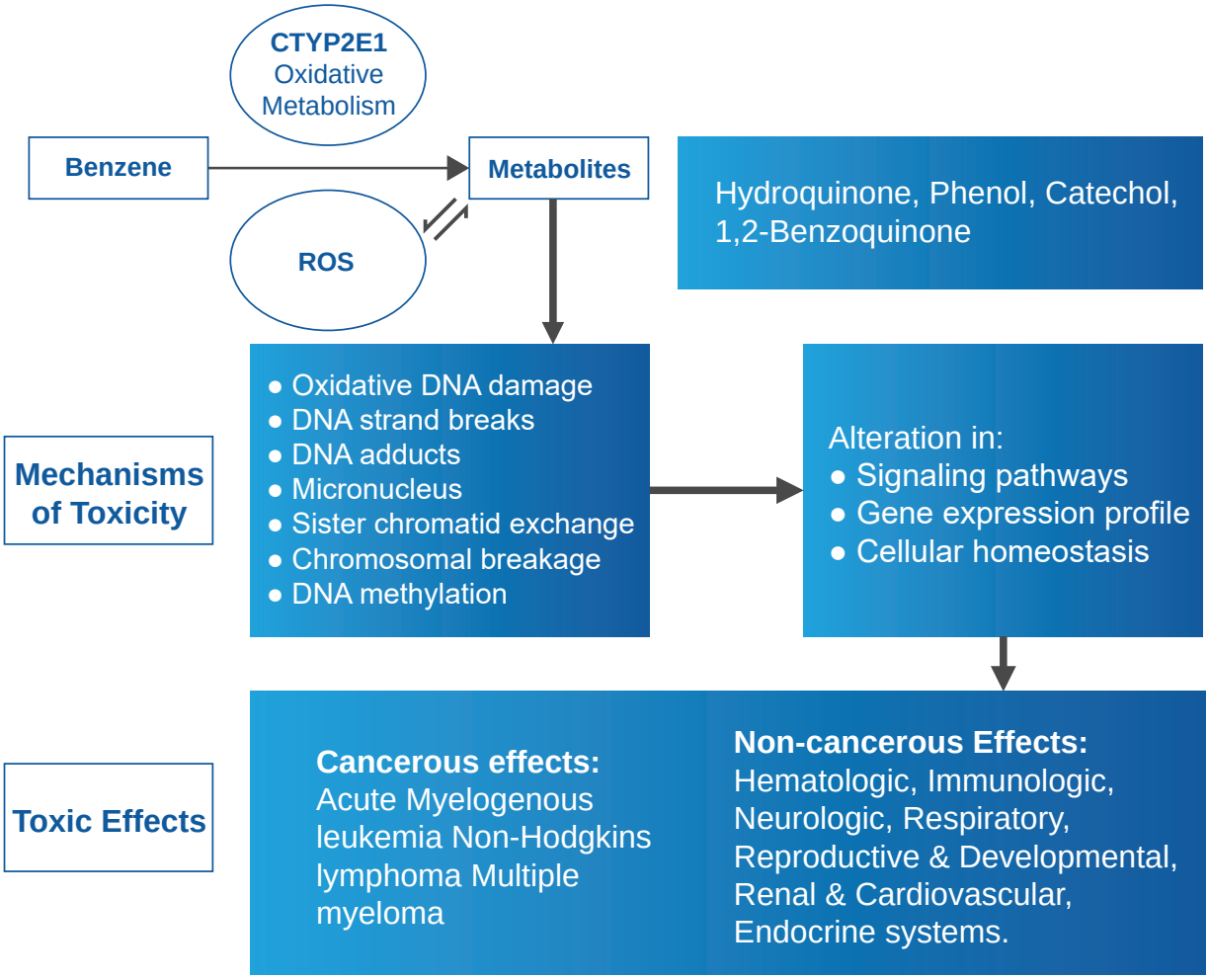
g) Toxicodynamic - Health effects of benzene

Benzene is mildly irritating to the skin, eyes, and respiratory tract because it is a lipid solvent and degreases the mucosal layer, particularly after prolonged or repeated contact with the liquid.

Benzene may cause central nervous system depression and arrhythmias to persons acutely exposed.

Longer-term or chronic exposure to benzene may cause anaemia, alterations to the immune system, and leukemia. The underlying processes involves formation of reactive oxygen species (ROS) as result of oxidation-reduction of the benzene metabolites such as hydroquinone and catechol (Figure 19).

Figure 19. Toxicodynamic of Benzene Exposure



Source: Bahadar et al. (2014)5

Appendix 2: Assessment of the Fitness to Wear Respirator

Respirators are used to reduce the workplace exposure to CHTH by preventing it from entering the worker's respiratory system. The hierarchy of control measures always placed personal protective equipment as the last option. This is because the failure of the protection will result in exposure to the workers.

Regulation 8, USECHH 2000 allows the use of a respirator to reduce the exposure to the workers by having an appropriate assign protection factor (and a respiratory protection programme).

Exposure to the worker while wearing respirator is possible with inappropriate selection, inadequate wear, and problem of wear due to work conditions and individual health conditions. The respirator itself increases the breathing effort so that some workers may not be able to wear it properly or wear it over a certain duration or even worsen a medical condition.

Thus, it is important for the OHD to assess the fitness to wear respirator in a medical surveillance program.

A. Respiratory Effects of Wearing Respirators

Wearing of respirators can:

a) Increase airways resistance.

b) Increase dead space volume.

c) Induce cough.

d) Aggravate cardiovascular conditions.

e) Cause discomfort.

f) Cause ergonomic concerns due to extra weight.

g) Contribute to psychosocial effects.

h) Cause skin problems.

i) Cause altered senses.

a) Increase airways resistance.

- The wearing of a negative pressure respirator increases the resistance to inspiration because of the increased resistance to air flow caused by the filter media and face piece flow channels.
- As the inspiratory resistance increases, the inspiratory muscles (diaphragm and intercostal muscles) fatigue faster.
- The respiratory protective equipment may add to the work of breathing and (in the case of heavy equipment) skeletal muscle work, leading to earlier dyspnoea and fatigue for a given submaximal exercise task, and to reduced maximal work capacity.
- This may hasten or make worse respiratory muscle fatigue when the person wearing a respirator is affected by severe chronic airways obstruction, severe emphysema,asthma (in some cases), and moderate to severe interstitial lung diseases, as well as by clinically significant heart disease.
- For those with a history of pneumothorax, there is at least a theoretical hazard associated with increased swings in pleural pressure which should be considered when performing respirator certification.
- Wearers of particulate air-purifying respirators may detect an increase in breathing resistance as the filter becomes loaded.
- The physician should remind the worker to request a filter or chemical cartridge change whenever he or she perceives any increase in breathing resistance, or notes irritation, odour, or taste of contaminants.

b) Increase dead space volume.

- Wearing a respirator mask adds to the dead space volume.
- This dead air space added to the anatomic dead air space, requires the wearerto increase the depth and frequency of breathing to obtain the same amount of fresh air.
- The response to increased dead space is increased respiratory rate and tidal volume, and consequent increased work of breathing.
- Thus, while using a respirator, such individuals should not suffer from an added strain.
- To date, there is no scientific basis for using a certain level of lung function as a “cut off” to predict which individuals can and which cannot successfully use a respirator in the workplace.

- It should be noted, however, that these are studies of small groups of individuals, and, as always in medicine, personal consideration should be given to any patient when examining his or her particular case.

c) Cough

- A respirator might add to the burden of a person who suffers from a chronic or acute cough condition.
- Individuals with a productive cough would need to remove the mask to get rid of the sputum. Also, coughing action might create enough pressure to break the seal of the mask on the face.
- This may be a contraindication to wearing a respirator at work.

d) Cardiovascular effects

- Wearing SCBA respirators can increase cardiac workload (due to the weight carried), whereas a negative pressure respirator, if well maintained, would not significantly increase it.
- Heavy respirators such as SCBA, may increase the heart rate by about 20% at submaximal physical activity, and reduce the maximum exertion level by the same amount.
- Concerns that positive pressure respirators might cause some decrease in the cardiac output have been shown not to be of clinical relevance.

e) Discomfort

- Significant thermal discomfort should be expected when wearing respirators, even half-face tight-sealed masks or paper/fabric respirators such as the ones used for TB, as the tight fit of the mask over the face causes a build-up of moist warm air inside the face piece.
- This effect is exaggerated when protective clothing is being used.
- Powered air-purifying respirators or respirators with exhalation valves are often considerably more comfortable.

f) Extra weight and Ergonomic concerns

- Self-contained breathing apparatuses may add up to 16 kg of weight to the worker.
- Neck and back muscles carry this additional weight, and fatigue can result.
- Some powered air-purifying respirators also add the weight of the battery powered pump to the belt.
- Because of the added weight load, particular attention should be given to conditions such as herniated disks, and other chronic musculoskeletal ailments.

g) Psychological and Social effects

- Psychological effects while wearing respirators vary from mild discomfort to real inability to tolerate the mask and anxiety [Morgan, 1983; ATS, 1996].
- Difficulty in tolerating the mask may even give rise to the subjective feeling of breathing difficulty.
- Disqualification for psychological reasons constitutes approximately 10% of the medical disqualification.

h) Skin problems

- Workers may develop local skin diseases and allergic reactions when wearing respirators.
- Skin occlusion may exacerbate pre-existing conditions.
- Facial anatomical abnormalities or the presence of a beard or a mustache can affect the respiratory face seal if they extend to sealing surfaces.

i) Altered senses

- The use of a respirator decreases visual fields because of the respirator edges.
- It also reduces hearing, voice clarity/loudness, communication, and sense of smell.
- Hard contact lenses are not recommended for use with respirators. A dislodged contact lens secondary to rubbing an irritated eye or caused by air pressure from a positive pressure or air supplied respirator could decrease vision and put the individual at risk.
- Perforation of the tympanic membrane is not a contraindication to respirator use. There is no inspiratory airflow down the eustachian tube, so this does not represent an alternate route of inhalation.
- The ability to hear and to respond to emergency alarms or warning devices may be impaired when wearing an airline respirator with a hood or helmet that covers the head.

Consideration is also required for the work conditions:

- Job characteristics
  - o Heavy workloads requiring oxygen consumption of more than 1.3 L/min
  - o Long work duration and irregular rest periods
- Work environment
  - o Heat stress
  - o High contaminant air concentration
  - o Environment hazardous to life
  - o Noise, confined spaces
- Psychological stresses
  - o Time pressure
  - o Contract work (i.e., piece work)
  - o Night or rotating shift work
- Equipment-related stresses
  - o Characteristics and type of personal protective equipment
  - o Amounts of time the respirator/other personal protective equipment must be worn.
  - o Impermeable protective work clothing

B. Medical Examination for Fitness to Wear Respirator

a) Medical History

A medical history such as recommended in the Guidelines on the Use of PPE Against Chemical Hazards (DOSH Malaysia 2005) can be utilized to identify the following:

1) Previously diagnosed disease, particularly stressing known cardiovascular or respiratory diseases.

2) Psychological problems or symptoms including claustrophobia.

3) Problems associated with breathing during normal work activities.

4) Visual or auditory impairments, including colour vision assessment.

5) Past problems with respirator use or worker concerns about the proposed use of respiratory protective devices.

6) Current usage of medication, especially current use of medications whose side effects might impact the cardiopulmonary or CNS system, or their ability to make appropriate decisions related to their own safety or the safety of others, including current use of alcohol.

7) Any known physical deformities or abnormalities, including disc herniation and other musculoskeletal and radicular symptoms that may interfere with respirator use.

8) Heat intolerance.

9) Previous occupations and use of respirators.

b) Physical Examination

1) Musculoskeletal condition and anatomical problems (especially for SCBAs).

2) Facial deformities and facial hair.

3) Use of prescription eyeglasses or contact lenses.

4) Hearing ability (should be sufficient to ensure communication and response to instructions and alarm systems).

5) Significant restrictive or obstructive respiratory diseases or significant diffusion disorders of the lung.

6) Cardiovascular diseases: evidence of symptomatic coronary artery disease, significant untreated arrhythmias, or history of recent myocardial infarction, uncontrolled hypertension, or related symptoms.

7) Endocrine disorders: conditions which may result in sudden loss of consciousness or response capability (i.e., poorly controlled insulin-dependent diabetes).

8) Neurological disability: inability to perform coordinated movements and conditions affecting response and consciousness; history of uncontrolled epilepsy.

9) Psychological condition: claustrophobia, severe anxiety.

10) Other conditions specific to the work situation such as skin conditions where occlusive materials may result in symptoms or aggravation of a pre-existing dermatitis.

c) Pulmonary function tests

- These should be performed only to assist decision making for patients with lung disease.
- There are special situations, however, during which performance of routine respiratory function tests is required, i.e., firefighters' and asbestos workers' certification for respirator use.
- Spirometry for measuring FEV1 and FVC is the minimal recommended, in order to establish the presence and degree of restrictive or obstructive impairment.

d) Chest X-ray

- When an abnormality is discovered during the evaluation, an x-ray may be helpful to further study the worker.
- This is not routinely done for fitness assessment to wear respirator.

e) Disqualification from Wearing Respirator

The following may disqualify an employee from wearing a respirator:

1) Facial deformities and facial hair, where the respirator forms a seal to the face.

2) Respiratory diseases affecting pulmonary function.

3) Symptomatic coronary artery disease, significant arrhythmias, or history of recent myocardial infarction.

4) Endocrine disorders which may cause the employee to suffer sudden loss of consciousness or response capability.

5) Inability to perform coordinated movements and conditions affecting response and consciousness due to neurological disabilities.

6) Use of medications that affect judgment, performance or reliability or alter the state of awareness or consciousness.

7) A history of claustrophobia may require further evaluation.

8) Any other condition which the physician believes might require special restriction.

f) Evidence that the respirator or the respirator wear is or was compromised.

- Smell of the chemical during use.
- Experiencing acute health effects of the chemical exposure.
- Note: Some chemicals have poor warning properties, so that low level leakages cannot be determined by the wearer.

Appendix 3: Approach to MS of the Respiratory System

Identifiable diseases caused by exposures to CHTH are:

- Acute respiratory conditions
- Occupational asthma
- Chronic restrictive disease
- Chronic obstructive disease
- Work aggravated conditions
- Mixed conditions
- Cancer

The pathologic responses of the lung to toxic agents can be divided into the general categories of:

- irritation
- inflammation
- necrosis
- edema
- emphysema
- fibrosis
- allergic responses
- cancer

Factors that determine the severity of signs and symptoms include the following:

- Type and strength of chemical
- Sites of respiratory tracts where most of the chemicals were deposited.
- Exposure environment: indoor, outdoor, heat, cold

- Length of exposure: seconds, minutes, hours
- Form of chemical: gas, vapor, particulate, liquid
- Protective measures used to avoid exposure to chemicals.
- Prior medical condition
- Age of the person

Table 28. Potential Effects of Inhaled Irritants

Site of injury	Acute effects	Chronic effects
Eye, nose, sinuses, oropharynx	Irritations, inflammations	Corneal scarring, nasal polyps
Upper airway	Laryngeal edema, upper airway obstruction	Laryngeal polyps
Lower airways	Tracheobronchitis, bronchorrhea, decrease	Asthma, bronchiectasis
Lung parenchyma	Pneumonia, pulmonary edema, adult respiratory distress	Pulmonary fibrosis, bronchiolitis obliterans

Effects of Irritations

a) Irritations and inflammation are the most common pathological response to chemical induced injuries either acute or chronic, with effects ranging from minor irritations that are relatively reversible to extensive damage resulting in permanent disability or death.

b) It can occur in any of the three anatomical divisions of the lung.

c) Examples of irritant gases include chlorine, sulphur dioxide, ozone, nitrogen dioxide, and ammonia.

d) Symptoms include cough, throat and eye irritation, rhinorrhea, phlegm, wheeze, breathing discomfort or difficulty and chest pain.

e) These symptoms must be sought out during the history taking of the medical examination session.

Occupational Asthma

There are two major types:

a) Sensitizer induced asthma: characterized by variable time during the sensitization to a CHTH. Important examples are Isocyanates, where the sensitization may occur in a few days only.

b) Irritant induced asthma: It occurs after substantial exposures to any irritating dust, mist, vapor, and fume.

The diagnosis of occupational asthma is made by confirming the diagnosis of asthma and by establishing a relationship with the work environment. Both intermittent respiratory symptoms and physiologic evidence of reversible or variable obstructions are present.

The relationship between asthma and the workplace may fit any of the following patterns:

a) Symptoms only occur at work.

b) Symptoms improve on weekends or vacations.

c) Symptoms occur regularly after the work shift.

d) Symptoms progressively increase over the course of the work week.

e) Symptoms improve after change in the work environment.

Examples of specific agents of concern are:

a) Isocyanates

b) Organic dusts such as cotton and vegetable dusts

c) Metal salts

d) Acid anhydrides

e) Wood dust

The MS programme approach includes these four areas:

a) Detailed history, including occupational and environmental exposure and history of health effects during work.

b) Thorough physical examinations.

c) Appropriate imaging studies.

d) Pulmonary function testing.

The respiratory systems health effects generally produce symptoms when there is significant exposures.

The physical examinations may be helpful, but it is generally insensitive for detection of mild respiratory tract injuries. Relevant non-occupational diseases should not be missed.

For dust exposed worker, the Chest X-ray should be interpreted according to the International Labour Organization (ILO) Classification of Radiographs for Pneumoconiosis.

Important parameters of a pulmonary functions obtained from spirometry are:

a) Forced Expiratory Volume in 1 second (FEV1)

b) Forced Vital Capacity (FVC)

c) FEV1/FVC ratio

The OHD should familiarize themselves with the correct procedures of Spirometry testing and the correct interpretations. False negatives and false positives can be significant with incorrect maintenance procedures of the spirometry equipment and testing.

The use of respiratory questionnaire such as MRC UK respiratory questionnaire in the medical surveillance programme is a recommended tool, such as in the Australian Medical Surveillance Guidelines and in the UK Guidelines.

However, it requires specific learning and experience, thus, OHD may learn and practice it, and use it for MS, when the confidence level is adequate.

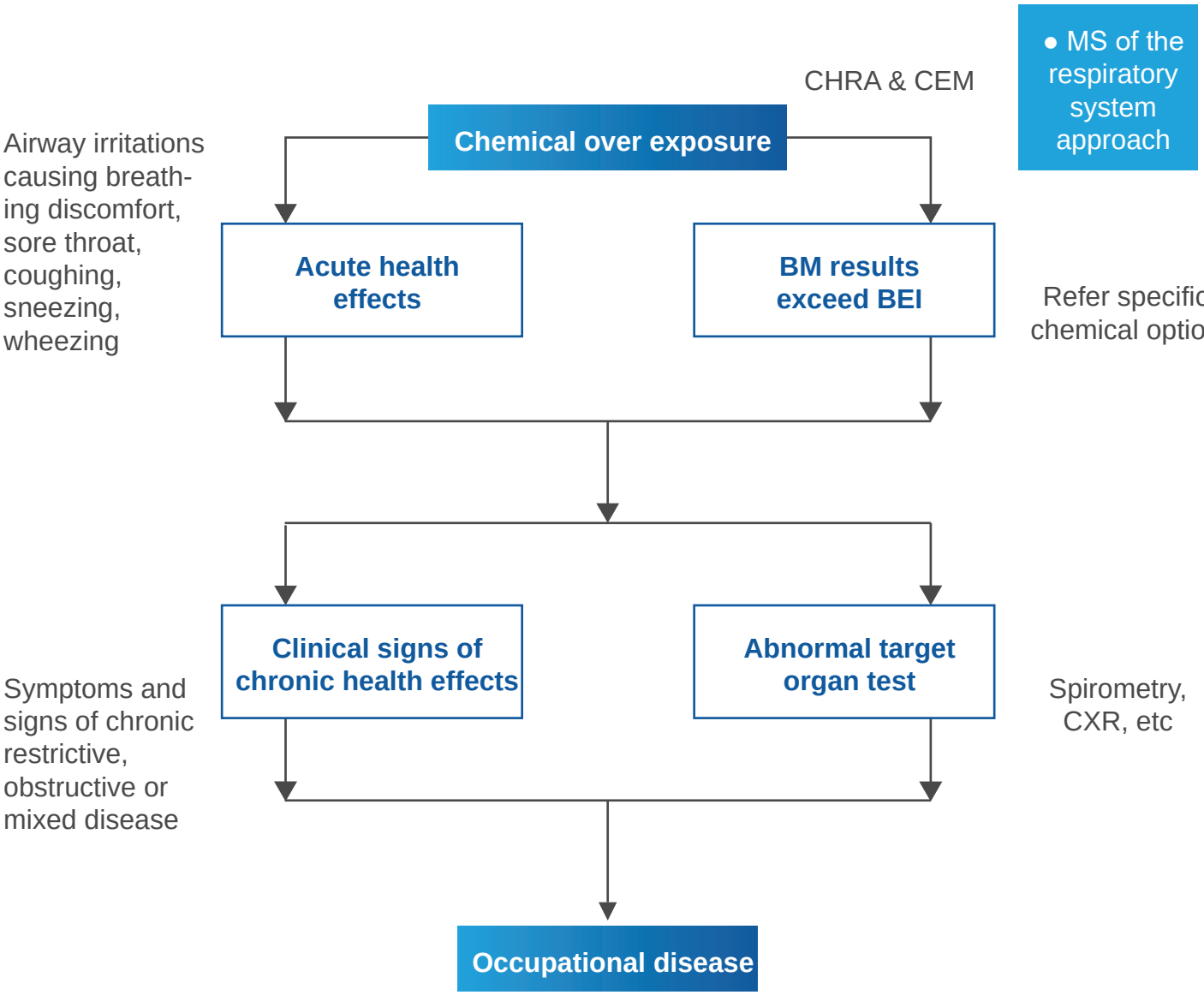


Figure 20. Approach to the Medical Surveillance of the Respiratory System

Appendix 4: Approach to Diagnosis of Occupational Liver Disease

Important Note

- Occupational Liver Disease (OLD) very rarely display pathognomonic signs, and because of the multifactorial causality of disease, the OHD should try to assess the relevance of occupational components in an all-encompassing approach.
- Taking an occupational history represents a key step in the clinical assessment of suspected OLD.
- Thus, in addition to the employee's symptoms, the work environment must be explored, bearing in mind that occupational exposure occurs most commonly by inhalation and through the skin. The list of information to be obtained from the employee is detailed in Figure 21.

1. Chronological summary of all work activities and their duration.

2. A detailed description of all the workplace, of the job and of a typical working day.

3. An inventory of all chemicals that are present and how are used.

4. Details of any measures to limit chemical exposure such as: workplace ventilation and the nature protective measures that are taken (requirement to wear special clothing and gloves, the use of masks, goggles, and other devices).

5. Enquiring if programmes of industrial hygiene, biological monitoring and medical surveillance are or have been in place and retrieve the results, if necessary, keeping in mind however that compliance with occupational exposure limits do not necessarily protect all workers from adverse effects

6. Enquire as to whether co-workers have similar symptoms and signs to those of a patient with suspected occupational liver disease. This may involve questioning and even examining co-workers. If several cases come to light, it may be possible to demonstrate an exposure-response relationship.

7. Enquire if compensation procedures have been undertaken and results are available.

8. Exposures to chemicals other than those present at workplaces, associated for instance with environmental air pollution, hobbies, recreational habits, and others should be ruled out

Figure 21. Critical information to be obtained from the worker with a suspicion of Occupational Liver Disease

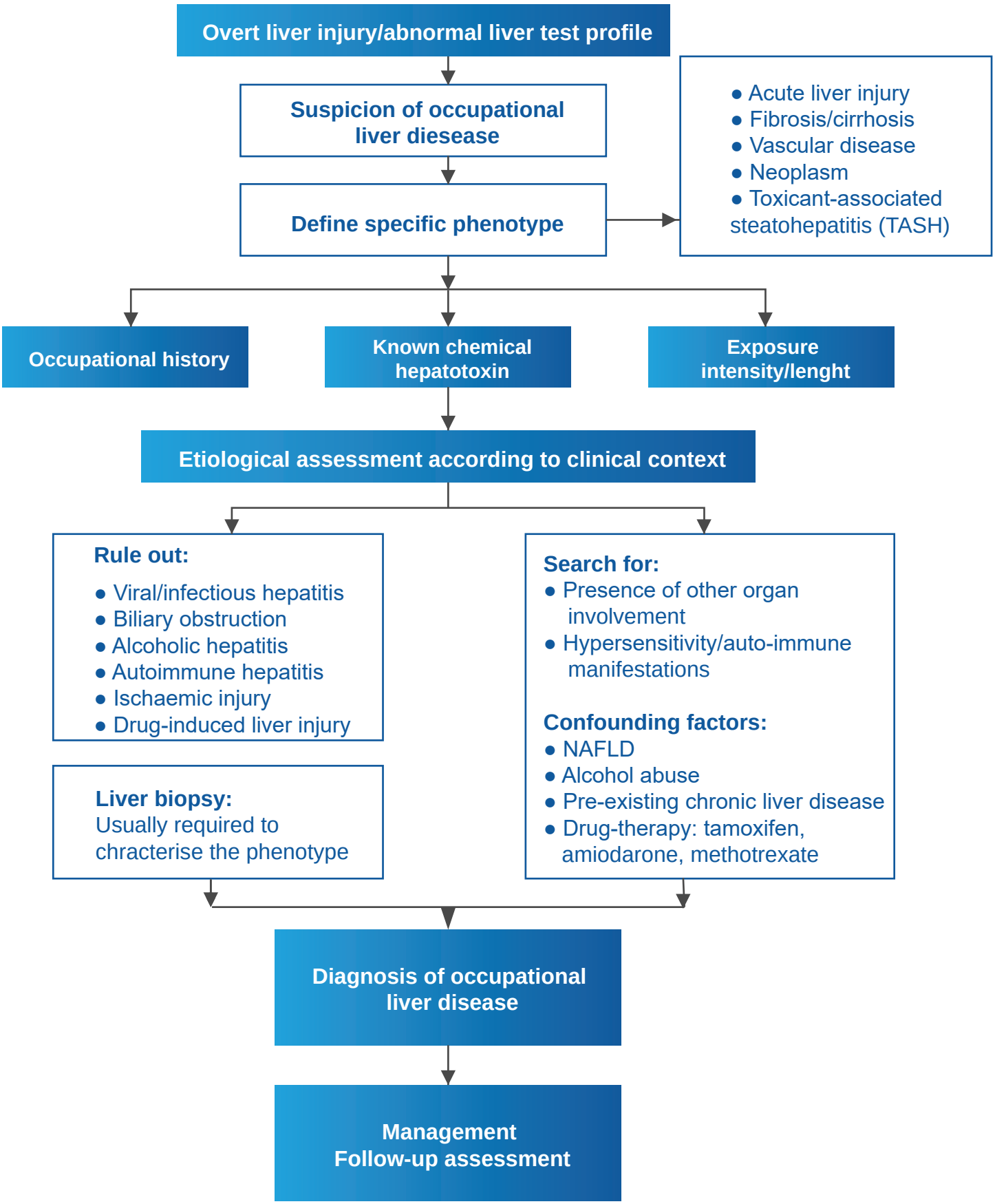


Figure 22. Schematic approach to the assessment and diagnosis of Occupational Liver Disease (OLD)

Table 29. Pathological Patterns and Morphological Features of Liver Disease Associated with Workplace-related Toxicants

Pathological patterns	Morphological features	Toxicants
Acute Damage		
Hepatocellular	Hepatocellular necrosis ± lobular inflammation	CCL4, chloroform, toluene, TNT, PCBs, chloronaphthalene, DMF, hydrazine, 2-nitropropance, phosphurs, DMA, halothane, TCE, tetrachloroethane, 1,4-dichlorobenzene, DMF
	Microvascular steatosis	DMF
Cholestatic/Mixed	Cholestasis, cholangitis Combined features syndrome	Methylenedianiline Nitrobenzene, paraquat, methylenedianiline
TAFLD	● Steatosis (macor/ microvesicular) ● Steato-hepatitis (steatosis + lobular Inflammation + hepatocellular ballooning)	Chloralkenes (PCE, TCE), VCM, chloroform, CCL4, volatile organic compounds (benzene, toluene, styrene, xylene), dioxins, chlordecone, DMF, hydrazine, arsenic, mercury, POPs, pesticides, and some nitro-organic compounds
Vascular	Sinusoidal obstruction syndrome	VCM, dioxin, pyrrolizidine alkaloids, arsenic, copper sulfate
	Peliosis	VCM
Chronic Damage		
Fibrosis	Periportal fibrosis Extensive fibrosis/cirrhosis	VCM, PCBs, chloronapthalene, Tetrachloroethane VCMalkaloids, arsenic, copper sulfate
Vascular	Porto-sinusoidal vascular disease (previously hepatoportal sclerosis)	VCM, sprays containing copper sulfate and lime

Chronic Damage		
Epithelial ● Hepatocellular carcinoma ● Cholangiocarcinoma		Arsenic, dimethylanitrosamine 1,2-Dichloropropance, dichloromethane
Vascular ● Angiosarcoma ● Epitheliod hemangioendothelio-ma		VCM, Arsenic VCM

Table 30. Summary of Current Clinical Serum Biomarkers of Liver Function and Injury

Serum biomarker	Tissue localization	Injury	Specific damage marker	Comments
ALT	Primarily localized to liver	Elevated due to liver necrosis and with heart and skeletal muscle injury (necrosis)	Hepatocellular necrosis	Commonly used to assess hepatocellular injury
AST	Localized in heart, brain, skeletal muscle, and liver tissue	Elevated due to liver or extrahepatic tissue injury	Hepatocellular necrosis	Less specific than ALT
Total bilirubin	Taken up, conjugated in liver, and secreted into bile	Marker of hepatobiliary injury and liver functions; also increased due to haemolysis	Cholestasis, biliary; Liver function	Conventional biliary; in conjunction with ALT, better indicator of disease severity in humans

ALP	Broad tissue localization	Marker of hepatobiliary injury	Cholestasis	Conventional biliary; associated with drug-induced cholestasis in humans
GGT	Activity localized to kidney > liver, pancreas	Marker of hepatobiliary injury	Cholestasis, biliary	Conventional biliary; high sensitivity in humans
Bile acids	Bile duct	Elevated with liver injury and functional change	Liver function	Levels influenced by diet and fasting
Clotting time		Increase with sever liver injury	Liver function	Liver fails to produce clotting factor increasing time; international normalized ratio equivalent to prothrombin time
Protein Levels		Decreased with severe liver injury	Liver function	Liver fails to synthesize enough protein especially albumin

Table 31. Clinical-diagnostic Types of Occupational Toxic Hepatitis

Type of disease	ALT	ALP	γ-gt	Bilirubin	Bile acids
Hepatocellular	> 2 ULN	N	> 2	Elevated levels	Elevated levels
Cholestatic	N	> 2 ULN	>4	Normal or moderate level	Elevated levels
Mixed	>2 ULN	≥ ULN	> 2	Normal of moderate level	Elevated levels
Thresholds of liver enzymes used to define acute liver injury and pattern of damage according to Ration (R) [4,5].					
a. Any one of the following **CRITERIA TO DEFINE LIVER INJURY***: 1) Alanine aminotransferase (ALT) level ≥5x upper limit of normality (ULN) 2) Alkaline phosphatase (ALP) level ≥2 x ULN (particularly if concomitantly elevated gamma-glutamyl transpeptidase (GGT) in the absence of bone disease 3) ALT level ≥3 x ULN and simultaneous total bilirubin (TB) level >2 x ULN					
b. PATTERN OF LIVER INJURY according to R value: R value is defined as: (ALT/ULN)/(ALP/ULN)*** Hepatocellular pattern: R ≥5 Cholestatic pattern: R ≤2 Mixed pattern: R value is >2 and <5					

*In workers with abnormal baseline liver blood tests, ULN is replaced by the mean baseline values obtained prior to the exposure to the suspect chemical, and the increases ALT, ALP and TB should be proportionate to this modified baseline.

***AST can replace ALT when this one is unavailable.

ALP, alkaline phosphatase; ALT, alanine aminotransferase; AST, aspartate aminotransferase; TB, total bilirubin; ULN, upper limit of normal.

Table 32. Category of Severity Description

Category of Severity	Description
Grade mild	Elevated alanine aminotransferase/alkaline phosphatase (ALT/ALP) concentration reaching criteria for liver injury* but bilirubin concentration <2 upper limit of normality (ULN)
Grade moderate	Elevated ALT/ALP concentration reaching criteria* for liver injury and bilirubin ≥2 x ULN or symptomatic hepatitis
Grade severe	Elevated ALT/ALP concentration reaching criteria for liver injur bilirubin concentration ≥2 x ULN, and one of the following: ● International normalized ratio ≥1.5 ● Ascites and/or encephalopathy and absence of underlying cirrhosis ● Other organ failure considered to be due to occupational
Grade fatal or liver transplantation	Death or transplantation due to liver injury

Category adapted from references 4 and 5
*Criteria for liver injury are defined in Table 3
The international normalized ratio (INR) or standardized prothrombin time

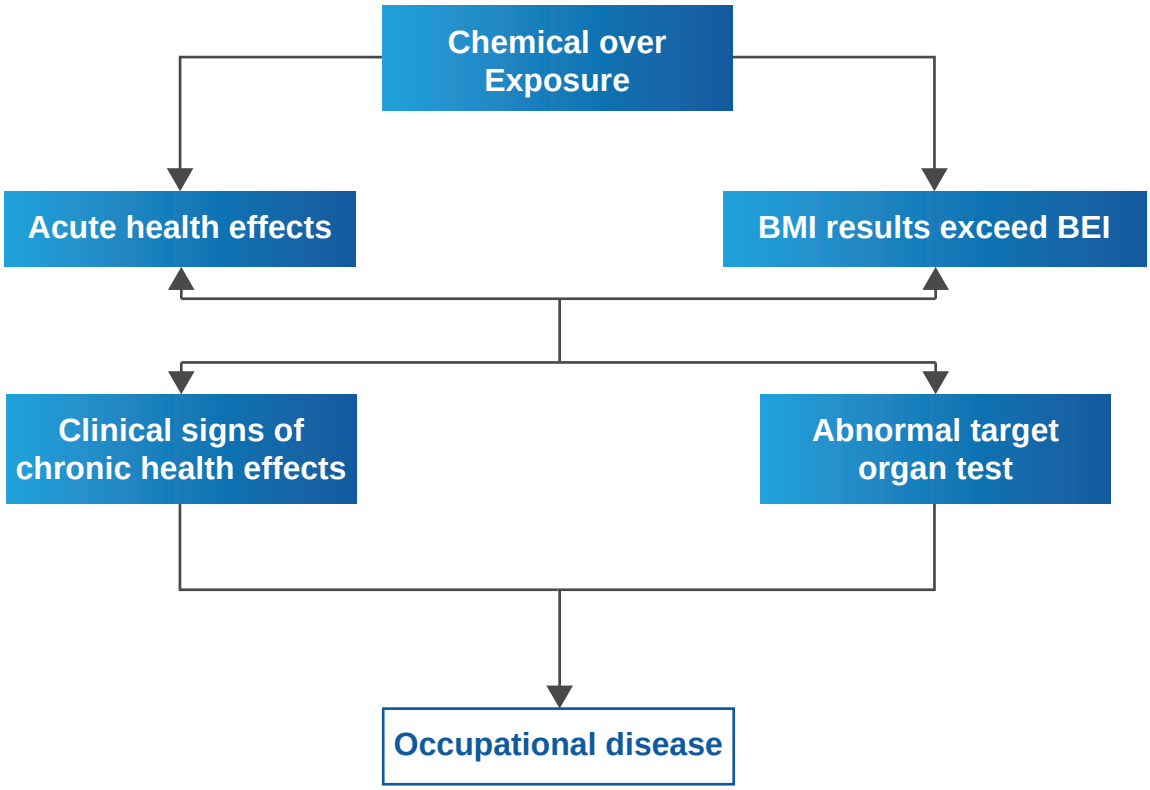


Figure 23. Development of Chemical Related Occupational Disease

NAFLD or Fatty Liver

- May progress to cirrhosis within 10 years.
- NAFLD (Non-Alcoholic Fatty Liver Disease) states as simple fat, which impacts and 20% to 30% of those individuals who progress to Non-Alcoholic Steatohepatitis (NASH). Another 20% to 30% of individuals progress to more advanced NASH fibrosis, and the final stage is NASH cirrhosis. Refer to Table 33 for typical features of NAFLD and Alcoholic Liver Disease.
- It used to be thought that progression from early stage NAFLD to cirrhosis took decades, but recent studies have shown that some people progress rapidly within 2 years. However, research has also shown that there is reversibility.

Table 33. Typical Features of Non-alcoholic Fatty Liver Disease (NAFLD) and Alcoholic Liver Disease

Characteristics	NAFLD	Alcoholic liver disease
Body weight	Increased	Variable
Fasting plasma glucose or HbA1c	Increased	Normal

Reported daily alcohol intake	<20 g for women, <30 g for men	>20 g for women, >30 g for men
ALT	Increased or normal	Increased or normal
ALT	Normal	Increased
AST:ALT ratio	<0.8 (>0.8 with more advanced disease)	>1.5
GGT	Increased or normal	Considerably increased
Triglyceride	Increased	Variable, may be considerably increased
HDL cholesterol	Low	Increased
Mean corpuscular volume	Normal	Increased

Appendix 5: Swedish Q16 Questionnaire

SWEDISH Q16 QUESTIONNAIRE FOR LONG TERM SOLVENT EXPOSED WORKER

Name:

Work Unit:

Company

This questionnaire is used to help determine whether long term over exposure to solvents has affected the central nervous system (brain):
Answer "yes" or "no" to each question. If a solvent-exposed worker answers "yes" to six (6) or more of these questions, referral for more in-depth evaluation may be indicated.

Soalan-soalan ini digunakan untuk menolong menentukan samada pendedahan berlebihan kepada 'solvents' telah pun memberi kesan kepada system saraf (otak):
Jawab "ya" atau 'tidak" untuk setiap soalan. Jika pekerja yang terdedah kepada 'solvents' menjawab "ya" untuk enam daripada soalan-soalan ini maka pemeriksaan yang terperinci adalah perlu.

1. Do you have a short memory?
   Adakah anda mudah lupa?

2. Have your relatives told you that you have a short memory?
   Adakah saudara-mara anda memberitahu anda yang anda mudah lupa?

3. Do you often have to make notes about what you must remember?
   Adakah anda perlukan peringatan supaya anda tidak lupa untuk melakukan sesuatu?

4. Do you often have to go back and check things you have done (turn off the stove, locked the door, etc)?
   Adakah anda perlu memeriksa semula perkara yang sepatutnya anda telah lakukan kerana anda was-was samada perkara tersebut telah pun anda lakukan?

5. Do you generally find it hard to get the meaning from reading newspapers and books?
   Adakah anda merasakan sukar untuk memahami maksud apa yang anda baca di surat khabar ataupun buku?

6. Do you have problems with concentrating?
   Adakah anda mengalami masalah untuk menumpukan perhatian?

7. Do you often feel irritated without any particular reason?
   Adakah anda mudah tersinggung tanpa sebab yang munasabah?

8. Do you often feel depressed without any particular reason?
   Adakah anda mudah rasa kemurungan tanpa sebab yang munasabah?

9. Are you abnormally tired?
Adakah anda rasa keletihan yang amat sangat?

10. Are you less interested in sex than what you think is normal?
Adakah anda kurang berminat terhadap sex berbanding dengan tahap yang anda rasa normal?

11. Do you have heart palpitations even when you don't exert yourself?
Adakah anda merasa berdebar-debar sekalipun anda tidak melakukan sebarang kerja?

12. Do you sometimes have a feeling of pressure in your chest?
Adakah anda merasa suatu tekanan pada dada anda?

13. Do you perspire without any particular reason?
Adakah anda berpeluh tanpa sebab-sebab tertentu?

14. Do you have a headache at least once a week?
Adakah anda kerap sakit kepala sekurang-kurangnya seminggu sekali?

15. Do you often have a painful tingling in some parts of your body?
Adakah anda merasa sakit 'semut-semut' di beberapa bahagian anggota badan anda?

16. Do you have any problems with buttoning and unbuttoning?
Adakah anda menghadapi masalah untuk mengancing butang atau membuka butang pakaian?

Appendix 6: The Mathias Criteria

Table 34. The Mathias Criteria for Probable Occupational Causation of Contact Dermatitis

No	Criteria
1.	*Is the clinical appearance consistent with contact dermatitis?* Yes: Identification of clinical features of eczema (pruritus, erythema, vesicles, exudation, crusting, signs of lichenification). No: Clinical appearance is not eczematous. Don't know: Seborrheic dermatitis, dyshidrotic eczema, nummular eczema, atopic eczema, and neurodermatitis all have clinical patterns that resemble an eczematous reaction.
2.	Are there workplace exposures to potential cutaneous irritants or allergens? Yes: The physician should inquire about all sources of workplace exposure, including personal protective equipment, creams, and soaps. It is important to be familiar with the toxicological data on these products. No: Toxicological data and/or clinical experience indicate that there is no irritant or allergic exposure at the workplace. Don't know: If the physician is unable to determine whether there is workplace exposure to irritants or allergens, this criterion should not be assessed.
3.	Is the anatomic distribution of dermatitis consistent with cutaneous exposure in relation to the job task? Yes: Contact dermatitis is usually more severe on surfaces that are exposed at work. No: The dermatitis spares skin surfaces with the greatest exposure but affects others. Don't know: There are exceptions to the above statement, for example, more permeable areas such as the eyelids, the face, and the genitals.
4.	Is the temporal relationship between exposure and onset consistent with contact dermatitis? Yes: The exposure preceded the onset of the symptoms. In the case of allergic contact dermatitis, the expected latent period can be as long as 6 months. No: Most of the symptoms occurred before exposure at the workplace. Don't know: If the latent period is more than 6 months, it will be difficult to establish a causal relationship. Workers aged between 50 and 60 years may have greater skin sensitivity due to age.

5	Are non-occupational exposures excluded as probable causes? Yes: Other irritants such as cosmetics and glues must be excluded by a thorough nonoccupational history and on occasions patch testing. No: Nonoccupational exposures may be the cause of the dermatitis. Don't know: Without a thorough exposure history, the physician cannot confidently exclude a nonoccupational cause
6.	Does dermatitis improve away from work exposure to the suspected irritant or allergen? Yes: There is improvement during leave, weekends, holidays, etc. No: The dermatitis does not improve after the removal of the workers from the workplace. Improvement may not be seen for up to 3 or 4 weeks in the case of chronic dermatitis. Don't know: Improvement off work or with workplace modifications are sometimes due to medical treatment.
7.	Is the temporal relationship between exposure and onset consistent with contact dermatitis? Yes: The exposure preceded the onset of the symptoms. In the case of allergic contact dermatitis, the expected latent period can be as long as 6 months. No: Most of the symptoms occurred before exposure at the workplace. Don't know: If the latent period is more than 6 months, it will be difficult to establish a causal relationship. Workers aged between 50 and 60 years may have greater skin sensitivity due to age.

Source: Assessment of the Mathias Criteria for Establishing Occupational Causation of Contact Dermatitis (Gomez de Carvallo et al. 2011)

Appendix 7: MS Recommended Forms

USECHH 1

USECHH 1

MEDICAL SURVEILLANCE PROGRAMME

EXAMINATION FORM

Occupational Safety and Health Act 1994 (Act 514)

Use and Standard of exposure of chemicals hazardous to health regulation 2000

Department of Department of Occupational safety and health

Ministry of Human Resources

Workplace:

Chemical(s):

Types of Medical Examination:

☐ Pre-placement

☐ Periodic

☐ Return To Work

☐ Exit

This document is confidential and must be kept by the employee. It must be produced to the attending OHD upon request. Please write clearly.

PART 1 – To be filled by employee.

A. GENERAL INFORMATION

Name of worker

_____

Address

_____

_____

District _____ State_____ Post-code ☐☐☐☐☐

Home Tel No. ☐☐☐ - ☐☐☐☐☐☐☐☐

IC No. ☐☐☐☐☐☐ ☐☐ ☐☐☐☐ Age ☐☐ years

Passport No. ☐☐☐☐☐

Gender ☐ Male ☐ Female | Status ☐ Single ☐ Married No.of child ☐ No. of years married ☐ years

Ethnic
☐ Malay
☐ Chinese
☐ Indian
☐ Others (specify) _____

Malaysian Citizen ☐ Yes ☐ No (Please specify country) _____

B. MEDICAL HISTORY

1. Have you been diagnosed with any disease? If yes, please provide details.

_____

_____

_____

_____

2. Are you on any medication or medical follow up? If yes, please provide details.

_____

_____

_____

_____

3. Have you ever been hospitalized? If yes, please provide details.

_____

_____

C. PERSONAL & SOCIAL HISTORY

Smoking ☐ Current smoker
☐ Ex-smoker
☐ Non-smoker

No. of year smoked
_____
No, of cigarettes nowdays
_____
No, of years vaping
_____

Vaping ☐ Yes
☐ No

Alcohol ☐ Yes if yes, how often? _____
☐ No

Hobby _____

Part-time job _____

D. RELEVANT FAMILY HISTORY

_____

_____

_____

E. OTHER HISTORY (IF RELEVANT)

_____

_____

_____

_____

_____

_____

_____

_____

_____

F. OCCUPATIONAL HISTORY

	Job tittle	Name of company	Duration of employment	Duration of Exposure to CHTH	Have you ever experienced incident of chemical exposure (eg. spill/splash)? If yes, please provide details
Present					
Past					

G. HISTORY OF TRAINING

	Y	N	Comment
1. Are you trained on: a. Safe handling of chemicals? b. Recognizing the signs and symptoms of diseases? c. Poisoning due to chemicals used in the workplace? d. Proper PPE usage?			
2. Do you use any PPE when handling the chemicals			

This is to certify that the above statement is true. I, hereby give consent to the OHD to perform medical examination, necessary tests, and communicate with the employer the results of my medical examination and work capability.

Signed by: **Witnessed by Doctor**

(**)** **(** **)**

Date: **Date:**

PART 2 – To be filled by OHD

Health effects experienced during chemical handling:

H. HISTORY OF HEALTH EFFECTS DUE TO CHEMICAL EXPOSURE

Respiratory

	Breathing discomfort or difficulty
	Cough
	Sore throat
	Sneezing

Central nervous system

	Drowsiness
	Dizziness
	Headache
	Confusion/Lethargy
	Nausea
	Vomiting

Skin and Eyes

	Eye irritations
	Blurred vision
	Blisters
	Burns
	Itching
	Rash
	Redness

Others (Elaborate frequency, severity etc):

_____

_____

_____

I. CLINICAL FINDINGS

I1. CURRENT HEALTH EFFECT DUE TO CHTH EXPOSURE

Describe the health effects currently experienced by the employee (refer to the Health Effects Monitoring of the relevant chemical in the Specific Guidelines or other relevant references).

_____

_____

_____

_____

_____

_____

I2. PHYSICAL EXAMINATION (Refer to the Health Effects Monitoring of the relevant chemical in the Specific Guidelines or other relevant references to list any abnormalities detected.)

a) Anthropometry	Weight (kg)	
	Height (cm)	
	BMI	
b) Vital sign	Blood pressure(mm/Hg)	
	Pulse rate (bpm)	
	Respiratory rate	
c) General appearance		
d) Organ/System	**Clinical Findings**	
i) Cardiovascular system		
ii) Ear, Nose and Throat		
iii) Eyes		
iv) Gastrointestinal		
v) Haematology		
vi) Kidney		
vii) Liver		
viii) Musculo-skeletal		
ix) Nervous System: Central and Peripheral Nervous		
x) Respiratory		
xi) Reproductive		
xii) Skin		
xiii) Others		

J. TARGET ORGAN FUNCTION TEST

Only relevant laboratory/other test(s) (e.g., Spirometry, chest X-ray, etc.) results pertaining to MS.

Please refer to the Specific MS Guidelines.

Test	Results/Findings	Comments

K. BIOLOGICAL MONITORING

Please refer to the specific guidelines and list down the relevant biomarker(s).

Biological Exposure Indices Determinants (BM/BEM)	Results

L. ASSESSMENT ON THE FITNESS TO WEAR RESPIRATOR WHERE APPLICABLE (refer Appendix 2 of the General MS Guideline)

Conclusion on fitness to wear respirator.

	Fit
	Not fit

Please Justify, _____

M. CONCLUSION OF MS FINDINGS
(the information will also be input in the forms USECHH 2, 4 and 5)

	MS Findings		if yes, is it work related?	
	Yes	No	Yes	No
History of health effects due to chemiical(s) exposure *(H)			Not applicable	Not applicable
Clinical findings *(I)				
Target organ function test result (please specify) *(J) _____ _____				
BEI determinant (BM/BEM) *(K) (please specify determinant): _____ _____				
Pregnancy/Breast feeding			Not applicable	Not applicable

*Information can be obtained from the relevant section in USECHH 1

Conclusion of fitness to work. ☐ Fit ☐ Not fit

N. RECOMMENDATION

MRP (please circle where applicable)
Temporary / Permanent: Yes / No

Date of MRP: _____ until _____

Next review date:

		-			-				

Recommendation to employer:

_____

_____

The implication of the above results has been explained to me by the OHD.

Signature of the employee:

Date:

Name of Occupational Health Doctor: _____
Name of clinic: _____

MMC No.

DOSH Registration No.

Clinic Tel. No.

Fax No.

Email _____

Date

_____

Occupational Safety and Health Act 1994 (Act 514)

Use and Standard of Exposure of Chemicals Hazardous to Health Regulations 2000

SUMMARY RECORDS OF EMPLOYEE

Name of worker: _____

Name of chemical: _____

No.	MS date	Type of assessment	History of health effects due to CHTH exposure	Clinical findings	Target organ function test (specify organ)	BEI determinant	Work relatedness	Conclusion of MS Findings (Fit/Not fit)	Date of MRP	Name of OHD/DOSH Reg. No.

Occupational Safety and Health Act 1994
(Act 514)

Use and Standard of Exposure of Chemicals Hazardous to Health Regulations 2000

CERTIFICATE OF FITNESS

Name of Person examined _____
NRIC/ Passport No. _____Date of Birth _____Sex_____
Name & Address of Employee _____
_____

Examination/Tests done and the results:

I hereby certify that I have examined the above-named person on_
_____
and that he is fit/not fit for work which may expose him t

Remarks (if any):

_____
Signature & Date

_____
Name of Occupational Health Doctor
(in BLOCK letters)

_____
DOSH Reg. No.

_____
Address of Practice

Occupational Safety & Health Act 1994 (Act 514)
Use and Standard of Exposure of Chemicals Hazardous to Health Regulations 2000

SUMMARY REPORT FOR MEDICAL SURVEILLANCE

Name of Workplace:_____
MyKKP Registration No.:_____
Address of Workplace: _____

Total number of workers in the workplace	
Name of the work unit where workers are in	
Total number of exposed workers in the work unit	
Total number of workers examined	

Individual Chemical: **(Use ONE USECHH 4 form for ONE chemical only)** _____
Date of CHRA conducted: -_____
CHRA report no.: _____
Indication for medical surveillance based on CHRA report:

☐ **Significant personal exposure (≥ 50% PEL)**
☐ **Others** (Please provide details) _____

☐ **Reported health effects**
☐ **Skin absorption**

MEDICAL SURVEILLANCE RESULTS

	No. of workers with normal findings	No. of workers with abnormal findings		No. of workers recommended for medical removal protection
		Occupational	Non-occupational	
History of health effects due to chemical exposure(*H)			**Not applicable**	**Not applicable**
Clinical findings (*I)				
Target organ function test(s) (*J). Please specify: _____				
BEI determinant (*K) (BM/BEM). Please specify determinant: _____				

Continue in separate sheet if required. Please include details of abnormal examination/test results in USECHH 5ii form and Medical Removal Protection in USECHH 5i form.

Total no. of employees recommended for MRP

_____

Name of Laboratory:

_____

Recommendation:

_____

_____

_____

_____

_____

*	Decision	Justification of Decision	Date of implementation
	Continue MS		
	Stop MS		

* Please √ where applicable

I hereby declare that all particulars given in this report are accurate to the best of my knowledge.

Name of occupational Health Doctor:_____

OHD Registration No:_____

Name of Practice & Address:_____

Tel No:_____ HP no:_____ Fax No:_____

Email address:_____

Date: Signature:

Submit this form within 30 days of completion of medical surveillance to the Director General, Department of Occupational Safety and Health, Putrajaya. Download this form at .dosh.gov.my Please ensure all items in the form are completed. Incomplete forms will not be accepted.

Occupational Safety and Health act 1994 (Act514)
Use and Standard of Exposure of Chemicals Hazardous to Health Regulation

MEDICAL REMOVAL PROTECTION

Temporay ☐ **Permanent** ☐

1. Name of Worker:_____
2. NRIC/Passport No:_____
3. Date of Birth:_____
4. Sex: _____
5. Name and Address of Workplace:_____
6. Data of starting employment: _____Duration of Employment (Years): _____
7.Health Hazard Present (Use one form for one chemical):_____

I certify that the above named person examined by me on (dd/mm/yy)_____ should not continue to work as (designated)_____ in (place of work) _____ department/selection for _____ months, subject to a review on (dd/mm/yy)_____

In the meantime, he should be given alternative work in another department/section which does not expose him to (name of individual chemical) _____

The reasons for my recommendations are as follows (Please √):

☐ Pregnancy
☐ Breastfeeding
☐ Abnormal BM/BEM result
☐ Adverse health effects based on clinical findings
☐ Target organ function test abnormality
☐ Specify others:_____

Name of OHD : _____
Address of Practice : _____
_____
Email Address: _____
H/P: _____ Tel:_____ Fax:_____

_____ _____
OHD signature Date

Note: This certificate should be completed in triplicate and the original copy forwarded to the director General. department of Occupational Safety and Health. Putrajaya and must include the actual results of the relevant examination/tests. The quantitive results (e.g. blood lead) the exact Diagrams and measurements units must be clearly stated. Also include a copy of qualitative results (e.g Chest X-ray). Incomplete form will not be accepted.

DETAILS OF WORKERS WITH ABNORMAL EXAMINATION RESULTS

Name of worker: _____

Name of chemical: _____

No.	Employee's name	NRIC/ Passport	Sex		Job category/ Designation	Type of assess-ment	History of health health effect due to CHTH exposure	Clinical findings	Target organ function test (specify organ)	BM determinant	Work relatedness	Recommen-dation/action taken (MRP, Retraining, Refit of PPE)	Conclusion of MS Findings (Fit/Not fit)
			M	F									

Submit this form together with USECHH 4 form within 30 days of completion of the medical surveillance to The Director General, Department of Occupational Safety and Health, Putrajaya. This form can be downloaded from http://www.dosh.gov.my Continue in separate sheet if required.

Appendix 8: Example of MS Recommended Forms -

Occupational Safety & Health Act 1994 (Act 514)
Use and Standards of Exposure of Chemicals Hazardous to Health Regulations 2000

SUMMARY REPORT FOR MEDICAL SURVEILLANCE

Name of Workplace: ABC Sdn Bhd
MyKKP Registration No.:123456
Address of Workplace: 1234, Jalan Kota, 05250 Alor Setar, Kedah

Total number of workers in the workplace	58
Name of the work unit where workers are in	Gluing
Total number of exposed workers in the work unit	15
Total number of workers examined	10

Individual Chemical: n-hexane
(***Use ONE USECHH 4 form for ONE chemical only)***
Date of CHRA conducted (Put not done if CHRA is not done):01/04/2021
CHRA report no.: RN458/21
Indication for medical surveillance based on CHRA report:

- [√] Significant personal exposure (≥ 50% PEL)
- [ ] Skin absorption
- [ ] Reported health effects
- [ ] Others

(please provide details)

MEDICAL SURVEILLANCE RESULTS				
	No. of workers with normal findings	No. of workers with abnormal findings		No. of workers recommended for medical removal protection
		Occupational	Non-occupational	
History of health effects due to chemical exposure (*H)	8	2		
Clinical findings (*I)	10	0	0	0
Target organ function test(s) (*J). Please specify: i. LFT ii. RFT	 8 10	 1 0	 1 0	 1 0
BEI determinant (*K) (BM/BEM). Please specify determinant: Urine 2,5 hexanedione	8	2	0	2
Continue in separate sheet if required. Please include details of abnormal examination/ test results in USECHH 5ii form and Medical Removal Protection in USECHH 5i form.				

Total no. of employees recommended for MRP = 2 (1 with abnormal HEM, 2 with abnormal BM)

Name of Laboratory: Asia Lab Sdn Bhd

Recommendation:

1) To perform MRP on 2 employees from 14/07/2022 – 14/09/2022
2) To repeat urine 2,5 hexanedione on Ali bin Ahmad and Abu bin Bakar on 14/08/2022
3) To repeat LFT on Asri bin Yassin and Abu bin Bakar on 14/08/2022
4) For all the 3 employees to have a review with OHD on 01/09/2022 after receipt of results
5) To inform CHRA Assessor on the abnormal findings for improvement of workplace hygiene.

*	Decision	Justification of Decision	Date of Implementation
√	Continue MS	MRP cases imply poor control and workplace hygiene.	14/07/2022
	Stop MS		

* Please √ where applicable

I hereby declare that all particulars given in this report are accurate to the best of my knowledge.

Name of Occupational Health Doctor: Azli bin Samad
OHD Registration No: JKKP 1111
Name of Practice & Address: Klinik Mutiara
Tel No: 04123456 HP no: 01988888 Fax No: 04589254
Email address: mutiara@yahoo.com

Date: 14/07/2022 **Signature: €**

Submit this form within 30 days of completion of medical surveillance to the Director General, Department of Occupational Safety and Health, Putrajaya. Download this form at .dosh.gov.my Please ensure all items in the form are completed. Incomplete forms will not be accepted.

Example USECHH 5ii

DETAILS OF WORKERS WITH ABNORMAL EXAMINATION RESULTS

Work unit: Gluing

Chemical: n-hexane

No.	Employee's Name	NRIC/ Passport	Sex		Job category / Designation	Type of assessment	History of health effects due to chemical exposure	Clinical findings	Target organ function test (specify organ)	BM determinant Urine 2,5 hexanedione	Work relatedness	Recommendation/action taken (MRP, Retraining, Refit of PPE)	Conclusion of MS Findings (Fit/Not fit)
			M	F									
1	Ali bin Ahmad	12345678	√		Fitter	Periodic MS	Nil	Nil	Liver - normal Kidney - normal	Exceed BEL	Yes	MRP	Unfit until 14/09/2022
2	Abu bin Bakar	23456789	√		Helper	Periodic MS	Nil	Nil	Liver – abnormal enzymes Kidney – normal	Exceed BEL	Yes	MRP	Unfit until 14/09/2022
3	Asri bin Yassin	34567890	√		Fitter	Periodic MS	Nil	Nil	Liver – abnormal enzymes Kidney – normal	Below BEL	No	Nil	Fit (to review LFT, KIV refer gastro)

Submit this form together with USECHH 4 form within 30 days of completion of the medical surveillance to The Director General, Department of Occupational Safety and Health, Putrajaya. This form can be downloaded from http://www.dosh.gov.my Continue in separate sheet if required.

Department of Occupational Safety and Health
Ministry of Human Resources

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