

HEALTH MONITORING FOR EXPOSURE TO HAZARDOUS CHEMICALS

GUIDE FOR PERSONS CONDUCTING
A BUSINESS OR UNDERTAKING

FEBRUARY 2013



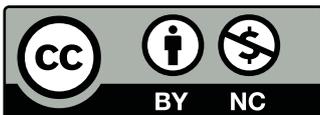
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Contact information
Safe Work Australia
Phone: 1300 551 832
Email: info@swa.gov.au
Website: www.swa.gov.au



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1. INTRODUCTION

The *Work Health and Safety (WHS) Act* places a duty on a person conducting a business or undertaking (PCBU) to ensure, so far as is reasonably practicable, the health of workers is monitored to prevent illness or injury.

The WHS Regulations place specific duties on a person conducting a business or undertaking to provide health monitoring to workers who use hazardous chemicals, including workers who are exposed to lead and asbestos.

This Guide is intended for persons conducting a business or undertaking who are required to provide health monitoring for workers. Separate guidance is available for workers and medical practitioners in the following documents:

- *Health Monitoring for Exposure to Hazardous Chemicals: Guide for Workers*
- *Health Monitoring for Exposure to Hazardous Chemicals: Guide for Medical Practitioners*
- *Hazardous Chemicals Requiring Health Monitoring*

1.1 What is health monitoring?

Health monitoring means monitoring of a person to identify changes in the person's health status because of exposure to certain substances. Health monitoring must be carried out by or be done under the supervision of a registered medical practitioner with experience in health monitoring. There are different types of health monitoring procedures used to assess exposure to hazardous chemicals, including:

Interview questions

This involves asking the worker questions about previous occupational history, medical history, lifestyle (for example dietary, smoking and drinking habits), and about symptoms related to exposure. It may also involve simple questions about how workers carry out their work, their personal hygiene at work or where they eat in the workplace. All of these questions provide information to assess current or previous exposure to hazardous chemicals.

Medical examination

This involves the use of standard clinical and medical assessments, tests and techniques to assess the presence of early or long term health effects, often at set intervals. It can include an assessment of medical history, occupational and previous exposure history, and a clinical examination. This can also include tests like spirometry (for testing lung function) and radiography, for example, a chest X-ray.

Biological effect monitoring

This is the measurement and assessment of early biological effects before health impairment occurs in exposed workers, for example measurement of reduction of cholinesterase activity levels in workers exposed to organophosphate pesticides.

Biological exposure monitoring

This involves measurement and evaluation of the levels of a hazardous chemical or its metabolites (break-down products) in body tissues, body fluids like urine or blood—for example blood lead levels, urinary cadmium—or in exhaled breath of an exposed person.

Choosing the most appropriate health monitoring method will depend on the type of chemical involved, the way the worker is exposed, the level of exposure, and if it is possible to use a proactive method, like biological exposure monitoring, which can show unacceptable levels of exposure to hazardous chemicals before adverse symptoms develop—rather than reactive methods, like a medical examination, which looks for signs and symptoms after they have developed. In many cases, more than one monitoring method may be used.

Although health monitoring is aimed primarily at identifying effects from workplace exposure to hazardous chemicals, it may also inadvertently capture information about exposures outside of work.

Health monitoring does not include air monitoring or other measures used to assess or control exposure to hazardous chemicals in the workplace. Health monitoring must never be used as an alternative to putting in place effective control measures. However, it can be used to help identify whether existing control measures are working effectively or whether new or more effective control measures should be implemented. It also provides a valuable opportunity for feedback to the PCBU from workers on the effectiveness of control measures.

It is important to remember health monitoring will only be effective if you act on the results. You should know when workers should be referred for further examination and how the results should be used to minimise risks to health and safety.

1.2 Key terms used in this Guide

Exposure standard means an exposure standard in the publication *Workplace Exposure Standards for Airborne Contaminants*.

Scheduled chemical means a chemical listed in Schedule 14 of the WHS Regulations that requires health monitoring—reproduced at Appendix A of this Guide.

Tests mean investigative techniques that can be used in the periodic assessment of individual workers to assist in determining their degree of exposure to or effect from hazardous chemicals.

2. WHEN IS HEALTH MONITORING REQUIRED?

2.1 Duty to provide health monitoring for hazardous chemicals

The WHS Regulations state that a person conducting a business or undertaking must ensure health monitoring is provided to a worker if the worker:

- is carrying out ongoing work using, handling, generating or storing hazardous chemicals and there is a *significant risk to the worker's health* because of exposure to a scheduled chemical or asbestos, see Appendix A.

There are specific requirements for lead, see Section 3.5.

Where a worker is at risk of exposure to asbestos from asbestos work, licensed or otherwise, health monitoring must also be carried out. The need for health monitoring for these workers should be determined on the basis of the potential for exposure, the frequency of potential exposure and the duration of the work being carried out. If a worker is carrying out licensed asbestos removal work, the WHS Regulations require health monitoring to be conducted prior to the worker commencing the work.

Health monitoring must also be provided if the worker:

- is using, handling, generating or storing hazardous chemicals and there is a *significant risk* the worker will be exposed to hazardous chemicals other than scheduled chemicals and either:
 - valid techniques are available to detect the effect on the worker's health, or
 - a valid way of determining exposure is available and it is uncertain on reasonable grounds whether exposure has resulted in the biological exposure standard being exceeded.

This means the need to provide health monitoring to workers is not restricted to those chemicals listed in Schedule 14 of the WHS Regulations or asbestos. The PCBU must determine the likelihood of exposure to a hazardous chemical in conjunction with the known health effects of the chemical and decide if a program of health monitoring is necessary—this can be done in consultation with professional advice from an occupational hygienist or a medical practitioner if necessary. Discussions with an occupational physician may be needed to determine if testing, for example biological monitoring, for the chemical being used is available to monitor potential effects on a worker's health status.

The PCBU should consider instigating a health monitoring program for chemicals that have severe known health effects. This includes chemicals which are known, or are presumed to be, carcinogenic, mutagenic or toxic to human reproduction, are respiratory or skin sensitisers or those with other known severe toxic effects. If there is uncertainty that working with a chemical may require a health monitoring program, specialist advice from an occupational hygienist or occupational physician should be sought. The PCBU should arrange an appointment with the registered medical practitioner for workers who are at significant health risk from exposure to hazardous chemicals or are suspected of being at significant risk at work, or if they have concerns about potential exposure.

Some examples of hazardous chemicals for which valid testing is available but are not listed in Schedule 14 of the WHS Regulations are given in Appendix B of this guide.

A checklist for providing health monitoring is included in Attachment C to this Guide.

2.2 Who pays for health monitoring?

The PCBU who engages the worker must pay all expenses for health monitoring including doctors' fees, testing and analysis costs for blood, urine or other samples, as well as other costs like travelling expenses and provide time off work.

If two or more PCBUs have a duty to provide health monitoring for a worker and have arranged for one of them to commission the health monitoring, costs of the health monitoring must be shared equally between those persons, unless otherwise agreed. For example, it may be agreed one of the persons will pay a greater proportion of the costs.

2.3 Deciding if there is significant risk

The level of risk to workers from exposure to hazardous chemicals depends on the hazards as well as the frequency, duration and amount of exposure, that is the dose. To determine the level of risk, it is necessary to draw together the information gathered about the hazardous chemical used and the way it is used in the workplace. This will involve considering:

- the nature and severity of the hazard for each hazardous chemical. This information should be available from the label and the safety data sheet (SDS) in most cases. However, in some instances the hazardous chemical that triggers health monitoring will be generated in the workplace, so a label and SDS may not always be available
- the degree of exposure of people in the workplace – this needs to be decided for each workplace, taking account of:
 - actual processes and practices in the workplace where the chemicals are used
 - the quantities of chemicals being handled
 - work practices and procedures and the way individual workers carry out their daily tasks
 - whether existing control measures adequately control exposure.

Air monitoring, which involves measuring the level of particular chemicals in the atmosphere at the workplace, can provide information about the degree of exposure in the workplace, as well as whether control measures are working effectively. Where workers report symptoms known to be associated with the hazardous chemical being used, including those provided in Chapter 5 of this Guide, this may indicate control measures are not working effectively and prompt remedial action will be required.

The risk may generally be described as 'not significant' or 'significant'. The risk can be regarded as 'not significant' if it is **unlikely** the worker will be exposed at a level that would adversely affect his or her health. A 'significant risk' means people in the workplace are **likely** to be exposed at a level that could adversely affect their health. For example, there would be a 'significant risk' if any of the following applies:

- exposure is high
- the substance used is highly toxic
- it is reasonably foreseeable leaks or spills of a hazardous chemical might occur.

2. WHEN IS HEALTH MONITORING REQUIRED?

In these circumstances, there are commonly three possibilities for describing the risk:

- the risks are significant, but already effectively controlled
- the risks are significant, and not adequately controlled
- there is uncertainty about the risks, there is not enough information about the hazards or there is uncertainty about the degree of exposure.

Where there is uncertainty about the risks, health monitoring is generally required. Air monitoring may also be required in this situation to ensure control measures are working, where serious health effects might result if control measures fail, or where the potential for exposure is high.

If risks from hazardous chemicals are already controlled in accordance with known control measures, including those that may be mentioned in the SDS, the risk is not considered to be significant and health monitoring is not required. For example, where a process is completely enclosed or workers are isolated from exposure, risks would be considered low and health monitoring would not be required.

If risks are significant but not adequately controlled, health monitoring is required. Existing control measures must also be reviewed and revised in this instance, to eliminate or minimise the risks so far as is reasonably practicable.

It is important to note that the conditions under which a person is exposed to a chemical can increase the amount of the chemical the person absorbs and increase the risk. For example, people who are exposed during strenuous activity breathe more heavily and can absorb more of the chemical. The individual characteristics of the worker, heart rate, respiration rate, diet, and whether they are a smoker can also be factors that increase the risks for workers. These factors should be considered as part of a risk assessment.

The *Code of Practice: Managing Risks of Hazardous Chemicals in the Workplace* provides further guidance on controlling exposure to hazardous chemicals.

2.4 Valid test methods for detecting health effects or exposure

Valid test methods for scheduled chemicals are known and provided in Schedule 14 of the WHS Regulations. These are reproduced in Appendix A. Other test methods may be used for scheduled chemicals provided they can detect health effects or biological levels from exposure to hazardous chemicals used at the workplace, and are equal to or better than the test methods listed in Appendix A. The decision to use other test methods than those prescribed in the WHS Regulations should be done in close consultation with the registered medical practitioner involved in the health monitoring.

If there is a significant risk to workers from exposure to hazardous chemicals that are not scheduled, you should discuss with a registered medical practitioner about whether valid test methods are available and health monitoring should be provided for the worker. Techniques and test methods used should be practical, accurate and safe. Health monitoring aims to detect adverse health effects at an early stage so action may be taken.

2. WHEN IS HEALTH MONITORING REQUIRED?

Researching and understanding the known health effects of a hazardous chemical and the known symptoms of exposure can help you to make informed decisions about what a test method might be. The SDS should contain detailed information about symptoms of exposure to the chemical, however, you should always seek professional help from the supervising medical practitioner, an occupational physician or an occupational hygienist to determine which tests should be carried out.

EXAMPLES OF APPROPRIATE TEST METHODS

Situation or type of chemical	Test method
For a chemical that is known to cause respiratory irritation or reduction in respiratory function	Spirometry (lung function) test may be used to assess exposure to the hazardous chemical.
For a chemical that is known to cause specific observable health effects like skin irritation or a rash	Health monitoring may involve simple observation of the worker's skin by an occupational nurse or through self-observation and reporting
Where exposure to the chemical is known to cause that chemical or a metabolite to be present in urine or blood	Urine or blood analysis for that chemical or metabolite may be used to assess exposure, see Section 2.5. This type of monitoring is useful to determine exposure to a chemical before a health effect can be detected. It is most useful to demonstrate the workplace controls are working and the worker is working in a safe environment. Validated analytical test methods must also be available. The analytical method should be specific to the chemical, should not deliver false positive results, and be reproducible and accurate.

Where there are no valid ways to detect illness or the link between work and illness is uncertain, health monitoring may take the form of an overview of sickness records, and symptom-reporting to a medical practitioner. Health monitoring also includes self-checks, for example skin checks for redness or itching, but this will only work if workers are told what to look for and know how to report symptoms.

2.5 Biological exposure monitoring

For some chemicals, it may be possible to determine whether the worker has been exposed to unsafe levels of a chemical by measuring and evaluating biological levels of the chemical or its metabolites (break-down products) in body tissues or body fluids, such as urine or blood, or in exhaled breath of an exposed person. This is known as biological exposure monitoring.

Non-invasive testing like urine sampling is preferred over invasive methods like blood sampling, if they are appropriate and provide the same degree of reliability and accuracy. However, for some chemicals, like inorganic lead, a blood sample is required.

3. HEALTH MONITORING PROCESS

3.1 Consultation with the worker

If you are required to provide health monitoring to a worker who will, or is likely, to use, handle, generate or store a hazardous chemical in your business or undertaking, you must give them information about the health monitoring requirements before they start work.

The Safe Work Australia publication *Health Monitoring for Exposure to Hazardous Chemicals: Guide for Workers* provides general information about health monitoring for exposure to hazardous chemicals. This publication is suitable for use by workers and it is recommended it is provided to workers who may require health monitoring.

Workers must be consulted regarding the selection of the registered medical practitioner who will supervise or perform the health monitoring.

Workers should also be told:

- possible health effects from exposure*
- health monitoring is a legal requirement in the WHS Regulations
- what a program of health monitoring aims to achieve and its benefits*
- what is involved in the health monitoring program, for example the frequency of testing and which tests, such as blood tests and respiratory tests, may be needed*
- any requirement for them to see a doctor or specialist
- how a registered medical practitioner is chosen and their qualifications
- how to report symptoms
- who pays for the health monitoring
- if and how monitoring results may affect their work tasks, for example explaining circumstances where the worker may need to move to other tasks
- the record keeping requirements
- health monitoring results are confidential and can only be disclosed to the regulator, the business or undertaking they work for, other PCBU's who have a duty to provide health monitoring for the worker, or another registered medical practitioner involved in the health monitoring, unless their consent is otherwise given.

*Note – Information should be provided so far as is known. In some instances, further details should be provided by the medical practitioner carrying out or supervising the health monitoring program.

3.2 Obtain services of a registered medical practitioner

Health monitoring must be carried out by or be done under the supervision of a registered medical practitioner with experience in health monitoring. Other than the medical practitioner having experience in the provision of health monitoring, there are no restrictions on who the medical practitioner is or by whom they are employed. For instance, practitioners may be single practitioners in a medical practice, occupational physicians who work for specialist occupational health organisations or they may provide specialist services and testing in certain areas of health monitoring like respiratory screening and chest X-rays.

The medical practitioner should prepare a program of health monitoring and either carry out the health monitoring program themselves or supervise the program undertaken by another suitably qualified person like an occupational health nurse.

The medical practitioner has the overall responsibility for health monitoring, however, they may need to seek advice from other professionals like an occupational physician, and may need to consult with other workplace health and safety professionals.

3.3 Chemicals requiring medical testing before commencing work

Baseline health monitoring is required for workers who undergo health monitoring for exposure to scheduled hazardous chemicals before they start work with that chemical. The baseline monitoring varies for each chemical. In some cases the baseline monitoring only involves collection of demographic data, previous work history and medical history. In other cases tests may be required. These may involve a physical examination, checking of respiratory function or skin checks, while in other cases testing of urine, saliva, mucous, hair or blood is required.

The PCBU should make acceptable arrangements for workers to participate in the health monitoring program, for example providing time off work to attend medical appointments related to the health monitoring program.

3.4 Information to be provided to the registered medical practitioner

Prior to commencing work with a scheduled chemical you should provide the registered medical practitioner undertaking or supervising the health monitoring with the following information:

About the business or undertaking and the worker

- name and address of your business or undertaking
- the name, date of birth, gender and current residential address of the worker

About the work

- a list of the hazardous chemicals that the worker is or will be exposed to and the dates that the worker last used the chemicals
- the work the worker is, or will be, carrying out and what has triggered the requirement for health monitoring
- if the worker has started that work, how long the worker has been carrying out that work
- the SDS for the chemical(s)
- relevant risk assessment reports, details of workplace exposure standards and results of air monitoring carried out at the workplace. This information is critical for the practitioner to understand all of the situations where workplace exposure could occur. Note the risk assessment reports should contain information about likely exposures at the workplace, including control measures that are in place to reduce exposure and investigations of results where workplace exposure standards have been exceeded.

Health monitoring report templates are provided in the separate documents for scheduled chemicals and asbestos, available from the Safe Work Australia website. These are not mandatory to use, however, they may be helpful when preparing information for the registered medical practitioner who is undertaking or supervising the health monitoring.

To ensure confidential information about the worker is not disclosed to the PCBU, the PCBU should fill in relevant parts of the form(s), such as contact details of the workplace and the work being performed, before giving it to the worker. The worker should complete other relevant parts of the form and give it to the medical practitioner.

Medical practitioners may have their own preferred proformas and specific requirements should be discussed with the medical practitioner supervising the health monitoring program.

3.5 Health monitoring requirements for inorganic lead

There are specific additional requirements for lead risk work. See WHS Regulations Part 7.2 or the section on inorganic lead in the document *Hazardous Chemicals Requiring Health Monitoring*.

For lead risk work, the WHS Regulations contain additional mandatory requirements to those described in this Guide, including:

- the frequency of biological monitoring and the circumstances when the frequency of monitoring *must* be increased
- when workers *must* be removed from and returned to lead risk work
- the requirement to arrange a medical examination for the worker within seven days after the worker is removed from the lead risk work.

3.6 Frequency of Health Monitoring

Health monitoring should be provided:

- before commencing work with the hazardous chemical. This is known as baseline monitoring and it is done so changes to the worker's health can be identified during periods of potential exposure
- during periods of exposure to the hazardous chemical, particularly where excessive exposure occurs, for example following spills or loss of containment.

Further details of frequency of testing during periods of potential exposure are provided in the guidelines for individual chemicals in the publication *Hazardous Chemicals Requiring Health Monitoring*

- where the worker has concerns that may relate to exposure to the hazardous chemical, for example where relevant symptoms are identified
- at termination of work with the hazardous chemical.

4. HEALTH MONITORING REPORT

You must take all reasonable steps to obtain a health monitoring report from the practitioner who is carrying out or supervising the monitoring as soon as practicable after the completion of the monitoring program, or at regular intervals for longer-term or ongoing health monitoring programs.

4.1 Content of health monitoring report

On receiving the health monitoring report, you should check it contains:

- the name and date of birth of the worker
- the name registration number and signature of the registered medical practitioner
- name and address of the business or undertaking
- the date health monitoring was carried out
- any test results that indicate whether or not the worker has been exposed to a hazardous chemical; for lead – any test results that indicate the worker has reached or exceeded the relevant blood lead level for that worker under regulation 415 of the WHS Regulations
- any advice that test results indicate the worker may have contracted a disease, injury or illness as a result of carrying out the work that triggered the requirement for health monitoring
- any recommendation that remedial measures be taken, including whether the worker can continue to carry out the type of work that triggered the requirement for health monitoring
- whether medical counselling is required for the worker in relation to the work that triggered the requirement for health monitoring.

The report should also contain:

- the date of sampling if blood, urine or other samples are taken
- results of biological monitoring and other tests carried out; for inorganic lead, the report must also contain the details of the pathology service used to carry out tests.

The health monitoring report should only contain information relating to the health monitoring program for the chemical(s) being used. It should not contain other confidential health information on workers, unless there is an obligation the PCBU should know. The report will not contain details of medical conditions disclosed to or diagnosed by the medical practitioner conducting the health monitoring if these have no relevance or bearing on the work being performed. If the worker has a pre-existing medical condition which may exacerbate the health effects of chemicals, it may be useful if this is brought to your attention so appropriate measures can be put in place. However, details of pre-existing medical conditions can only be included in the health monitoring report with the worker's written permission.

The medical practitioner will inform you of pastimes to be avoided, like tobacco smoking, or dietary intakes which may exacerbate the effects of hazardous chemicals being used.

4.2 Responding to the information in the report

Once you have the health monitoring report from the registered medical practitioner, you should consider the results and recommendations and advice contained in it.

As soon as practicable, you must provide a copy of the health monitoring report to:

- the worker
- all other PCBUs who have a duty to provide health monitoring for the worker
- the regulator if the report contains any of the following:
 - advice that the test results indicate the worker may have contracted a disease, injury or illness as a result of carrying out work with the chemical
 - recommendation that remedial measures be taken including whether the worker can continue to carry out work with the hazardous chemical that triggered the requirement for health monitoring
 - for lead risk work, test results that indicate the worker has reached a blood lead level at or above the relevant removal level.

Recommendations involving the worker

You should arrange for the worker to see the medical practitioner again if it is recommended in the health monitoring report and explain to the worker remedial measures that need to be taken. Treatment programs for adverse health effects should only be discussed between the worker and the medical practitioner.

Recommendations for the PCBU

If the report recommends a worker should not be exposed to a hazardous chemical for a specified period of time or should only work under conditions specified by the practitioner, these recommendations must be followed. For example, you may need to assign the worker to alternative work or another location where exposure to the hazardous chemical will not occur. This should be done after consultation with the worker and the practitioner. The worker must not return to that work until cleared to do so by the medical practitioner.

Examine work practices and procedures at the workplace to determine whether tasks are being performed correctly and whether controls are being bypassed. If necessary, review and revise your training programs.

You must also review and, if necessary, revise the control measures for the hazardous chemical. This is also necessary if the report contains test results that indicate the worker has been exposed to the hazardous chemical and has elevated levels of the hazardous chemical or metabolites in his or her body for that chemical, or if there is advice that the test results indicate the worker may have contracted a disease, injury or illness from carrying out work with the chemical.

You should continue to provide workers who have been removed from work with hazardous chemicals, or transferred to other work, with information concerning the results of workplace assessments and their health status.

4.3 Record of health monitoring reports

Health monitoring reports for workers must be identified as a record in relation to that worker. These records should be kept separate from information obtained for other purposes, for example records of examinations that are not connected with health monitoring.

CONFIDENTIALITY

Health monitoring reports and results must be kept as confidential records and must not be disclosed to another person without the worker's written consent, except where the records are required to be given under the WHS Regulations to any of the following:

- the regulator
- another PCBU who has a duty to provide health monitoring for the worker
- a person who must keep the record confidential under a duty of professional confidentiality.

Note: health monitoring may include longer-term epidemiological studies.

Health monitoring reports should be kept separate from normal workers' records like payroll or human resources data, to prevent the reports being accessed inadvertently by unauthorised people. The report cannot be used for any purpose other than for health monitoring. Similarly, blood or tissue samples, X-rays, questionnaires or other materials taken for health monitoring must not be used for any other purpose.

HOW LONG TO KEEP THE RECORD

Health monitoring records for all workers must be kept for at least 30 years after the record is made, even if the worker no longer works at your workplace. For asbestos health monitoring, these records must be kept for at least 40 years, due to the long period of time it can take for asbestos-related disorders to develop.

5. INFORMATION ON SCHEDULED CHEMICALS AND ASBESTOS

This Chapter contains supplementary information on the scheduled chemicals in Appendix A to assist in assessing the risks of exposure. Key information includes work activities that represent a high risk of exposure for each chemical, routes of entry into the body, as well as key symptoms of exposure. Information about a chemical's carcinogenicity, germ cell mutagenicity and reproductive toxicity classification is provided. This information is provided on an advisory basis and classifications are taken from the European Union's Annex VI to Regulation (EC) No 1272/2008, updated by the 1st Adaption to Technical Progress to the Regulation*. Annex VI includes lists of GHS classification information for certain substances or groups of substances. These classifications are legally binding within the European Union.

Further toxicological information is available in the *Hazardous Chemicals Requiring Health Monitoring guidelines*.

5.1 Acrylonitrile

Acrylonitrile vapour is heavier than air. It has a pungent odour of onion or garlic that does not provide adequate warning of hazardous concentrations. Exposure can result in asphyxiation in poorly ventilated, enclosed or low-lying areas.

WORK ACTIVITIES THAT MAY REPRESENT A HIGH RISK EXPOSURE

The major uses of acrylonitrile are in the manufacture of polymers, resins, plastics and nitrile rubbers. Examples of work activities involving acrylonitrile which require special attention when assessing exposure include acrylic fibre production, especially in the procedure where solvent is removed from newly-formed fibres.

ROUTE OF ENTRY INTO THE BODY

The primary route of acrylonitrile entry into the body is through inhalation. Acrylonitrile can also be absorbed through the skin in quantities sufficient to cause health effects.

ACUTE EFFECTS

Acute overexposure can cause rapid onset of eye, nose, throat and airway irritation, headache, sneezing, nausea and vomiting. Weakness and light-headedness may also occur.

Acrylonitrile can act as an asphyxiant, causing symptoms like profound weakness, headache, nausea, shortness of breath, dizziness, collapse, convulsions, asphyxia and death.

Prolonged skin contact with the liquid may result in absorption with systemic effects (these are health effects that occur in other parts of the body from where exposure occurred) and the formation of large blisters several hours after exposure.

CHRONIC EFFECTS

Repeated spills on exposed skin may result in dermatitis. Acrylonitrile is a skin sensitiser. Chronic inhalation may cause headache, insomnia, irritability, nose bleeds, respiratory difficulties and abnormal liver function.

GHS CARCINOGEN CLASSIFICATION

Carcinogenicity Category 1B (May cause cancer).

■ * Source: <http://esis.jrc.ec.europa.eu/index.php?PGM=cla>.

5.2 Arsenic (inorganic)

WORK ACTIVITIES THAT MAY REPRESENT A HIGH RISK EXPOSURE

Examples of work activities involving inorganic arsenic which require special attention when assessing exposure include:

- manufacture of arsenic compounds, the most important of which is arsenic trioxide (As_2O_3)
- formulation and application of insecticides (lead arsenate, calcium arsenate, arsenic trioxide and pentoxide), weed killers, rat poison, fungicides (copper aceto-arsenite or Paris green), wood preservative like copper chrome arsenic (arsenic pentoxide); in the past used as cattle dip (arsenic trioxide) and sheep dip (sodium arsenite). Only a few arsenic-containing wood preservatives and fungicides are currently registered for use in Australia
- production of pigments (arsenic trisulphide and trioxide), ceramic enamels and anti-fouling paints (arsenic trioxide)
- hide preservation in the leather industry (arsenic trioxide)
- hardening copper, lead and other alloys
- copper, zinc and lead smelting.

CONTROLS

The PCBU must ensure exposure to inorganic arsenic is adequately controlled. Exposure should be controlled in accordance with the hierarchy of controls. Processes should be enclosed where practicable. Due to the severe toxicity of arsenic, personal protective equipment (PPE) should always be used regardless of other control measures in place.

POTENTIAL HEALTH EFFECTS FOLLOWING USE OF INORGANIC ARSENIC

The relative toxicity of arsenic containing compounds depends primarily on its chemical type, valence state, for example As(III) or As(V), solubility and physical form. Soluble compounds of arsenic, for example sodium arsenite, are more toxic than insoluble compounds like arsenic sulphide.

The toxicity of trivalent arsenite, for example, arsenic trioxide or arsenic trichloride, is typically greater than that of pentavalent arsenate (arsenic pentoxide). Arsine gas (AsH_3) produces clinical symptoms different from other arsenic compounds and is the most toxic arsenic compound.

ROUTE OF ENTRY INTO THE BODY

The primary route of inorganic arsenic entry into the body is through inhalation of arsine gas or airborne arsenic fumes or dusts. The particle size of airborne arsenic determines whether arsenic will reach the lower respiratory tract or be deposited in the upper airways and be swallowed. In addition, soluble forms of inorganic arsenic compounds are well absorbed from the gastro-intestinal tract (60-90 per cent). Some arsenic compounds, for example arsenic acid and arsenic trichloride, may be absorbed through the skin. Inorganic arsenic does not cross the blood-brain barrier but does cross the placenta meaning extra precautions may be necessary if pregnant women carry out tasks involving arsenic.

5. INFORMATION ON SCHEDULED CHEMICALS AND ASBESTOS

ACUTE EFFECTS

Acute clinical symptoms from arsenic exposure will vary widely with the type and chemical state of the arsenic involved. Acute effects are generally the result of short-term exposures to high concentrations of arsenic.

Arsine gas is a potent haemolytic poison (it destroys blood cells) in both acute and chronic exposures. Subsequent jaundice may be severe. Signs and symptoms of toxicity include nausea, vomiting and diarrhoea, apprehension and malaise (a general feeling of being unwell, discomfort or uneasiness), rapid heart rate and difficulty breathing. Acute kidney failure is frequent and often fatal.

Acute poisoning by arsenic compounds other than arsine gas rarely occurs in industry, but has been reported to have occurred as a result of inhalation and through skin absorption, as well as from ingestion. Arsenic can cause convulsions, coma and death in severe poisoning.

Exposure by oral ingestion to toxic doses of arsenic salts leads within one to two hours to acute gastrointestinal symptoms of vomiting and severe abdominal pain. Cardiovascular effects progress through vasodilation (decrease in blood pressure), cardiac depression then shock. The CNS effects are headache, coma, convulsions, and cerebral oedema (fluid accumulation in the brain). Sensory loss in the peripheral nervous system and motor dysfunction (problems controlling movement) can occur one to two weeks after large exposures.

If inhaled, mucous membrane irritation, difficulty breathing and pulmonary oedema (fluid accumulation in the lungs) may occur.

CHRONIC EFFECTS

- contact dermatitis, scaling, blistering of the skin, hyperpigmentation (darkening of the skin) and hyperkeratotic lesions on the skin
- in the presence of sweat, skin abrasions, chafing or wounds, arsenic readily promotes ulceration of the skin
- conjunctivitis
- mucous membrane irritation and perforation of the nasal septum
- weakness, loss of appetite, gastrointestinal disturbances
- liver disease
- damage to nervous system
- destruction of blood cells.

GHS CARCINOGEN AND REPRODUCTIVE TOXICANT CLASSIFICATIONS

The European Union has determined arsenic acid and its salts with the exception of those specified elsewhere in Annex VI, arsenic trioxide and arsenic pentoxide are classified as Carcinogenicity Category 1A (May cause cancer).

Lead hydrogen arsenate is classified as Carcinogenicity Category 1A (May cause cancer) and Reproductive Toxicity Category 1A (May damage the unborn child, suspected of damaging fertility).

5.3 Asbestos

Asbestos is the fibrous form of mineral silicates belonging to the serpentine and amphibole groups of rock-forming minerals. The commercial types which have been used in Australia are the serpentine: chrysotile (white asbestos); and the amphiboles: crocidolite (blue asbestos) and amosite (brown or grey asbestos).

WORK ACTIVITIES THAT MAY REPRESENT A HIGH RISK EXPOSURE

Examples of work activities involving asbestos which require special attention when assessing exposure include:

- asbestos removal and demolition work in buildings, power stations, boilers and ships
- maintenance workers, like electricians, and computer cabling installers and air-conditioning installers working in ceiling spaces of buildings where sprayed asbestos has not been removed, sealed or encapsulated.

In some industries, like mining, major excavation and siteworks, for example road building, amphiboles like tremolite and anthophyllite, may be present as geological contaminants.

CONTROLS

The WHS Regulations contain specific requirements for managing risks associated with asbestos. Further information is available in the following Safe Work Australia publications:

- *Code of Practice - How to Safely Remove Asbestos*
- *Code of Practice - How to Manage and Control Asbestos in the Workplace.*

ROUTE OF ENTRY INTO THE BODY

The primary route of asbestos entry into the body is through inhalation.

RESPIRATORY EFFECTS

Inhalation of high concentrations of asbestos may result in asbestosis, a progressive scarring of lung tissue. Further development of scar tissue (fibrosis) may occur after the cessation of exposure. Effects of asbestos on the pleurae (a thin membrane which envelops the lungs) include plaques with and without calcification, diffuse pleural thickening and effusions (excess fluid).

The two main forms of cancer associated with the inhalation of asbestos are lung cancer and mesothelioma. Mesothelioma is cancer of the pleura or less commonly, the peritoneum (lining of the abdominal cavity) and can result from brief periods of exposure and a pattern of repeated exposure can lead to a substantial cumulative exposure.

GHS CARCINOGEN CLASSIFICATION

Carcinogenicity Category 1A (May cause cancer)

5.4 Benzene

Benzene, an aromatic hydrocarbon, is a natural component of crude and refined petroleum.

WORK ACTIVITIES THAT MAY REPRESENT A HIGH RISK EXPOSURE

Examples of work activities involving benzene which may require special attention when assessing exposure include:

- refining operations, for example maintenance of equipment used for handling benzene-containing refinery streams and sampling benzene-containing refinery streams in open containers
- chemical manufacturing
- handling of petrol, that is, storage and transport, for example filling rail tankers and top-filling road tankers with petrol
- motor vehicle repair – working on vehicle fuel systems
- plastics and rubber manufacturing
- steel production – by-product of coal coking
- firefighting – emission from burning synthetic polymers like polyvinyl chloride and urethane foam.

ROUTE OF ENTRY INTO THE BODY

The routes of benzene entry into the body are inhalation, ingestion and skin absorption.

ACUTE EFFECTS

Acute exposure to high concentrations of benzene vapours can result in irritation of the skin, eyes and respiratory system and in central nervous system depression and arrhythmias.

Central nervous system: The acute effects from exposure to high levels of benzene, that is 500 to 1000 parts per million (ppm) in air, are central nervous system depression, narcosis, unconsciousness, coma and death. Benzene concentrations of about 20 000 ppm are fatal to humans within five to 10 minutes.

Exposures of 50 to 150 ppm for several hours can cause headaches, a feeling of weariness and general weakness. Symptoms of CNS depression include headache, nausea and vomiting, dizziness, slurred speech, euphoria, fatigue, weakness, irritability, disorientation, confusion, loss of consciousness and death.

Respiratory system: All organic solvents irritate the respiratory tract to some degree as a consequence of the defatting action of solvents. Respiratory tract irritation from solvents is usually confined to the upper airways, including the nose and sinuses. Overexposure can cause accumulation of fluid in the lungs, exacerbation of asthma or, less commonly, induction of reactive airway dysfunction. Symptoms of irritation of the upper respiratory tract are marked by sore nose and throat, cough and possibly chest pain. If the eyes are not protected by vapour goggles irritation of the eyes may result.

5. INFORMATION ON SCHEDULED CHEMICALS AND ASBESTOS

CHRONIC EFFECTS

Benzene is both haematotoxic (destroys red blood cells) and leukaemogenic (causes leukaemia). Chronic exposure to levels of 100-500 ppm have resulted in depression of bone marrow haemopoiesis (process for production of blood cells). For bone marrow depression, the lowest observed adverse effect level in humans is 7.6 ppm.

Acute myeloid leukaemia occurs more frequently in workers exposed to benzene at work. Several reports suggest exposure to benzene may be related to non-Hodgkins lymphoma and multiple myeloma.

GHS CARCINOGEN AND GERM CELL MUTAGEN CLASSIFICATIONS

Carcinogenicity Category 1A (May cause cancer) and Germ Cell Mutagenicity Category 1B (May cause genetic defects).

5.5 Cadmium

WORK ACTIVITIES THAT MAY REPRESENT A HIGH RISK EXPOSURE

Examples of work activities involving cadmium and its compounds which require special attention include:

- processes like welding, soldering, oxy-cutting and smelting
- welding or oxy-cutting of cadmium alloy and cadmium plate
- the use of cadmium-silver alloys for silver soldering or brazing
- electroplating
- manufacture of cadmium alloys
- extraction of cadmium from mineral ore smelters
- opening containers and weighing out cadmium powders
- charging cadmium powders into process plant
- grinding, discharging and packaging cadmium powders
- working with nickel-cadmium batteries
- manufacture and handling of paints and plastics containing cadmium pigments and the recycling of these plastics
- textile production.

Special attention should be given to acute exposures, including high temperature processes where cadmium fumes are evolved.

Non-workplace exposure to cadmium can occur through smoking cigarettes.

5. INFORMATION ON SCHEDULED CHEMICALS AND ASBESTOS

ROUTE OF ENTRY INTO THE BODY

The primary route of entry of cadmium into the body is through inhalation. Only small cadmium particles are absorbed by the alveoli, and these small particles are typically found in fumes and cigarette smoke.

There is some risk of ingestion if personal hygiene is inadequate. Absorption of cadmium by the body may be increased in the presence of calcium and iron deficiency. Skin absorption is not significant.

ACUTE EFFECTS

Acute cadmium poisoning has been reported among workers after exposure to the intensely irritating fume of heated cadmium, with symptoms delayed for several hours. Signs include severe tracheobronchitis, inflammation of the lung, and accumulation of fluid in the lungs. The mortality rate for the acute pulmonary disease is about 20 per cent. Average airborne concentrations responsible for fatal cases have been estimated at 50 mg/m³ for a period of about one hour. Relatively mild cases resemble metal fume fever.

High ingestion exposure of soluble cadmium salts causes acute gastroenteritis.

CHRONIC EFFECTS

Long-term work-related exposure to cadmium has caused severe chronic effects, predominantly in the lungs and kidneys. Renal toxicity may be caused by chronic inhalation or chronic ingestion of cadmium. Proteinuria (excretion of protein in the urine) is commonly associated with exposure to cadmium and is irreversible. Several studies of both occupationally and environmentally exposed populations have shown cadmium exposure as low as 2-4 nmol/mmol creatinine, approximately equivalent to 2-4 µg/g creatinine, is associated with the occurrence of tubular proteinuria.

Cadmium exposure is also associated with lung effects, primarily characterised by chronic obstructive airway disease. Early minor changes in lung function tests may progress to respiratory failure with continued cadmium exposure.

Mild, reversible anaemia caused by depression of haemoglobin is also associated with chronic cadmium exposure.

GHS CARCINOGEN, GERM CELL MUTAGEN AND REPRODUCTIVE TOXICANT CLASSIFICATIONS

The following are some cadmium-containing chemicals with carcinogen, germ cell mutagen and reproductive toxicant classifications:

- Cadmium (non-pyrophoric) and Cadmium oxide (non-pyrophoric): Carc. 1B, Muta. 2, Repr. 2.
- Cadmium (pyrophoric): Carc. 1B, Muta. 2, Repr. 2
- Cadmium chloride: Carc. 1B, Muta. 1B, Repr. 1B
- Cadmium fluoride: Carc. 1B, Muta. 1B, Repr. 1B
- Cadmium sulphate: Carc. 1B, Muta. 1B, Repr. 1B
- Cadmium sulphide: Carc. 1B, Muta. 2, Repr. 2
- Cadmium cyanide: Carc. 2
- Cadmium fluorosilica (cadmium hexafluorosilicate(2-)): Carc. 2
- Cadmium formate (cadmium diformate): Carc. 2
- Cadmium iodide: Carc. 2.

5. INFORMATION ON SCHEDULED CHEMICALS AND ASBESTOS

KEY

Abbreviation	Meaning	Hazard statement
Carc. 1B	Carcinogenicity Category 1B	May cause cancer
Carc. 2	Carcinogenicity Category 2	Suspected of causing cancer
Muta. 1B	Germ Cell Mutagenicity Category 1B	May cause genetic defects
Muta. 2	Germ Cell Mutagenicity Category 2	Suspected of causing genetic defects
Repr. 1B	Reproductive Toxicity Category 1B	May damage fertility, may damage the unborn child
Repr. 2	Reproductive Toxicity Category 2	Suspected of damaging fertility, suspected of damaging the unborn child

5.6 Chromium (inorganic)

WORKPLACE SKIN CARE PROGRAM

The PCBU must ensure workers' skin is inspected weekly by a competent person. The competent person is one who has acquired through training, qualification or experience, the knowledge and skills to carry out this task. This could be an occupation health nurse, for example. Particular attention should be paid to the skin of the hands and forearms. Where skin abnormalities occur, the PCBU must arrange for the worker to see the medical practitioner.

RESPIRATORY SYMPTOMS

Respiratory symptoms should be reported to the registered medical practitioner.

WORK ACTIVITIES THAT MAY REPRESENT A HIGH RISK EXPOSURE

Examples of work activities involving inorganic chromium and its compounds which require special attention include:

- welding, cutting and hard-facing of stainless steel
- manual metal arc welding of high chromium steels
- chrome plating
- refractory production
- addition of cement to gravel and sand to make concrete
- leather tanning
- timber preservation using copper chrome arsenate
- chromate use in the textile industry
- chrome pigment use, for example in paints.

5. INFORMATION ON SCHEDULED CHEMICALS AND ASBESTOS

POTENTIAL HEALTH EFFECTS FOLLOWING EXPOSURE TO INORGANIC CHROMIUM

The adverse effects of chromium and its inorganic compounds vary according to valence state (for example Cr(III) or Cr(VI)), water solubility and dose. However, the hexavalent chromium compounds—chromates, dichromates and chromic acid—are of most concern in both acute exposures and chronic exposure to lower concentrations.

ROUTE OF ENTRY INTO THE BODY

The routes of inorganic chromium entry into the body are through inhalation, ingestion and skin absorption. Work-related exposure generally occurs through inhalation and skin contact. The absorption of chromium is dependent on the valence state and water-solubility of the chromium compound. Soluble forms of hexavalent chromium are readily absorbed by inhalation. Skin absorption may also occur.

ACUTE AND CHRONIC EFFECTS

Hexavalent chromium

Irritant and corrosive effects

Chromium (VI) in aerosols, dusts or liquids irritates or even corrodes the skin and the mucous membranes of the eyes and respiratory tract. The spraying of chromic acid can give rise to serious eye lesions and intense exposure to chromic acid particulates may give rise to fluid build-up in the lungs.

Chrome ulcers (chrome 'holes'). Deep, round holes, clearly marked, usually at the base of the nails, the finger joints, the skin between the fingers, the back of the hand and the forearm. They may also appear at other sites. The lesions are only slightly painful, tend to be clean but they take a long time to heal and scars are left.

Perforation of the nasal septum. Intense chromium (VI) airborne exposure for two weeks, or less intense exposure for several months may cause painless ulceration, accompanied by foul nasal discharge.

Allergic effects

Allergic dermatitis. At concentrations below those resulting in irritation, skin sensitivity is the most common effect following exposure to chromium compounds. Chromium is one of the most common contact sensitisers in industrialised countries. Allergic dermatitis is well known in printers, cement workers, metal workers, painters, textile workers and leather tanners. Chromate sensitivity, once induced, may prove difficult to deal with in multiple settings and is very persistent once developed.

Asthma. Inhaled chromium is a respiratory tract irritant, resulting in airway irritation and airway obstruction. Respiratory sensitisation, may develop with chemical substances of low molecular mass, resulting in generalised bronchospasm and typical asthmatic attacks, which occur on subsequent low exposure levels to dusts, aerosols or welding fumes.

Chronic effects

Chronic obstructive pulmonary disease. Prolonged inhalation of chromium (VI) particulates can cause chronic respiratory irritation with hyperaemia (increased blood flow to tissues), chronic inflammation of the lung, chronic bronchitis, broncho-pneumonia, and emphysema.

Trivalent chromium

Trivalent compounds are generally poorly absorbed through intact skin. However, once the skin is broken, absorption may occur. The trivalent compounds are allergenic, but much less so than the hexavalent compounds.

5. INFORMATION ON SCHEDULED CHEMICALS AND ASBESTOS

GHS CARCINOGEN, GERM CELL MUTAGEN AND REPRODUCTIVE TOXICANT CLASSIFICATIONS

The following are some chromium-containing chemicals with carcinogen, germ cell mutagen and reproductive toxicant classifications:

- Chromium (VI) trioxide: Carc. 1A , Muta. 1B, Repr. 2 (Suspected of damaging fertility)
- Zinc chromates including zinc potassium chromate: Carc. 1A
- Ammonium dichromate: Carc. 1B, Muta. 1B, Repr. 1B (May damage fertility, may damage the unborn child)
- Calcium chromate: Carc. 1B
- Chromic oxychloride: Carc. 1B, Muta. 1B
- Chromium-III-chromate: Carc. 1B
- Chromium (VI) compounds, with the exception of barium chromate and of compounds specified elsewhere in Annex VI: Carc. 1B
- Lead sulfochromate yellow [C.I. Pigment Yellow 34]: Carc. 1B, Repr. 1A (May damage the unborn child, suspected of damaging fertility)
- Lead chromate: Carc. 1B, Repr. 1A (May damage the unborn child, suspected of damaging fertility)
- Lead chromate molybdate sulfate red [C.I. Pigment Red 104]: Carc. 1B, Repr. 1A (May damage the unborn child, suspected of damaging fertility)
- Potassium chromate: Carc. 1B, Muta. 1B
- Potassium dichromate: Carc. 1B, Muta. 1B, Repr. 1B (May damage fertility, may damage the unborn child)
- Sodium chromate (VI): Carc. 1B, Muta. 1B, Repr. 1B (May damage fertility, may damage the unborn child)
- Sodium dichromate: Carc. 1B, Muta. 1B, Repr. 1B (May damage fertility, may damage the unborn child)
- Strontium chromate: Carc. 1B
- 2:1 mixture of: 4-(7-hydroxy-2,4,4-trimethyl-2-chromanyl)resorcinol-4-yl-tris(6-diazo-5,6-dihydro-5-oxonaphthalen-1-sulfonate) and 4-(7-hydroxy-2,4,4-trimethyl-2-chromanyl)resorcinolbis(6-diazo-5,6-dihydro-5-oxonaphthalen-1-sulfonate): Carc. 2
- Trisodium-bis(7-acetamido-2-(4-nitro-2-oxidophenylazo)-3-sulphonato-1-naphtholato)chromate(1-): Muta. 2.

5. INFORMATION ON SCHEDULED CHEMICALS AND ASBESTOS

KEY

Abbreviation	Meaning	Hazard statement
Carc. 1A	Carcinogenicity Category 1A	May cause cancer
Carc. 1B	Carcinogenicity Category 1B	May cause cancer
Carc. 2	Carcinogenicity Category 2	Suspected of causing cancer
Muta. 1B	Germ Cell Mutagenicity Category 1B	May cause genetic defects
Muta. 2	Germ Cell Mutagenicity Category 2	Suspected of causing genetic defects
Repr. 1A	Reproductive Toxicity Category 1A	Hazard statements vary between chemicals. See the information above.
Repr. 1B	Reproductive Toxicity Category 1B	Hazard statements vary between chemicals. See the information above.
Repr. 2	Reproductive Toxicity Category 2	Hazard statements vary between chemicals. See the information above.

5.7 Creosote

WORK ACTIVITIES THAT MAY REPRESENT A HIGH RISK EXPOSURE

Coal tar creosote is a timber preservative for use where there is a high fungal decay and termite hazard in the ground or in marine and fresh waters. Uses include marine piles, jetty bracing, sea walls, railway sleepers, power or telecommunication line poles. Work-related exposure to creosote may occur during manufacture, use, transport, or disposal of creosote or creosoted wood products.

Non-wood uses or sources of exposure include anti-fouling applications on concrete marine pilings, component of roofing pitch, fuel oil and a lubricant for die moulds, rubber or tyre industry, iron foundry work, steel plant work, aluminium smelters, coke or gas manufacturing plants, and clean-up of creosote contaminated sites. Other reported uses include animal and bird repellent, insecticide, animal dip and fungicide.

ROUTE OF ENTRY INTO THE BODY

The routes of creosote entry into the body are through inhalation and skin absorption. Accidental ingestion is unlikely unless poor hygiene and work practices allow it.

PHOTOSENSITIVITY

Creosote is known to cause photosensitisation. Photosensitivity is an abnormally high reactivity in the skin or eyes to ultraviolet radiation or natural sunlight. It may be induced by ingestion, inhalation or skin contact with certain substances known as photosensitisers. Symptoms will vary with the amount of ultraviolet radiation, type and amount of photosensitiser, skin type, and age and gender of the person exposed.

Photosensitisation of the skin and eyes can be caused by exposure to specific industrial chemicals like creosote. The skin can be affected by direct contact or by inhalation. The eyes can be affected by volatile fumes. In certain occupations, the risk from exposure to particular photosensitising chemicals and solar ultraviolet radiation is severe. For example, exposure to tar and sunlight can cause precancerous and cancerous skin lesions. Exposure to coal tar fumes can cause simultaneous inflammation of the conjunctiva and cornea.

5. INFORMATION ON SCHEDULED CHEMICALS AND ASBESTOS

Where workers report photosensitivity, an appointment should be arranged with the medical practitioner and workers should receive counselling on the potential health effects of creosote on the skin.

Where a health monitoring report indicates photosensitivity or other health effects related to exposure, the PCBU must review control measures and carry out recommended remedial action. The worker must be informed of the results of the health monitoring.

ACUTE EFFECTS

Deaths have occurred following ingestion of about 1 to 2 g of creosote in children or about 7 g in adults. Symptoms of acute poisoning include:

- systemic (health effects that occur in other parts of the body from where exposure occurred) – nausea and vomiting, diarrhoea, anorexia and difficulty in swallowing, salivation, abdominal discomfort, respiratory distress, cyanosis (blue colouration of the skin), pupillary changes, convulsive movements, rapid pulse and shock
- neurological – headaches, fainting, vertigo and mental disturbances.

Contact with creosote or creosote vapour may cause irritation of the skin. The skin may become red, papular (small solid lesions), vesicular (sac-like lesions) or ulcerated, depending on the period of exposure. Increased photosensitisation may occur, particularly on the face or hands. Vapours and contact can produce an intense burning of the membranes of the eyes and respiratory tract. Eye contact can lead to conjunctivitis and keratitis (inflammation of the cornea).

CHRONIC EFFECTS

Chronic exposure may provide sufficient absorption to show the systemic effects listed above.

GHS CARCINOGEN CLASSIFICATION

Creosote, from distillation of coal tar, is classified as Carcinogenicity Category 1B (May cause cancer).

5.8 Crystalline silica

WORK ACTIVITIES THAT MAY REPRESENT A HIGH RISK EXPOSURE

Crystalline silica is found in varying proportions in aggregates, mortar, concrete and stone. Examples of work activities involving crystalline silica which require special attention when assessing exposure include:

- excavation, earth moving and drilling plant operations
- clay and stone processing machine operations
- paving and surfacing
- mining and mineral ore treating processes
- construction labouring activities
- brick, concrete or stone cutting, especially using dry methods
- abrasive blasting—blasting agent must not contain >1 per cent crystalline silica
- foundry casting.

5. INFORMATION ON SCHEDULED CHEMICALS AND ASBESTOS

CONTROLS

A combination of controls is generally necessary to control exposure to crystalline silica dust. Wherever possible, wet processes and water suppression systems should be used to prevent dust generation and disbursement. Ventilation and good house-keeping will be necessary. Restricting access to the work area and staff rotation will assist in reducing exposure.

ROUTE OF ENTRY INTO THE BODY

The primary route of crystalline silica entry into the body is through inhalation.

ACUTE EFFECTS

Symptoms of exposure to crystalline silica include respiratory irritation, shortness of breath and asthma symptoms. Acute silicosis (lung fibrosis) can occur after short exposure to very high levels of silica. This could occur in exposure in confined spaces where respiratory protection is not worn. The condition causes rapidly progressive dyspnoea (shortness of breath) and death, usually within months of onset.

CHRONIC EFFECTS

Chronic silicosis may be the simple type where single nodules (masses of tissue) are present in the lung or it may progress to massive fibrosis with substantial impairment of lung function. Accelerated silicosis is rare but can develop within two to five years with intense exposure to free silica. An increased susceptibility to tuberculosis occurs in workers with established silicosis.

Lower levels of chronic exposure to silica can cause chronic bronchitis and chronic obstructive airways disease.

Several studies have also linked crystalline silica with renal disease. Some studies have indicated crystalline silica is a potential human carcinogen, but have provided little support that work-related silica exposure is a direct acting cancer initiator. However, there is strong evidence people with many forms of pulmonary fibrosis, including silicosis, have a major risk of developing lung cancer.

5.9 Isocyanates

WORK ACTIVITIES THAT MAY REPRESENT A HIGH RISK EXPOSURE

The largest volume use of isocyanates is in the production of polyurethane foams. Examples of work activities involving isocyanates which 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, for example in vehicle paints
- processes where heat decomposition of polyurethane products occurs, like in welding, heat removal of electrical insulating varnishes and hot wire cutting of foam.

5. INFORMATION ON SCHEDULED CHEMICALS AND ASBESTOS

Spray painters using two-pack polyurethane paints are the group at highest risk. The repair and refinishing of cars entails the sprayed on application of isocyanate-containing coatings on almost every vehicle. The most commonly used diisocyanates are toluene diisocyanate (TDI), methylene diphenyl diisocyanate (MDI) and hexamethylene diisocyanate (HDI). The risk of exposure depends on the volatility of the compound and the application process. The most volatile of the isocyanates are those with low molecular weight like HDI and TDI used in spray painting and polyurethane foam manufacturing. More recently the isocyanates like HDI used in paints have been partially polymerised into the form of pre-polymers so they become less volatile, but the spray painting process creates a mist of easily inhaled fine particles.

CONTROLS AND PPE

The PCBU must ensure exposure to isocyanate is adequately controlled. The level of control required depends on the process being employed. Where appropriate, for example in spraying operations, engineering controls like closed systems and mechanical ventilation should be used to minimise exposure to isocyanates and personal protective equipment (PPE) should not be used as a substitute for other control measures. However, given the potentially significant risks isocyanates pose to workers, PPE should be used where isocyanate exposure may occur. The PCBU must provide PPE which is appropriate for the work and be properly fitted and regularly maintained. Both the PCBU and the worker are responsible for ensuring the PPE is used as directed, and is maintained in accordance with manufacturer's instructions.

ROUTE OF ENTRY INTO THE BODY

The primary route of isocyanate entry into the body is through inhalation, however, adverse health effects can also be experienced by skin contact.

ACUTE EFFECTS

HDI and TDI and other volatile isocyanates are acute irritants of the eyes, mucous membranes, respiratory tract and skin. Isocyanate splashes in the eyes can cause severe chemical conjunctivitis.

Mild respiratory exposure may cause slight irritation of the nose and throat and headaches. With higher exposure there may be acute bronchial irritation with coughing, shortness of breath and bronchospasm, abdominal distress, nausea and vomiting, chemical pneumonitis (inflammation of the lung tissue) and pulmonary oedema (fluid accumulation in the lungs). Reactive airways dysfunction syndrome (RADS) is new onset asthma which begins within hours following a single exposure to inhaled irritants at very high concentrations and continues to be symptomatic at three months or longer.

Acute dermatitis results from either massive skin contamination or a hyper-responsiveness of the skin.

CHRONIC EFFECTS

Chronic exposure to isocyanates can cause contact dermatitis, immune sensitisation and asthma and, less commonly, hypersensitivity pneumonitis.

Isocyanates appear to be weak human skin irritants and sensitisers. 4,4'-diisocyanate dicyclohexyl methane is an exception, being a potent skin sensitiser. 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 like blistering and swelling.

5. INFORMATION ON SCHEDULED CHEMICALS AND ASBESTOS

Smoking has been identified as increasing the risk of work-related asthma in workers exposed to isocyanates.

Sensitisation usually occurs after a latency period following the first exposure to isocyanates. This period is highly variable: from several weeks, often less than two years but in 20% of cases, 10 years or more. Exposure to higher concentrations from spills may increase the risk of sensitisation. Once sensitisation has occurred, then subsequent exposure to airborne concentrations well below the exposure standard can cause asthmatic reactions like chest tightness, wheezing and shortness of breath, and increases in the background level of airway responsiveness. Exposure of sensitised workers may initiate reduction in respiratory capacity immediately on exposure, some hours later or both. Some workers become extremely sensitive to isocyanates and the high likelihood of chronic work-related asthma, which depends on duration of symptoms prior to cessation of exposure, places a high priority on primary prevention of sensitisation.

A rare consequence of chronic isocyanate exposure is hypersensitivity pneumonitis, an inflammatory reaction in terminal airways. Symptoms include shortness of breath, malaise (a general feeling of being unwell, discomfort or uneasiness) and fever occurring several hours after work with isocyanates.

Adverse health effects resulting from exposure to isocyanates normally arise during the ordinary working period, soon after contact occurs. Occasionally, symptoms do not appear for several hours following exposure. Because of this, symptoms are often not correlated with workplace exposure. It is important workers are informed of the potential for the delayed onset of health effects caused by isocyanate exposure. Workers should be told to report adverse health effects which they think may be related to isocyanate exposure so the root-cause can be investigated.

People with a history of asthma, atopic (allergic) conditions, hay fever, recurrent acute bronchitis, interstitial pulmonary fibrosis, pulmonary tuberculosis, occupational chest disease or impaired lung function are at greater risk of adverse health effects and should be warned against risk of exposure to isocyanates. Current evidence suggests workers with a history of developing hypersensitive allergic reactions or asthma does not preclude them working with isocyanates. However, exposure to isocyanates is likely to cause respiratory irritation and may aggravate pre-existing asthma.

GHS CARCINOGEN CLASSIFICATIONS

The following isocyanates are classified as Carcinogenicity Category 2 (Suspected of causing cancer):

- 4,4'-Methylene diphenyl diisocyanate
- 2,2'-Methylene diphenyl diisocyanate
- o-(p-Isocyanatobenzyl)phenyl isocyanate
- Methylene diphenyl diisocyanate (MDI)
- Toluene-2,4-diisocyanate
- Toluene-2,6-diisocyanate
- Toluene diisocyanate (TDI)

5.10 Lead (inorganic)

WORK ACTIVITIES THAT MAY REPRESENT A HIGH RISK EXPOSURE (LEAD PROCESSES)

It is a requirement of the regulations a PCBU determines whether a job is a lead risk job requiring health monitoring.

The following lead processes may involve significant exposures to lead:

- work that exposes a person to lead dust or lead fumes arising from the manufacture or handling of dry lead compounds
- work in connection with the manufacture, assembly, handling or repair of, or parts of, batteries containing lead that involves the manipulation of dry lead compounds, or pasting or casting lead
- breaking up or dismantling batteries containing lead, or sorting, packing and handling plates or other parts containing lead that are removed or recovered from the batteries
- spraying molten lead metal or alloys containing more than five per cent by weight of lead metal
- melting or casting lead alloys containing more than five per cent by weight of lead metal in which the temperature of the molten material exceeds 450°C
- recovering lead from its ores, oxides or other compounds by thermal reduction process
- dry machine grinding, discing, buffing or cutting by power tools alloys containing more than five per cent by weight of lead metal
- machine sanding or buffing surfaces coated with paint containing more than one per cent by dry weight of lead
- a process by which electric arc, oxyacetylene, oxy gas, plasma arc or a flame is applied for welding, cutting or cleaning, to the surface of metal coated with lead or paint containing more than one per cent by dry weight of lead metal
- radiator repairs that may cause exposure to lead dust or lead fumes
- fire assays if lead, lead compounds or lead alloys are used
- hand grinding and finishing lead or alloys containing more than 50 per cent by dry weight of lead
- spray painting with lead paint containing more than one per cent by dry weight of lead
- melting lead metal or alloys containing more than 50 per cent by weight of lead metal if the exposed surface area of the molten material exceeds 0.1 square metre and the temperature of the molten material does not exceed 450°C
- using a power tool, including abrasive blasting and high pressure water jets, to remove a surface coated with paint containing more than one per cent by dry weight of lead and handling waste containing lead resulting from the removal
- a process that exposes a person to lead dust or lead fumes arising from manufacturing or testing detonators or other explosives that contain lead
- a process that exposes a person to lead dust or lead fumes arising from firing weapons at an indoor firing range

5. INFORMATION ON SCHEDULED CHEMICALS AND ASBESTOS

- foundry processes involving:
 - melting or casting lead alloys containing more than one per cent by weight of lead metal in which the temperature of the molten material exceeds 450°C
 - dry machine grinding, discing, buffing or cutting by power tools lead alloys containing more than one per cent by weight of lead metal, and
- a process decided by the regulator to be a lead process under regulation 393.

ROUTE OF ENTRY INTO THE BODY

Inorganic lead uptake occurs as a result of ingestion or inhalation of inorganic lead particles. Not only are particulates in air as dusts and fumes significant sources of exposure in the workplace, but also eating and smoking with contaminated hands due to poor personal hygiene.

The respiratory tract provides the most efficient route of absorption while gastrointestinal absorption is relatively poor in adults. Absorption of inhaled lead is affected by various factors including personal characteristics, physical activity, particle size and solubility of the airborne lead.

ACUTE EFFECTS

In adults, high-dose exposure can lead to anaemia, abdominal colic (intense cramping of stomach muscles), peripheral neuropathy (damage to peripheral nervous system), central neuropathy (damage to central nervous system) with toxic encephalopathy (progressive degeneration of certain parts of the brain), nephropathy (kidney disease) and sterility. Tremors, stupor, seizures which are difficult to control, coma or death may result from very severe acute poisoning.

CHRONIC EFFECTS

High, chronic workplace exposure to lead damages the peripheral nervous system (nerves of the arms and legs), resulting in local paralysis, or 'lead palsy'. Workers with lower levels of exposure may experience fatigue, irritability, depression, insomnia, headaches and subtle evidence of intellectual decline.

Exposure to inorganic lead may also damage the formation and functioning of red blood cells. Anaemia is one of the most characteristic symptoms of high and prolonged exposure. Low to moderate exposure may result in cardiovascular effects, including increased blood pressure and heart problems.

Other chronic effects include kidney damage or failure, discomfort in the stomach, nausea, anorexia, weight loss and dyspepsia (indigestion or upset stomach). Higher levels may cause severe abdominal cramping and constipation. Chronic exposure to lead can also result in reproductive effects including reduced sperm counts, decreased sperm mobility and viability, and more frequent spontaneous abortions and still births.

When inorganic lead enters the body it does not undergo biological transformation. Lead is a cumulative poison which means if more lead is being absorbed by the body than it is able to excrete, the amount stored in the body will increase over time.

Pregnant or breastfeeding women and the foetus or young child are at high risk from lead. Pregnant or breastfeeding women and people under the age of 16 should be considered for exclusion from lead-risk work to eliminate exposure. This may be recommended by the registered medical practitioner.

5. INFORMATION ON SCHEDULED CHEMICALS AND ASBESTOS

GHS CARCINOGEN AND REPRODUCTIVE TOXICANT CLASSIFICATIONS

The following are examples of lead chemicals with carcinogen and reproductive toxicant classifications:

- Lead hexafluorosilicate: Repr. 1A
- Silicic acid, lead nickel salt: Carc. 1A (May cause cancer by inhalation), Repr. 1A
- Lead compounds with the exception of those specified elsewhere in Annex VI: Repr. 1A
- Lead diazide: Repr. 1A
- Lead diazide, [\geq 20% phlegmatiser]: Repr. 1A
- Lead chromate: Carc. 1B, Repr. 1A
- Lead di(acetate): Repr. 1A
- Trilead bis(orthophosphate): Repr. 1A
- Lead acetate, basic: Carc. 2, Repr. 1A
- Lead(II) methanesulphonate: Repr. 1A
- Lead sulfochromate yellow: Carc. 1B, Repr. 1A
- Lead chromate molybdate sulfate red: Carc. 1B, Repr. 1A
- Lead hydrogen arsenate: Carc. 1A (May cause cancer), Repr. 1A.

KEY

Abbreviation	Meaning	Hazard statement
Carc. 1A	Carcinogenicity Category 1A	May cause cancer
Carc. 1B	Carcinogenicity Category 1B	May cause cancer
Carc. 2	Carcinogenicity Category 2	Suspected of causing cancer
Repr. 1A	Reproductive Toxicity Category 1A	May damage the unborn child, suspected of damaging fertility

5.11 Mercury (inorganic)

WORK ACTIVITIES THAT MAY REPRESENT A HIGH RISK EXPOSURE

Mercury exists in three forms: liquid and vapour states (Hg^0) and inorganic mercury salts (Hg^{1+} and Hg^{2+}).

Examples of work activities involving inorganic mercury and its compounds which require special attention when assessing exposure include:

- manufacture of amalgams, for example tin amalgam, amalgam of gold, copper and zinc used in dentistry for filling teeth, amalgamated zinc used in electric batteries and sodium amalgam used in the laboratory in conjunction with water as a reducing agent
- dental work involving mercury
- manufacture of pigments and antifouling paints (mercuric oxide) and vermilion (mercuric sulphide) in the paint and colour industry
- extraction of gold and silver from roasted pyrites (mercuric sulphate)
- extraction of gold from tailings

5. INFORMATION ON SCHEDULED CHEMICALS AND ASBESTOS

- laboratory work with mercury in closed or confined spaces
- the use of mercury-containing fungicides
- exploration/production, refining and processing of natural gas
- the use of fluorescent lamps and electrical meters.

ROUTE OF ENTRY INTO THE BODY

The absorption and toxicity of inorganic mercury in the body depends on its chemical and physical form. Generally, liquid elemental mercury (Hg^0) is poorly absorbed, less than one per cent, through the intestinal tract and skin. About 75 to 80 per cent of inhaled mercury vapour is absorbed across alveolar membranes into the bloodstream. Absorbed mercury vapour readily crosses the blood-brain barrier and the placenta.

Small amounts, less than 10 per cent, of mercurous (Hg^{1+}) and mercuric (Hg^{2+}) salts are absorbed following ingestion. Absorption through the skin of ionic mercury salts can cause toxicity. Generally, mercuric salts are more soluble and more toxic than mercurous salts.

ACUTE EFFECTS

In acute poisoning, the respiratory system is affected by inhaled mercury vapour and the gastrointestinal system is affected by ingested mercury salts.

Acute inhalation exposure to inorganic mercury vapour may rapidly produce cough, chest pain, shortness of breath, fever, nausea, vomiting, diarrhoea and a metallic taste in the mouth. Stomatitis (inflammation of the mucous linings in the mouth), colitis, nephrotic syndrome and salivation may occur. High concentrations cause corrosive bronchitis and interstitial pneumonitis (inflammation of the lung tissue). The uptake of mercury vapour into the central nervous system produces tremor and increased excitability. High exposures have resulted in death.

Acute mercurial poisoning is usually the outcome of ingestion. The acute lethal dose of most mercury salts is one to four grams for an adult. The gastrointestinal tract and kidney are affected by ingestion of mercury salts. Ingestion of corrosive mercury salts results in immediate gastroenteritis. Mercurial stomatitis characterised by glossitis (inflammations and discolouration of the tongue) and ulcerative gingivitis (swelling of the gums) may appear within 24 to 36 hours.

In some people, metallic mercury has been shown to cause allergic reactions like skin rashes.

CHRONIC EFFECTS

The primary organ system affected by chronic exposure to elemental mercury is the nervous system, and the kidney is the primary organ affected by chronic exposure to mercury salts. In chronic poisoning resulting from exposure to elemental mercury or the dust of inorganic mercurial compounds, early symptoms may include nausea, frequent headaches, tiredness and chronic diarrhoea. The characteristic features are stomatitis, muscular tremors and psychotic disturbances.

Effects on the mouth may vary from a mere metallic taste to excessive salivation, bleeding of the gums, ulceration and loosening of the teeth. Muscular tremors appear early, often starting in the fingers and spreading to the tongue, lips, eyes and lower limbs. These become apparent when the individual goes to perform some defined action like writing, which may become so disordered by the tremor it is illegible. The neurological disturbance, mercurial erethism or erethism mercurialis, manifests itself in abnormal shyness and loss of confidence, coupled with irritability, vague fears and depression. In advanced cases, there may be loss of memory, psychotic changes like hallucination, or intellectual deterioration.

5. INFORMATION ON SCHEDULED CHEMICALS AND ASBESTOS

Phenyl mercury acetate has a strong corrosive action and will cause local blistering of the skin. Mercury fulminate is particularly prone to cause a vesicular (sac-like lesions) dermatitis, especially affecting the fingers, and irritation of the eyes and eyelids. Workers exposed to mercury vapour may be found to have a discolouration of the lens of the eye, which is indicative of mercury exposure rather than of intoxication.

GHS GERM CELL MUTAGEN AND REPRODUCTIVE TOXICANT CLASSIFICATIONS

Mercury is classified as Reproductive Toxicity Category 1B (May damage the unborn child). Mercury dichloride is classified as Germ Cell Mutagenicity Category 2 (Suspected of causing genetic defects) and Reproductive Toxicity Category 2 (Suspected of damaging fertility).

5.12 4,4'-Methylene bis(2-chloroaniline) (MOCA)

WORK ACTIVITIES THAT MAY REPRESENT A HIGH RISK EXPOSURE

Examples of work activities involving MOCA which require special attention when assessing exposure include:

- dispensing MOCA powder
- processes where spattering of MOCA in the dry or molten state occurs
- manual moulding of semi-set polyurethane products.

ROUTE OF ENTRY INTO THE BODY

Skin absorption is likely to be the main route of entry in to the body and is associated with the following factors:

- poor housekeeping, for example visible MOCA granules on floors and work benches
- poor personal hygiene practices
- inadequate personal protection.

ACUTE EFFECTS

Exposure to MOCA can cause methaemoglobinaemia (high levels of methaemoglobin in the blood) which causes hypoxia (oxygen deprivation) and cyanosis (blue colouration of skin). MOCA can also have an effect on the kidneys causing haematuria (blood in the urine) and proteinuria (high protein levels in urine). MOCA is also a respiratory tract irritant and an eye irritant.

CHRONIC EFFECTS

Long term exposure to MOCA has been associated with cancer of the bladder.

GHS CARCINOGEN CLASSIFICATION

MOCA is classified as Carcinogenicity Category 1B (May cause cancer).

5.13 Organophosphate pesticides

WORK ACTIVITIES THAT MAY REPRESENT A HIGH RISK EXPOSURE

Organophosphate insecticides are widely used on a large variety of crops and are usually dispersed as an aerosol consisting of the pesticide adsorbed on an inert fine particle dissolved in a hydrocarbon solvent. They have also found widespread use around the home and garden to control insects.

5. INFORMATION ON SCHEDULED CHEMICALS AND ASBESTOS

Examples of work activities involving organophosphate pesticides (OP) which require special attention when assessing exposure include:

- pest control operators who use OP everyday in their work
- manufacture and packaging
- transport, storage and distribution
- handling used containers, for example scrap recovery
- agricultural and horticultural activities like mixing, loading and application where direct handling of the chemical occurs, see Table 1 Definition of pattern of use and action required
- veterinary activities like cattle and sheep dipping – see Table 1 Definition of pattern of use and action required
- seasonal field workers exposed to pesticide residues – see Table 1 Definition of pattern of use and action required
- laboratory workers undertaking research on OP.

RISK MANAGEMENT

Because of the seasonal nature of the use of OP in the rural sector, special attention should be paid to the risk management process. A list of work tasks involving the use of OP should be developed and the pattern of use and potential for exposure of workers should be considered. The patterns of use will determine the need for and timing of health monitoring. Once this assessment is completed, the need for health monitoring for each operation should be considered, noting the definition of pattern of use and action required – see Table 1. Proposed health monitoring should be recorded.

All use of OP should be recorded.

Special attention should be given to acute exposures, including spills.

ROUTE OF ENTRY INTO THE BODY

For most OP, skin exposure and subsequent absorption through intact skin represents the most important route of entry in the workplace. It should be noted that many organophosphorous pesticides oxidise to a more active form following the application process thus representing an increased hazard to workers who may come into skin contact with sprayed surfaces.

The oral route of entry is important in accidental ingestion and deliberate ingestion. Work-related accidental ingestion may occur as a result of poor work practices and lack of personal hygiene. If swallowed, OP are rapidly absorbed from the stomach.

The inhalation route is generally less important. Inhalation of OP depends on the volatility of the compound, on the type of formulation and on the technique of application, for example spraying.

OP are also absorbed through mucous membranes and eyes. If the concentrate of the more toxic OP is splashed into the eye, absorption may be very rapid.

5. INFORMATION ON SCHEDULED CHEMICALS AND ASBESTOS

ACUTE EFFECTS

The first symptoms of organophosphate poisoning can occur within minutes of exposure to a concentrate or a highly toxic organophosphate pesticide. A common situation is for symptoms to occur an hour or so after inadvertent skin exposure to a working solution of the insecticide.

Local effects at the site of exposure may occur without symptoms and signs of systemic absorption (absorption that occurs in other parts of the body from where exposure occurred). A splash in the eye may cause blurred vision. Inhalation may cause bronchoconstriction resulting in coughing, wheezing and shortness of breath, and produce an excess of respiratory tract secretions. This may result in a feeling of chest tightness and a watery nasal discharge. Splashes on the skin may cause localised sweating and fasciculations (involuntary muscle twitching).

Symptoms and signs usually reach their maximum severity 24 to 48 hours after onset and usually regress over the next one to six days. In the case of massive exposure, death usually occurs within 24 hours. If splashed in the eye or swallowed, absorption may be rapid.

Another short-term effect of organophosphates is the intermediate syndrome. This is characterised by transient muscle weakness of the limbs, neck and respiratory muscles, which begins one to four days after a poisoning incident and may continue for up to several weeks.

CHRONIC EFFECTS

Symptoms of poisoning usually do not occur until cholinesterase enzyme activity has been reduced to between 60 to 25 per cent of an individual's baseline. Chronic low level exposures may lead to cumulative effects. Thus workers continually exposed may be at high risk even at low level exposures. Once exposure has ceased, serum cholinesterase regenerates, but depending upon the severity of poisoning, may take several days and occasionally longer to return to normal, particularly if treatment is not given.

Continual exposure may cause persistent anorexia, weakness and malaise (a general feeling of being unwell, discomfort or uneasiness). Certain neurobehavioural effects may be seen. In delayed polyneuropathy where there is damage to nerves of the peripheral nervous system, the interval between acute exposure and the onset of neuropathy may be up to four weeks. Symptoms include tingling and burning sensations in the hands and feet followed by weakness in the lower limbs and ataxia (lack of coordination). In severe cases the upper limbs may be affected.

Many OP cause primary irritant dermatitis; only a few are known to cause allergic contact dermatitis, for example parathion and malathion.

GHS CARCINOGEN, GERM CELL MUTAGEN AND REPRODUCTIVE TOXICANT CLASSIFICATIONS

While most OP are not classified as carcinogens, germ cell mutagens or reproductive toxicants, you should check the relevant safety data sheet for classification information.

5. INFORMATION ON SCHEDULED CHEMICALS AND ASBESTOS

Table 1 Definition of pattern of use and action required

Definition of pattern of use	Action required
Baseline	<ul style="list-style-type: none"> ■ Baseline measurement should be carried out – two measurements are desirable, at a time when there has been at least four weeks without exposure.
<p>Very occasional use</p> <p>If use of organophosphate pesticides is only half a day every month or less, then this is <i>very occasional use</i>.</p>	<ul style="list-style-type: none"> ■ Use should be recorded. ■ No test is needed unless the worker has symptoms which could be related to organophosphate pesticides during or after use, or there has been an 'exposure incident' leading to symptoms.
<p>Intermittent use</p> <p>If use of organophosphate pesticides is for two to three days at a time, all day with gaps of time of a month or more between use, then this is <i>intermittent use</i>.</p>	<ul style="list-style-type: none"> ■ Use should be recorded. ■ Test during the period of peak exposure/use. Testing provides valuable information on the effectiveness of controls. ■ Controls must be updated if levels of exposure indicate high work-related exposure. ■ No further testing is needed unless the worker has symptoms which could be related to organophosphate pesticides during or after use, there has been an 'exposure incident' leading to symptoms, or there is concern 'overexposure' may have occurred.
<p>Seasonal use</p> <p>If use of organophosphate pesticides is say four days a week, and extends over a long season then, this is <i>seasonal use</i>.</p>	<ul style="list-style-type: none"> ■ Use should be recorded. ■ For heavy or seasonal use, testing should occur during the period of peak exposure/use. Workers exposed should be tested at the end of the work shift on the last day of a work week, early in the season, once work practices have settled, in order to check the effectiveness of work practices and controls. Adjustments to controls can be made if necessary. Workers must be advised of their results, that is percentage depression of cholinesterase from their baseline values. ■ The timing of further tests should be based on the nature of the work and the previous test results. A worker having greater than 20% depression from baseline values should be retested at an early stage. ■ No further test is needed unless the person has symptoms which could be related to organophosphate pesticides during or after use, there has been an 'exposure incident' leading to symptoms, or there is concern 'overexposure' may have occurred.

5.14 Pentachlorophenol (PCP)

WORK ACTIVITIES THAT MAY REPRESENT A HIGH RISK EXPOSURE

PCP was once used as a preservative against timber-destroying fungi, sapstain moulds and some timber-boring insects and termites. Chloro-dibenzodioxins and dibenzofurans are known contaminants of PCP. Pentachlorophenol (PCP) is not currently approved for use as an agricultural or veterinary chemical in Australia.

Examples of work activities involving PCP which require special attention when assessing exposure include:

- workers who handle or breathe air near wood that has been preserved with PCP.

ROUTE OF ENTRY INTO THE BODY

The routes of PCP entry into the body are through inhalation and skin absorption. Accidental ingestion is unlikely unless poor hygiene and work practices allow it.

ACUTE EFFECTS

The most important effect of PCP inhalation is acute poisoning centring on the circulatory system. Physiological injury is mainly muscular with heart failure.

Irritation of the eyes, mucous membranes and upper respiratory tract has been reported at airborne dust or mist concentrations of 1.0 mg/m³. Airborne concentrations as low as 0.3 mg/m³ may cause some irritation of the upper respiratory tract. Direct skin contact can lead to skin irritation and systemic toxicity (toxicity which occurs in other parts of the body from where exposure occurred). A 10 per cent solution may cause irritation after a single brief exposure, whereas prolonged or repeated contact with a one per cent solution would be required to produce the same result. A solution of 0.1 per cent concentration may lead to adverse systemic effects.

Systemic effects from acute PCP poisoning include weakness, loss of appetite, gastrointestinal disturbance, weight loss, nausea, vomiting, chest pain, excessive sweating, fever, headaches and dizziness, inflammation of the conjunctiva, corneal opacity, increased heart rate, increased breathing rate, respiratory distress and enlargement of the liver. In fatal cases, body temperature is often very high and death may occur as early as three hours after the onset of symptoms. The risk of serious consequences is greater in hot weather. People with significantly impaired liver or kidney function may be more susceptible to poisoning from this substance.

CHRONIC EFFECTS

Chronic exposure is associated with inflammation of the upper respiratory tract and bronchitis, blood effects like aplastic anaemia (bone marrow does not produce enough new blood cells), effects on the kidney and liver, effects on the immune system, irritation of the eyes, nose and skin, increased prevalence of conjunctivitis, chronic sinusitis, polyneuritis (inflammation of the peripheral nervous system) and dermatitis.

Bronchitis has been reported at airborne dust or mist concentrations of 1.0 mg/m³. Repeated exposure to PCP may cause an acne-like rash and liver and kidney damage. Deaths have occurred among workers involved in crop and herbicidal spray operations where a 1.5 to 2 per cent PCP solution was used without adequate control measures.

5. INFORMATION ON SCHEDULED CHEMICALS AND ASBESTOS

Aspirin, when absorbed in large amounts, may enhance the risk of toxicity for PCP-exposed workers. People taking medications like phenytoin, warfarin, furosemide, ethacrynic acid, naproxen and ibuprofen on a long-term basis may be at increased risk of PCP-induced toxicity.

GHS CARCINOGEN CLASSIFICATION

Pentachlorophenol is classified as Carcinogenicity Category 2 (Suspected of causing cancer).

5.15 Polycyclic aromatic hydrocarbons (PAH)

PAH are organic compounds consisting of two or more benzene molecules fused together and contain only carbon and hydrogen. They are formed during the combustion of organic material.

WORK ACTIVITIES THAT MAY REPRESENT A HIGH RISK EXPOSURE

Examples of work activities involving PAH exposure which require special attention when assessing exposure include:

- coke plant work
- aluminium primary plants
- tar roofing
- asphalt road surfacing
- diesel emissions.

ROUTE OF ENTRY INTO THE BODY

The routes of PAH entry into the body are through inhalation, ingestion and skin absorption.

TARGET ORGAN/EFFECT

There is evidence workers exposed to high airborne levels of some PAHs show increased risk of lung, kidney, bladder, gastrointestinal and skin cancers.

Polycyclic aromatic hydrocarbons are known to cause photosensitisation. Photosensitivity is an abnormally high reactivity in the skin or eyes to ultraviolet radiation or natural sunlight. It may be induced by ingestion, inhalation or skin contact with certain substances known as photosensitisers. Symptoms will vary with the amount of ultraviolet radiation, type and amount of photosensitiser, skin type, and age and gender of the person exposed.

Photosensitisation of the skin and eyes can be caused by exposure to specific industrial chemicals like creosote. The skin can be affected by direct contact or by inhalation. The eyes can be affected by volatile fumes. In certain occupations, the risk from exposure to particular photosensitising chemicals and solar ultraviolet radiation is severe. For example, exposure to tar and sunlight can cause precancerous and cancerous skin lesions. Exposure to coal tar fumes can cause simultaneous inflammation of the conjunctiva and cornea.

CHRONIC EFFECTS

Toxic effects on the skin from PAH are enhanced by exposure to ultraviolet light. The skin is prone to erythema (reddening), photosensitivity and skin lesions on sun exposed areas with progression to skin cancer. PAH are irritating to the eyes and can cause photosensitivity. Cough, chronic bronchitis and haematuria (blood in the urine) have also been noted.

5. INFORMATION ON SCHEDULED CHEMICALS AND ASBESTOS

GHS CARCINOGEN, GERM CELL MUTAGEN AND REPRODUCTIVE TOXICANT CLASSIFICATIONS

Benzo[a]pyrene, benz[a]anthracene and dibenz[a,h]anthracene are classified as Carcinogenicity Category 1B (May cause cancer). Benzo[a]pyrene is also classified as Germ Cell Mutagenicity Category 1B (May cause genetic defects) and Reproductive Toxicity Category 1B (May damage fertility, may damage the unborn child).

5.16 Thallium

WORK ACTIVITIES THAT MAY REPRESENT A HIGH RISK EXPOSURE

Examples of work activities involving thallium and its compounds which require special attention when assessing exposure include:

- laboratory analysis where thallium malonate-formate (Clerici's reagent) is used for mineralogic analysis of rocks, ores and sand, and separation of diamonds
- production of pigments, luminous paints, artificial gems, coloured glass, and special optical glasses for lenses and prisms, electronic devices and switches
- smelters, power plants, cement factories, with a risk of exposure from cleaning fossil fuel furnaces or flues and metal machining.

Special attention should also be given to acute exposures, including reagent spills, that may occur in the above processes.

POTENTIAL HEALTH EFFECTS FOLLOWING EXPOSURE TO THALLIUM

Pure thallium is odourless and tasteless and extremely toxic.

The relative toxicity of a thallium compound depends on its water solubility. The more water soluble forms (sulphate, acetate, malonate and carbonate) are more toxic than the less water soluble forms (sulphide and iodide).

ROUTE OF ENTRY INTO THE BODY

The routes of thallium entry into the body are through inhalation, ingestion and skin absorption. Thallium and thallium salts are rapidly absorbed by intact skin, by inhalation and through the mucous membrane of the gastro-intestinal tract.

ACUTE EFFECTS

Thallium and thallium compounds are extremely toxic. For adults, doses which have proved lethal vary between 6 and 40 mg/kg.

Following ingestion of a single toxic dose, symptoms of acute poisoning may occur within 12 hours to two days and include severe abdominal pain, vomiting, diarrhoea, gastrointestinal bleeding, tremors, delirium, convulsions, paralysis and coma leading to death in one to two days. The acute reaction may subside to be followed in approximately 10 days by the development of polyneuritis (inflammation of peripheral nervous system), psychosis, delirium, optic nerve damage and blindness, increased heart rate and blood pressure, skin eruptions and hepatic or renal injury. Hair loss occurs within 15 to 20 days.

5. INFORMATION ON SCHEDULED CHEMICALS AND ASBESTOS

CHRONIC EFFECTS

Thallium may act as a cumulative poison with chronic intoxications and a sudden release from tissue stores may lead to acute toxic symptoms. Long-term low-level exposure may give rise to a mild clinical set of symptoms including polyneuropathy, causing damage to the peripheral nervous system, and partial hair loss. At a higher exposure level, fatigue, anorexia, leg joint pain, optic nerve damage with visual disturbances, and increasing nerve damage may occur.

GHS CARCINOGEN, GERM CELL MUTAGEN AND REPRODUCTIVE TOXICANT CLASSIFICATIONS

The European Union has determined that thallium and thallium compounds are not classified as carcinogens, germ cell mutagens or reproductive toxicants.

5.17 Vinyl chloride

WORK ACTIVITIES THAT MAY REPRESENT A HIGH RISK OF EXPOSURE

Examples of work activities involving vinyl chloride which require special attention when assessing exposure include production of polyvinyl chloride (PVC), in particular, during cleaning of autoclaves.

ROUTE OF ENTRY INTO THE BODY

The primary routes of vinyl chloride entry into the body are through inhalation and ingestion.

ACUTE EFFECTS

The central nervous system (CNS) is the primary target of vinyl chloride acute toxicity. There may be a latent period of hours to days between exposure and symptom onset. Symptoms include euphoria, dizziness, ataxia (lack of coordination), fatigue, drowsiness, headache and loss of consciousness. With inhalational exposure, signs and symptoms increase in severity over a range of 8000 to 20 000 ppm in air. Exposure to higher concentrations can cause death. Sub-lethal CNS effects resolve quickly when the victim is removed from further exposure.

Vinyl chloride gas inhalation can cause mild respiratory tract irritation, wheezing and chemical bronchitis. These effects are transient and resolve quickly following removal from exposure.

CHRONIC EFFECTS

Chronic exposure to lower levels of vinyl chloride of around 100-1000 ppm has been associated with a range of symptoms collectively termed 'vinyl chloride disease' which includes Raynaud's syndrome (problems with the blood vessels which supply the skin), scleroderma (skin and connective tissue tighten and harden) and acro-osteolysis (loss of bone and tissue in the fingers and toes).

Chronic exposure to vinyl chloride exposure may also cause liver and spleen fibrosis, portal hypertension (high blood pressure in portal venous system) and cirrhosis of the liver. Other effects of chronic exposure include decreased ability to move or feel and immune system phenomena like purpura (red or purple discolouration on the skin) and thrombocytopenia (decreased number of platelets in the blood).

GHS CARCINOGEN CLASSIFICATION

Vinyl chloride is classified as Carcinogenicity Category 1A (May cause cancer).

APPENDIX A - HAZARDOUS CHEMICALS REQUIRING HEALTH MONITORING

The information in this Appendix is taken from Schedule 14 of the WHS Regulations and Regulation 436 (asbestos).

Hazardous chemicals requiring health monitoring		
	Hazardous chemical	Type of health monitoring
1	Acrylonitrile	Demographic, medical and occupational history Records of personal exposure Physical examination
2	Arsenic (inorganic)	Demographic, medical and occupational history Records of personal exposure Physical examination with emphasis on the peripheral nervous system and skin Urinary inorganic arsenic
3	Asbestos	Demographic, medical and occupational history Records of personal exposure Physical examination
4	Benzene	Demographic, medical and occupational history Records of personal exposure Physical examination Baseline blood sample for haematological profile
5	Cadmium	Demographic, medical and occupational history Records of personal exposure Physical examination with emphasis on the respiratory system Standard respiratory questionnaire to be completed Standard respiratory function tests including for example, FEV ₁ , FVC and FEV ₁ /FVC Urinary cadmium and β 2-microglobulin Health advice, including counselling on the effect of smoking on cadmium exposure
6	Chromium (inorganic)	Demographic, medical and occupational history Physical examination with emphasis on the respiratory system and skin Weekly skin inspection of hands and forearms by a competent person

APPENDIX A - HAZARDOUS CHEMICALS REQUIRING HEALTH MONITORING

Hazardous chemicals requiring health monitoring		
	Hazardous chemical	Type of health monitoring
7	Creosote	Demographic, medical and occupational history Health advice, including recognition of photosensitivity and skin changes Physical examination with emphasis on the neurological system and skin, noting any abnormal lesions and evidence of skin sensitisation Records of personal exposure, including photosensitivity
8	Crystalline silica	Demographic, medical and occupational history Records of personal exposure Completion of a standardised respiratory questionnaire Standardised respiratory function test, for example, FEV ₁ , FVC and FEV ₁ /FVC Chest X-ray full size PA view
9	Isocyanates	Demographic, medical and occupational history Completion of a standardised respiratory questionnaire Physical examination of the respiratory system and skin Standardised respiratory function tests, FEV ₁ , FVC and FEV ₁ /FVC
10	Lead (inorganic)	Demographic, medical and occupational history Physical examination Biological monitoring
11	Mercury (inorganic)	Demographic, medical and occupational history Physical examination with emphasis on dermatological, gastrointestinal, neurological and renal systems Urinary inorganic mercury
12	4,4'-Methylene bis(2-chloroaniline) (MOCA)	Demographic, medical and occupational history Physical examination Urinary total MOCA Dipstick analysis of urine for haematuria Urine cytology

APPENDIX A - HAZARDOUS CHEMICALS REQUIRING HEALTH MONITORING

Hazardous chemicals requiring health monitoring		
	Hazardous chemical	Type of health monitoring
13	Organophosphate pesticides	Demographic, medical and occupational history including pattern of use Physical examination Baseline estimation of red cell and plasma cholinesterase activity levels by the Ellman or equivalent method Estimation of red cell and plasma cholinesterase activity towards the end of the working day on which organophosphate pesticides have been used
14	Pentachlorophenol (PCP)	Demographic, medical and occupational history Records of personal exposure Physical examination with emphasis on the skin, noting any abnormal lesions or effects of irritancy Urinary total pentachlorophenol Dipstick urinalysis for haematuria and proteinuria
15	Polycyclic aromatic hydrocarbons (PAH)	Demographic, medical and occupational history Physical examination Records of personal exposure, including photosensitivity Health advice, including recognition of photosensitivity and skin changes
16	Thallium	Demographic, medical and occupational history Physical examination Urinary thallium
17	Vinyl chloride	Demographic, medical and occupational history Physical examination Records of personal exposure

APPENDIX B – ADDITIONAL EXAMPLES OF CHEMICALS TO CONSIDER FOR HEALTH MONITORING

Health monitoring requirements are not limited to chemicals in Schedule 14 or asbestos and health monitoring must be carried out where there is significant risk of exposure to any hazardous chemical where a valid technique is available to detect the effect on a worker's health. Below are some examples of hazardous chemicals and testing methods which are not listed in schedule 14 of the WHS Regulations where you may wish to consider implementing a health monitoring program for your workers.

SOME HAZARDOUS CHEMICALS WHICH MAY REQUIRE HEALTH MONITORING		
	Hazardous chemical	Type of health monitoring
1	Antimony	Demographic, medical and occupational history Records of personal exposure Physical examination with emphasis on the respiratory system and skin Urinary antimony level
2	Beryllium	Demographic, medical and occupational history Records of personal exposure Physical examination with emphasis on respiratory and dermatological systems Urinary beryllium
3	Carbon disulphide	Demographic, medical and occupational history Physical examination with emphasis on the respiratory system and skin Urinary 2-thiothiazolidine-4-carboxylic acid level
4	Cobalt	Demographic, medical and occupational history Physical examination with emphasis on respiratory systems and skin Urinary cobalt level
5	Cyclophosphamide	Demographic, medical and occupational history Urinary cyclophosphamide
6	Ethyl benzene	Demographic, medical and occupational history Records of personal exposure Physical examination Baseline blood sample for haematological profile Urinary mandelic acid
7	Nickel	Demographic, medical and occupational history Physical examination with emphasis on dermatological and respiratory systems Urinary nickel

APPENDIX B – ADDITIONAL EXAMPLES OF CHEMICALS TO CONSIDER FOR HEALTH MONITORING

SOME HAZARDOUS CHEMICALS WHICH MAY REQUIRE HEALTH MONITORING		
	Hazardous chemical	Type of health monitoring
8	Styrene	Demographic, medical and occupational history Records of personal exposure Physical examination Baseline blood sample for haematological profile Urinary mandelic acid
9	Toluene	Demographic, medical and occupational history Records of personal exposure Physical examination Baseline blood sample for haematological profile Urinary hippuric acid or o-cresol or s-toluymercapturic acid
10	Xylene	Demographic, medical and occupational history Records of personal exposure Physical examination Baseline blood sample for haematological profile Urinary toluric acid

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APPENDIX C - CHECKLIST FOR PROVIDING HEALTH MONITORING

- Inform the worker or potential worker of health monitoring requirements before the worker carries out work with the hazardous chemical. Consult with the worker.
- Provide the worker with a copy of *Health Monitoring for Exposure to Hazardous Chemicals: Guide for Workers*.
- Engage a registered medical practitioner with experience in health monitoring to supervise the health monitoring program and ensure they conduct or supervise the health monitoring program.
- Provide the registered medical practitioner with relevant information described in section 3.4 of this guide.
- Decide the type of health monitoring to use according to the WHS Regulations in consultation with the registered medical practitioner.
- Obtain the health monitoring report, identify the record as a confidential record in relation to the worker and keep the record at least 30 years after the record is made or 40 years in the case of asbestos.
- Provide a copy of the health monitoring report to the worker.
- Provide a copy of the health monitoring report to all other PCBU's who have a duty to provide health monitoring for the worker.
- Provide a copy of the health monitoring report to the regulator if the report advises the worker is suffering a disease, injury or illness as a result of exposure or recommends you take remedial action.
- Take action where the health monitoring report has any of the following:
 - has test results indicating the worker has been exposed to the chemical and has an elevated level of the chemical or its metabolites in his or her body for that hazardous chemical
 - advises the worker is suffering a disease, injury or illness as a result of exposure
 - recommends you take remedial action
 - advises medical counselling is required.

