HAZARDOUS CHEMICALS REQUIRING HEALTH MONITORING

MARCH 2013

safe work australia

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Safe Work Australia is an Australian Government statutory agency established in 2009. Safe Work Australia consists of representatives of the Commonwealth, state and territory governments, the Australian Council of Trade Unions, the Australian Chamber of Commerce and Industry and the Australian Industry Group.

Safe Work Australia works with the Commonwealth, state and territory governments to improve work health and safety and workers' compensation arrangements. Safe Work Australia is a national policy body, not a regulator of work health and safety. The Commonwealth, states and territories have responsibility for regulating and enforcing work health and safety laws in their jurisdiction.

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WORKSAFE ACT
WorkCover New South Wales
Workplace Standards Tasmanian Government
NT WorkSafe
Government of South Australia SafeWork SA
WORKSAFE Western Australia
Queensland Government

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1. INTRODUCTION

HAZARDOUS CHEMICALS REQUIRING HEALTH MONITORING

This Guide is intended for use by medical practitioners carrying out or supervising a health monitoring program for workers who may be exposed to the following hazardous chemicals and asbestos. It should be read in conjunction with the *Health Monitoring for Exposure to Hazardous Chemicals: Guide for Medical Practitioners.*

This document provides information about the known hazards of each chemical, symptoms of exposure, medical tests that should be used during health monitoring, and information on when to recommend certain actions like removal from work.

It also includes examples of health monitoring reports that may be used by the medical practitioner. Other forms and formats are acceptable and may be used.

Classification information on each chemical's known carcinogenicity, germ cell mutagenicity and reproductive toxicity is also provided on an advisory basis where this information is known. Classification information is 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[1]. Annex VI includes lists of GHS classification information for certain substances or groups of substances. These classifications are legally binding within the European Union. Additional hazard classes and categories not mentioned in this document may also apply.

1 Source: http://esis.jrc.ec.europa.eu/index.php?PGM=cla.

ACRYLONITRILE

BASELINE HEALTH MONITORING BEFORE STARTING WORK IN AN ACRYLONITRILE PROCESS

1. **Collection of demographic data**
2. **Work history**
3. **Medical history**
4. **Physical examination**

A physical examination will be conducted with emphasis on the central nervous system (CNS), respiratory system and skin, only if work and medical history indicates this is necessary, for example the presence of symptoms.

DURING EXPOSURE TO AN ACRYLONITRILE PROCESS

5. **Medical examination**

Where workers are excessively exposed to acrylonitrile, for example following spills or loss of containment, are suspected of being excessively exposed to acrylonitrile, or have concerns about acrylonitrile exposure, for example where relevant symptoms are identified, the person conducting a business or undertaking should arrange an appointment with the registered medical practitioner.

AT TERMINATION OF WORK IN AN ACRYLONITRILE PROCESS

6. **Final medical examination**

A final medical examination will be conducted, with emphasis on CNS, respiratory system and skin.

SUPPLEMENTARY INFORMATION ON ACRYLONITRILE

7. **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 procedures where solvent is removed from newly-formed fibres.

Special attention should also be given to acute exposures that may occur in the above process.

8. Non-work sources

There are a number of potential sources of non-work-related exposure to acrylonitrile. These include:

- previously used as a fumigant
- acrylonitrile can be found in car exhaust
- present in cigarette smoke
- food may contain acrylonitrile as a result of migration from food containers.

Consumer exposure to acrylonitrile from skin contact with acrylic fibres and from ingestion of foods contaminated with residual acrylonitrile in packaging materials is estimated at a maximum of 2.2 and 33 ng/kg/day respectively [1].

9. Route of entry into the body

The primary route of acrylonitrile entry into the body is through inhalation, with an average respiratory retention of 52 per cent. Acrylonitrile can also be absorbed percutaneously in quantities sufficient to cause health effects.

10. Target organ/effect

Central nervous system – headache, dizziness, general weakness.

Liver – hepatocellular damage.

Skin – irritation, burns, blisters, sensitisation.

Respiratory tract – irritation.

Eyes – irritation.

Carcinogen – GHS Carcinogenicity Category 1B (May cause cancer), multiple sites.

11. 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 is a cellular asphyxiant with actions similar to cyanide, 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 and the formation of large blisters after a latent period of several hours.

12. Chronic effects

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

13. Carcinogenicity

Acrylonitrile has been shown to cause cancer in laboratory animals. Some studies of workers potentially exposed to acrylonitrile have demonstrated an increased incidence of cancer of the lung, gastrointestinal tract and prostate.

14. Carcinogen classification

Acrylonitrile is classified according to the GHS as Carcinogenicity Category 1B (May cause cancer).

REFERENCED DOCUMENTS

1. National Industrial Chemicals Notification and Assessment Scheme, *Acrylonitrile, Priority Existing Chemical Assessment Report No. 10*, Feb 2000.

FURTHER READING

Agency for Toxic Substances and Disease Registry, *Medical Management Guidelines for Acrylonitrile*. http://www.atsdr.cdc.gov/MMG/MMG.asp?id=443&tid=78

Australian Chemical Industry Council, *Code of Practice on the Safe Handling of Acrylonitrile,* Australian Chemical Industry Council, Melbourne, 1992.

International Agency for Research on Cancer, *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Volume 71: Re-evaluation of Some Organic Chemicals, Hydrazine and Hydrogen Peroxide*, International Agency for Research on Cancer, Lyon, 1999.

International Programme on Chemical Safety, *Environmental Health Criteria 28: Acrylonitrile*, International Programme on Chemical Safety, World Health Organization, Geneva, 1983.

Lauwerys RR, Hoet P, *Industrial Chemical Exposure Guidelines for Biological Monitoring*, 3rd Ed, Lewis Publishers, Boca Raton, 2001.

National Toxicology Program, *Acrylonitrile*, in 12th Report on Carcinogens, United States Department of Health and Human Services, Public Health Service, 2011.

World Health Organisation/International Program on Chemical Safety, *Concise International Chemical Assessment Document 39: Acrylonitrile*, WHO, Geneva, 2002.

This health monitoring report is a confidential health record and must not be disclosed to another person except in accordance with the Work Health and Safety Regulations or with the consent of the worker.

There are two sections. Complete both sections and all questions if applicable.

Section 1 is to be forwarded to the PCBU who has engaged your services.

Section 2 may contain confidential information which may not be relevant to the health monitoring program being carried out. This section should be retained by the medical practitioner. Information which is required to be given to the PCBU should be summarised in part 7 of section 1.

SECTION 1 – THIS SECTION TO BE RETURNED TO THE PCBU			
1. PERSON CONDUCTING A BUSINESS OR UNDERTAKING			
Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
2. OTHER BUSINESSES OR UNDERTAKINGS ENGAGING THE WORKER			
Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
3. WORKER		(✓) all relevant boxes	
Surname:	Given names:		
Date of birth: DD/MM/YYYY	Sex:	☐ Male	☐ Female
Address:			
Suburb:			Postcode:
Current Job:	Tel(H):		Mob:
Date started employment : DD/MM/YYYY			
4. EMPLOYMENT IN ACRYLONITRILE RISK WORK		(✓) all relevant boxes	
1. ☐ New to acrylonitrile work			
2. ☐ New worker but not new to acrylonitrile work			
3. ☐ Current worker continuing in acrylonitrile work			
4. Worked with acrylonitrile since DD/MM/YYYY			
5. Satisfactory personal hygiene (for example nail biting, frequency of hand washing)		☐ Yes	☐ No
6. Risk assessment completed		☐ Yes	☐ No

5. WORK ENVIRONMENT ASSESSMENT	(✓) all relevant boxes		
Date of assessment: DD/MM/YYYY			
Acrylonitrile Industry			
☐ Production	**Controls:**		
☐ Automotive	Wear gloves	☐ Yes	☐ No
☐ Other (specify):	Respirator use	☐ Yes	☐ No
	Local exhaust ventilation	☐ Yes	☐ No
	Overalls / work clothing	☐ Yes	☐ No
	Laundering by employer	☐ Yes	☐ No
	Wash basins & showers (with hot & cold water)	☐ Yes	☐ No
	Smoking or eating in workshop	☐ Yes	☐ No
	Personal hygiene:		
	Clean Shaven	☐ Yes	☐ No
	Shower & change into clean clothes at end of shift	☐ Yes	☐ No

6. BIOLOGICAL MONITORING RESULTS Include at least the previous two test results (if available)

Date	Tests performed	Recommended Action and/or Comment
1. DD/MM/YYYY		
2. DD/MM/YYYY		
3. DD/MM/YYYY		
4. DD/MM YYYY		
5. DD/MM/YYYY		
6. DD/MM/YYYY		
7. DD/MM YYYY		
8. DD/MM/YYYY		

7. RECOMMENDATIONS (by Medical Practitioner) (✓) all relevant boxes		
1. ☐ Suitable for work with acrylonitrile		
2. ☐ Counselling required		
3. ☐ Review workplace controls		
4. ☐ Repeat health assessment in month(s)/ week(s)		
5. ☐ Removal from work with acrylonitrile	On DD/MM/YYYY	
6. ☐ Medical examination by Medical Practitioner	On DD/MM/YYYY	
7. ☐ Fit to resume work	From DD/MM/YYYY	
8. ☐ Referred to Medical Specialist (respiratory/dermatology/other): On DD/MM/YYYY Specialist's name:		
Additional comments or recommendations arising from health monitoring:		
Medical Practitioner (responsible for supervising health monitoring)		
Name:	Signature	Date: DD/MM/YYYY
Tel:	Fax:	Registration Number:
Medical Practice:		
Address:		
Suburb:		Postcode:

SECTION 2 – THIS SECTION TO BE RETAINED BY THE MEDICAL PRACTITIONER			
1. PERSON CONDUCTING A BUSINESS OR UNDERTAKING			
Company / Organisation name:			
Site address:			
Suburb:		Postcode:	
Site Tel:	Site Fax:	Contact Name:	
2. OTHER BUSINESSES OR UNDERTAKINGS ENGAGING THE WORKER			
Company / Organisation name:			
Site address:			
Suburb:		Postcode:	
Site Tel:	Site Fax:	Contact Name:	
3. WORKER		(✓) all relevant boxes	
Surname:		Given names:	
Date of birth: DD/MM/YYYY	Sex:	☐ Male	☐ Female ☐ Pregnant/Breast Feeding?
Address:			
Suburb:		Postcode:	
Current Job:	Tel(H):	Mob:	
Date started employment : DD/MM/YYYY			
4. GENERAL HEALTH ASSESSMENT (if applicable)			
Symptoms of:	**Comments**	**Further testing?**	
Skin disorders		☐ Yes	☐ No
Headaches, dizziness		☐ Yes	☐ No
Respiratory disorders		☐ Yes	☐ No
Irritation of eyes, nose or throat		☐ Yes	☐ No
CNS		☐ Yes	☐ No
Others:		☐ Yes	☐ No

Height _____cm Weight _____kg Bp _____/_____ mmHg		☐ Yes ☐ No

5. OTHER MEDICAL HISTORY, FAMILY MEDICAL HISTORY, CURRENT MEDICATION, COMMENTS, TESTS OR RECOMMENDATIONS (use separate sheet if necessary)

Medical Practitioner (responsible for supervising health monitoring)

Name:	Signature	Date: DD/MM/YYYY
Tel:	Fax:	Registration Number:
Medical Practice:		
Address:		
Suburb:		Postcode:

ARSENIC (INORGANIC)

BASELINE HEALTH MONITORING BEFORE STARTING WORK IN AN INORGANIC ARSENIC PROCESS

Baseline health monitoring of the worker is required before the worker starts work in an inorganic arsenic process so that changes to the worker's health during inorganic arsenic work can be detected.

1. **Collection of demographic data**
2. **Work history**
3. **Medical history**
4. **Physical examination**

A physical examination will be conducted, with an emphasis on the peripheral nervous system and skin.

5. **Investigation**

A baseline level of arsenic in urine will be determined. The preferred method is by separation of the dietary arsenic from the inorganic arsenic and its metabolites by chromatographically separating the arsenic species, or by liquid-liquid extraction. Chromatographic separation is the superior technique as liquid-liquid extraction does not totally separate dietary arsenic from the inorganic arsenic metabolites and a small false positive may result.

It is recommended the test result not be reported adjusted to creatinine, however, the creatinine result should be provided separately in order to assist with the interpretation of the test result.

Where separation of dietary arsenic from the inorganic arsenic and its metabolites is not available total urinary arsenic, corrected for creatinine, may be determined. However, results using this method may be distorted where workers have recently eaten seafood or smoke heavily. In this instance, workers should be advised to abstain from seafood (including fish sauce, shrimp paste, fish and shellfish) and red wine for three days and seaweed for at least four days prior to urine collection.

DURING EXPOSURE TO AN INORGANIC ARSENIC PROCESS

6. **Monitoring exposure to inorganic arsenic**

The registered medical practitioner will assess exposure to inorganic arsenic through urinary inorganic arsenic testing, preferably by separation of the dietary arsenic from the inorganic arsenic and its metabolites.

Testing should be carried out at 90 day intervals, unless results consistently show urinary arsenic levels are low in which case the medical practitioner may decide less frequent testing is necessary. Tests should be done at end of a work shift at the end of the work week.

Where inorganic arsenic and its metabolites are present in urine above 0.47 μmol/L (35 μg/L) this indicates the worker may have been exposed to arsenic at work.
In this instance the following actions will be carried out:

- a repeat urine analysis for inorganic arsenic and its metabolites will be performed at the same time of the day to confirm test results
- a medical examination will be conducted, with particular emphasis on the peripheral nervous system and skin

- the person conducting a business or undertaking will be advised to:
  - review control measures at the workplace
  - carry out recommended remedial action
  - inform the worker of the results of the health monitoring.

Exposure to arsenic at the current exposure standard of 0.05 mg/m^3 TWA would be expected to result in a urinary arsenic level of approximately 54 µg inorganic As/L, as the sum of arsenic metabolites in the urine.

It is recommended biological monitoring is conducted as soon as practicable after the last potential exposure because inorganic arsenic in urine has a half-life of one to four days. The half-life needs to be taken into account during biological monitoring.

REMOVAL FROM WORK

Where a medical examination indicates the worker is displaying symptoms of exposure to arsenic, the medical practitioner should recommend the worker be removed from arsenic work. The worker must not return to inorganic arsenic work until they have been assessed as medically fit to return to work by the medical practitioner supervising the health monitoring.

Where total urinary arsenic is determined workers should be advised to abstain from seafood and red wine for three days and seaweed for at least four days prior to urine collection. Where test results show total urinary arsenic above 35 µg/L the sample should be tested for inorganic arsenic and its metabolites.

Since many of the health effects resulting from exposure to arsenic are chronic, for example carcinogenicity, the worker will not always display symptoms of exposure. So a removal level based on biological monitoring of urinary arsenic should be considered.

AT TERMINATION OF WORK IN AN INORGANIC ARSENIC PROCESS

7. Final medical examination

A final medical examination will be conducted and will include skin and neurological checks.

8. Continuing Medical Surveillance

People with skin or neurological signs due to arsenic should be advised to seek continuing medical surveillance.

SUPPLEMENTARY INFORMATION ON INORGANIC ARSENIC

Arsenic is a metalloid widely distributed in the earth's crust. It occurs in trace quantities in rock, soil, water and air. It is a common contaminant in most mineral ores. The main non-work source of arsenic is seafood. Arsenic exists in three common valence states:

- the metalloid (As^0)
- arsenite (trivalent state, As^{3+})
- arsenate (pentavalent state, As^{5+}).

9. 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 the 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)
- 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.

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

Arsenic from occupational sources occurs predominantly as As(III) and As(V). Both As(III) and As(V) are metabolised in the body and can be excreted in urine as the less toxic compounds, dimethyl arsinic acid (DMAv) and monomethyl arsonic acid (MMAv). In people exposed to high levels of As(III) or As(V) not all of the inorganic species will be converted in the body to MMAv or DMAv, and therefore As(III) and As(V) may also be excreted in urine. Total inorganic arsenic test result is the summation of MMAv + DMAv + As(III) + As(V).

10. Non-work sources

Fish, shellfish and seaweed contain organic arsenic compounds like arsenobetaine (AB) and a small amount of DMAv, which are excreted in urine unchanged. Seaweed can be consumed in the form of kelp as a dietary supplement or in Japanese cuisine. Seaweed contains a significant amount of arsenic predominantly in the form of arsenosugars. Urine total arsenic may reach levels of 1000 µg/g creatinine a few hours after eating a seafood meal with virtually all of this being in the form of organic arsenic.

MMAv is the metabolite from exposure to As(III) or As(V). DMAv is present in seafood and is the main metabolite from exposure to As(III) or As(V). As(III) and As(V) will be found present in the urine when moderate to high exposures have been experienced and the sample has been taken within 24 hrs of exposure.

Arsenobetaine is only present in seafood. Urinary excretion proportions are approximately 15-25% MMAv, 40-75% DMAv and 20-25% As(III) and/or As(V). These proportions can vary depending on exposed species, time after exposure and dose level.

11. Potential Health Effects Following Use Of Inorganic Arsenic

The relative toxicity of arsenic containing compounds depends primarily on its chemical type, valence state, 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.

12. 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 after mucociliary clearance. 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 percutaneously. Inorganic arsenic does not cross the blood-brain barrier but does cross the placenta.

13. Target organ/effect

Skin and mucous membranes – dermatitis, skin ulcers, hyperpigmentation, keratoses, skin cancer.

Nervous system – peripheral neuropathy.

Respiratory tract – irritation of nose, throat and lungs, perforation of nasal septum, lung cancer.

Gastrointestinal – irritation.

Circulatory system – peripheral vascular disease.

Bone marrow – pancytopaenia.

Liver – hepatocellular damage.

Hearing – potential ototoxin.

14. 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 in both acute and chronic exposures. Arsine gas combines with haemoglobin in erythrocytes to produce severe haemolysis with anaemia, haemoglobinuria and haematuria. Subsequent jaundice may be severe. Signs and symptoms of toxicity include nausea, vomiting and diarrhoea, apprehension and malaise, tachycardia and dyspnoea. Acute renal 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 percutaneous absorption, as well as from ingestion.

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, cardiac depression then shock. The CNS effects are headache, coma, convulsions, and cerebral oedema. Sensory loss in the peripheral nervous system and motor dysfunction can occur one to two weeks after large exposures. Anaemia and leucopenia occur a few days following exposure. Arsenic intoxication may also result in hepatic toxicity, including toxic hepatitis and elevated liver enzyme levels. Arsenic can cause convulsions, coma and death in severe poisoning.

If inhaled, mucous membrane irritation, dyspnoea and pulmonary oedema may occur.

15. Chronic effects

- contact dermatitis, scaling, blistering of the skin, hyperpigmentation 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, gastro-intestinal disturbances
- liver cirrhosis and portal hypertension. There may also be an increased risk of liver cancer
- peripheral neuropathy initially of hands and feet; essentially sensory. In more severe cases, motor paralysis may occur
- peripheral vascular insufficiency has been observed in people with chronic exposure to arsenic in drinking water
- bone marrow depression with pancytopaenia. Anaemia and leucopaenia are common in chronic arsenic toxicity, and are often accompanied by thrombocytopaenia and mild eosinophilia.

16. Carcinogenicity

Basal cell carcinomas, squamous cell carcinomas, Bowen's disease of the skin and lung carcinomas have been associated with chronic arsenic exposure. Skin cancers have been observed most commonly following exposure to medications containing trivalent arsenic compounds, particularly Fowler's solution, and environmental exposure to arsenic through drinking water.

Arsenic-induced skin cancer is frequently characterised by lesions over the entire body, mostly in unexposed areas like the trunk, palms and soles. Skin lesions manifest after a latent period of three to seven years for pigmentation changes and keratoses and up to 40 years for skin cancer.

A number of studies have shown an association between lung cancer and exposure to inorganic arsenic with the consistency between studies and biological gradient arguing for a causal relationship. There is a suggestion of an increased risk of liver, kidney and bladder cancer in some studies.

Work-related exposure to inorganic arsenic, especially in mining, copper smelting and pesticide work, has been associated with an increased risk of cancer.

17. Carcinogen and reproductive toxicant classifications

Arsenic acid and its salts with the exception of those specified elsewhere in Annex VI, arsenic trioxide, arsenic pentoxide and triethyl arsenate are classified according to the GHS 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).

FURTHER READING

Agency for Toxic Substances and Disease Registry, *Toxicological Profile for Arsenic*, Agency for Toxic Substances and Disease Registry, United States Department of Health and Human Services, Public Health Service, Atlanta, 2007.

International Agency for Research on Cancer, *Overall Evaluations of Carcinogenicity: An Updating of IARC Monographs Volumes 1-42*, Supplement No. 7, International Agency for Research on Cancer, Lyon, 1987.

Lauwerys RR, Hoet P, *Industrial Chemical Exposure Guidelines for Biological Monitoring*, 3rd Ed, Lewis Publishers, Boca Raton, 2001.

World Health Organisation/International Program on Chemical Safety. *Environmental Health Criteria 224: Arsenic and Arsenic Compounds*, World Health Organization, Geneva, 2001.

This health monitoring report is a confidential health record and must not be disclosed to another person except in accordance with the Work Health and Safety Regulations or with the consent of the worker.

There are two sections. Complete both sections and all questions if applicable.

Section 1 is to be forwarded to the PCBU who has engaged your services. A copy of laboratory report(s) must be attached > > > >

Section 2 may contain confidential information which may not be relevant to the health monitoring program being carried out. This section should be retained by the medical practitioner. Information which is required to be given to the PCBU should be summarised in part 7 of section 1.

SECTION 1 – THIS SECTION TO BE RETURNED TO THE PCBU			
1. PERSON CONDUCTING A BUSINESS OR UNDERTAKING			
Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
2. OTHER BUSINESSES OR UNDERTAKINGS ENGAGING THE WORKER			
Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
3. WORKER		(✓) all relevant boxes	
Surname:		Given names:	
Date of birth: DD/MM/YYYY	Sex:	☐ Male	☐ Female
Address:			
Suburb:			Postcode:
Current Job:	Tel(H):		Mob:
Date started employment : DD/MM/YYYY			
4. EMPLOYMENT IN ARSENIC RISK WORK		(✓) all relevant boxes	
1. ☐ New to arsenic (inorganic) work			
2. ☐ New worker but not new to arsenic (inorganic) work			
3. ☐ Current worker continuing in arsenic (inorganic) work			
4. Worked with arsenic (inorganic) since DD/MM/YYYY			
5. Satisfactory personal hygiene (for example nail biting, frequency of hand washing)		☐ Yes	☐ No
6. Risk assessment completed		☐ Yes	☐ No

5. WORK ENVIRONMENT ASSESSMENT		(✓) all relevant boxes	
Date of assessment: DD/MM/YYYY			
Arsenic (Inorganic) Industry			
☐ Manufacture	**Controls:**		
☐ Pesticides/Insecticides/ Fungicides	Wear gloves	☐ Yes	☐ No
☐ Leather Industry	Respirator use	☐ Yes	☐ No
☐ Gold Smelting	Local exhaust ventilation	☐ Yes	☐ No
☐ Nickel Smelting	Overalls / work clothing	☐ Yes	☐ No
☐ Tin Smelting	Laundering by employer	☐ Yes	☐ No
☐ Other Metal Industry	Wash basins & showers (with hot & cold water)	☐ Yes	☐ No
☐ Other (specify):	Smoking or eating in workshop	☐ Yes	☐ No
	Personal hygiene:		
	Clean Shaven	☐ Yes	☐ No
	Shower & change into clean clothes at end of shift	☐ Yes	☐ No

6. BIOLOGICAL MONITORING RESULTS Include at least the previous two test results (if available)		
Date	**Tests performed**	**Recommended Action and/or Comment**
1. DD/MM/YYYY		
2. DD/MM/YYYY		
3. DD/MM/YYYY		
4. DD/MM/YYYY		
5. DD/MM/YYYY		
6. DD/MM/YYYY		
7. DD/MM/YYYY		
8. DD/MM/YYYY		
Has worker abstained from consuming seafood and red wine for three days or seaweed for four days before urine test? ☐ Yes ☐ No		

7. RECOMMENDATIONS (by Medical Practitioner) (✓) all relevant boxes
1. ☐ Suitable for work with arsenic (inorganic)
2. ☐ Counselling required
3. ☐ Review workplace controls

4. ☐ Repeat health assessment in _____ month(s) / _____ week(s)		
5. ☐ Removal from work with arsenic (inorganic)	On DD/MM/YYYY	
6. ☐ Medical examination by Medical Practitioner	On DD/MM/YYYY	
7. ☐ Fit to resume work	From DD/MM/YYYY	
8. ☐ Referred to Medical Specialist (respiratory/dermatology/other): On DD/MM/YYYY Specialist's name:		
Additional comments or recommendations arising from health monitoring:		
Medical Practitioner (responsible for supervising health monitoring)		
Name:	Signature	Date: DD/MM/YYYY
Tel:	Fax:	Registration Number:
Medical Practice:		
Address:		
Suburb:		Postcode:

SECTION 2 – TO BE RETAINED BY THE MEDICAL PRACTITIONER			
1. PERSON CONDUCTING A BUSINESS OR UNDERTAKING			
Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
2. OTHER BUSINESSES OR UNDERTAKINGS ENGAGING THE WORKER			
Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
3. WORKER		(✓) all relevant boxes	
Surname:		Given names:	
Date of birth: DD/MM/YYYY	Sex:	☐ Female ☐ Male ☐ Pregnant/Breast Feeding?	
Address:			
Suburb:			Postcode:
Current Job:	Tel(H):		Mob:
Date started employment : DD/MM/YYYY			
4. GENERAL HEALTH ASSESSMENT (if applicable)			
Symptoms of:	**Comments**	**Further testing?**	
Skin disorders		☐ Yes	☐ No
Headaches, dizziness		☐ Yes	☐ No
Respiratory disorders		☐ Yes	☐ No
Irritation of eyes, nose or throat		☐ Yes	☐ No
Numbness in hands or feet		☐ Yes	☐ No
CNS		☐ Yes	☐ No
Others		☐ Yes	☐ No

Height _____cm Weight _____kg Bp _____/_____ mmHg		☐ Yes ☐ No

5. OTHER MEDICAL HISTORY, FAMILY MEDICAL HISTORY, CURRENT MEDICATION, COMMENTS, TESTS OR RECOMMENDATIONS (use separate sheet if necessary)

Medical Practitioner (responsible for supervising health monitoring)

Name:	Signature	Date: DD/MM/YYYY

Tel:	Fax:	Registration Number:
Medical Practice:		
Address:		
Suburb:		Postcode:

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ASBESTOS

BASELINE HEALTH MONITORING BEFORE STARTING ASBESTOS RELATED WORK OR REMOVAL WORK

1. **Collection of demographic data**
2. **Work history**
3. **Medical History**

Administration of a standardised respiratory questionnaire - **see Appendix 2.**

4. **Physical Examination -** see Appendix 1

A physical examination will only be conducted if indicated by work and medical history. Emphasis should be on the respiratory system.

5. **Investigation**

Standardised respiratory function tests will be conducted to determine the worker's baseline respiratory function. Current evidence does not support screening for lung cancer with chest radiography or sputum cytology.

DURING ASBESTOS RELATED WORK OR REMOVAL WORK

6. **Monitoring exposure to asbestos**

If a worker is carrying out licensed asbestos removal work, the Work Health and Safety Regulations require health monitoring is conducted prior to the worker commencing the work. The frequency of health monitoring should be determined by a risk assessment and the significance and frequency of past or future exposure. Health monitoring should be conducted at regular intervals, for example once every two years.

Monitoring should include administration of the standardised respiratory questionnaire. It would not ordinarily include respiratory function tests, chest X-ray or physical examination unless clinical indications are present or they are recommended by the medical practitioner.

AT TERMINATION OF ASBESTOS RELATED WORK OR REMOVAL WORK

7. **Final medical examination**

A final medical examination will be conducted with emphasis on the respiratory system.

SUPPLEMENTARY INFORMATION ON ASBESTOS

8. **Work activities that may represent a high risk exposure**

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).

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 and site excavation, for example during road building, amphiboles, like tremolite and anthophyllite, may be present as geological contaminants.

POTENTIAL HEALTH EFFECTS FOLLOWING EXPOSURE TO ASBESTOS

9. Route of entry into body/absorption/excretion pharmacology

Although asbestos is a hazardous material it can only pose a risk to health if the asbestos fibres become airborne and are inhaled. Inhalation is the primary route of entry to the body. Small fibrous particles may become airborne and inhaled. Respirable fibres are fibres that are more likely to reach the small airways and alveolar region of the lung and are defined as having a length of more than five microns, and an aspect ratio (length/width) greater than 3:1.

10. Factors affecting risks of contracting asbestos related disease

This depends on factors including:

- fibre type
- size and shape of fibres
- concentration of asbestos fibres in the inhaled air
- period of time over which the person was exposed.

Long fibres of asbestos have more potential to cause disease than short fibres and a high aspect ratio has also been implicated as an important factor in the pathogenesis of asbestos-related disease, particularly mesothelioma.

Much of the current burden of asbestos-related disease is a result of past heavy industrial exposure among those who manufactured and installed asbestos products. Mesothelioma can result from brief periods of exposure and a pattern of repeated exposure can lead to a substantial cumulative exposure.

11. Sources of non-occupational exposure to asbestos

Low levels of asbestos fibres are present in the environment from the breakdown of asbestos products. Environmental weathering of asbestos-cement sheets in roofing and wall cladding, disturbance of asbestos from a variety of building materials like insulation, ceiling tiles, and floor tiles and asbestos release to air from clutches and brakes in cars and trucks results in asbestos fibres being widespread in the environment.

The typical environmental background in outdoor air is 0.0005 fibres/ml and 0.0002 fibres/ml in indoor air[1]. The daily inhalation volume for an average adult is 22 m^3 or 22000 litres[2]. This means 5500 fibres are breathed/day by the average person (proportion of time spent indoors = 20 hours/day). Despite this the general population does not contract asbestos-related disease in significant numbers. The background rate of mesothelioma is less than one per million per year. By comparison, the annual death rate for a 40 year old male in 2008 was 1.6 per thousand or 1600 per million[3].

INCIDENTAL EXPOSURE

People who may have been exposed to asbestos are often anxious and concerned about the possible effects on their health. There is at present no post-exposure prophylaxis for the effects of inhaled asbestos fibres, although in smokers the risk of asbestos-induced lung cancer (but not mesothelioma) can be reduced by stopping smoking. There are also no generally available techniques for determining individual lung burdens of asbestos fibres, other than post-mortem. Asbestos related damage to the lungs takes years to develop and become visible on chest X-rays, and X-ray examinations cannot indicate whether or not asbestos fibres have been inhaled. Given this, and the long latency period, there is no reason to subject individuals with a suspected incidental exposure to even a small dose of ionizing radiation.

DISEASES AND SYMPTOMS OF ASBESTOS EXPOSURE[4]

Pleural plaques

An indicator of exposure to asbestos. They are diagnosed with a high degree of accuracy on high-resolution computed tomography (HRCT) imaging. Latency period is usually 20 or more years after the onset of exposure to asbestos dust.

Benign asbestos pleural effusion

An exudative pleural effusion that usually resolves spontaneously but may be followed by progressive pleural fibrosis.

Progressive pleural fibrosis (diffuse pleural thickening)

Recognised on a plain radiograph as pleural thickening that obliterates a costophrenic angle, and is present on HRCT by definition when a lesion equals or exceeds 8 cm in height and 5 cm in width. It may result in impaired lung function, particularly reduced lung volumes, with elevation of the diffusion constant.

Transpulmonary bands (crow's feet)

An extension of subpleural fibrosis along bronchovascular sheaths and arise from visceral pleural plaques and evident on computed tomography (CT).

Rounded atelectasis

This is the most common of the benign masses caused by exposure to asbestos. It usually occurs in the subpleural, posterior, or basal region of the lower lobes. Pleural thickening is always present and is commonly greatest near the mass.

- 1 ATSDR. 2001. Toxicological profile for Asbestos. Atlanta: Agency for Toxic Substances and Disease Registry www.atsdr.cdc.gov
- 2 Enhealth Dept Health and Ageing Environmental Health Risk Assessment www.health.gov.au
- 3 Australian Bureau of Statistics at www.ausstats.abs.gov.au
- 4 De Klerk N, Henderson D, Jones M, Leigh J, Musk AW, Shilkin K, Williams V, 'The diagnosis and attribution of asbestos-related diseases in an Australian context', Adelaide Workshop on Asbestos-Related Diseases, *J Occup Health Safety – Aust NZ*, vol 18(5), pp 443-452, 2002.

Asbestosis

Diffuse interstitial pulmonary fibrosis following asbestos exposure is recognised clinically by the presence of crackles on auscultation, small irregular opacities radiographically, and restrictive changes in lung function. CT has a higher sensitivity for minor interstitial changes compared with chest X-ray and is best seen on prone films. Prone scans abolish the gravity dependent subpleural density at the lung bases which obscures early disease.

The early changes of asbestosis are subpleural dots, subpleural lines, septal lines and small honeycomb cysts. In subjects who have had asbestos exposure, idiopathic pulmonary fibrosis (IPF) is indistinguishable from asbestosis clinically, physiologically, radiologically and pathologically except that the presence of pleural plaques increases the likelihood asbestos is responsible for the fibrosis. Rapidly progressive fibrosis is more likely in IPF than asbestosis.

Typically asbestosis causes a restrictive pattern on pulmonary function tests. A forced vital capacity (FVC), a total lung capacity (TLC) and/or a diffusing capacity of the lung for carbon monoxide, that is DLCO less than the 95% confidence lower limit, suggest the presence of an interstitial fibrotic process consistent with asbestosis. Constriction of bronchioles, with decreased expiratory flow rates at low lung volumes (FEF_{25-75}), may be the earliest functional impairment.

Malignant mesothelioma of the pleura and peritoneum

Malignant mesothelioma has a strong association with a history of asbestos exposure often at levels less than the cumulative exposures required to induce asbestosis or lung cancer. The amphibole varieties of asbestos (crocidolite and amosite) are substantially more potent than chrysotile for mesothelioma induction. There is a long latency period from 10 to 50 years between exposure and the development of mesothelioma with mean latency 37.4 years.

Malignant mesothelioma is locally aggressive and invasive with mean survival of 17.6 months from first symptom appearing.

Lung cancer

The relative frequencies of the large and small cell varieties are similar to those that are seen in smokers without asbestos exposure. The risk is dose dependent and the effects of tobacco smoking and asbestos are synergistic. The average latency is 20-30 years.

MESOTHELIOMA REGISTER

The Australian Mesothelioma Registry (AMR)[5] is a database that contains information about people with mesothelioma. It monitors all new cases of mesothelioma diagnosed in Australia from 1 July 2010.

Each state and territory cancer registry provides the AMR with information about each person diagnosed with mesothelioma on or after 1st July 2010 in Australia. Notification of cancer is a legal requirement for all public and private hospitals, radiotherapy departments, nursing homes, pathology laboratories and outpatient departments.

12. Carcinogen classification

Asbestos is classified according to the GHS as Carcinogenicity Category 1A (May cause cancer).

5 http://www.mesothelioma-australia.com/home-page.aspx

APPENDIX 1

This health monitoring report is a confidential health record and must not be disclosed to another person except in accordance with the Work Health and Safety Regulations or with the consent of the worker.

1. PERSON CONDUCTING A BUSINESS OR UNDERTAKING		
Company / Organisation name:		
Site address:		
Suburb:		Postcode:
Site Tel:	Site Fax:	Contact Name:
2. OTHER BUSINESSES OR UNDERTAKINGS ENGAGING THE WORKER		
Company / Organisation name:		
Site address:		
Suburb:		Postcode:
Site Tel:	Site Fax:	Contact Name:
3. WORKER		(✓) all relevant boxes
Surname:	Given names:	
Date of birth: DD/MM/YYYY	☐ Male	☐ Female
Home Address:		
Suburb:		Postcode:
Current Job:	Tel(h):	Mobile:
Date examined: DD/MM/YYYY	Length of employment: YEARS/MONTHS	
4. WORK TASKS / ENVIRONMENT		(✓) all relevant boxes
Before this work, did you work in any other dusty environment or in a job with exposure to asbestos?	☐ Yes	☐ No

5. OCCUPATIONAL HISTORY		
Date e.g. 2004-2011	**PCBU and occupation(s)**	Note any exposures to dust, fibres, mists, fumes, chemicals
1.		
2.		
3.		
4.		
5.		

6. QUESTIONS ABOUT PRESENT WORK	(✓) all relevant boxes
1. ☐ How many years have you worked at your present work?	_____ years
2. ☐ How many days per week do you usually work?	_____ days
3. ☐ How many hours per day do you usually work?	_____ hours
4. In what types of work/tasks are you exposed to asbestos?	Hours per week

5. Do you ever wear breathing protection at work?	☐ Yes	☐ No	☐ Sometimes
6. How many years have you used breathing protection?		_____ years	

7. Do you wear equipment and clothing as protection against asbestos?
Circle answer.

	Never	Occasionally (<50% of the time)	Sometimes (50-79% of the time)	Usually (80-100% of the time)
Hand	1	2	3	4
Body	1	2	3	4
Eyes	1	2	3	4
Respiratory	1	2	3	4

8. What equipment/clothing do you use as protection against asbestos exposure?

Hand/s:

Body:

Eyes:

Respiratory:

9. Do you wear disposable protective garments?

☐ Yes How are they disposed of?

☐ No Are asbestos fibres vacuumed from work clothes with an asbestos vacuum cleaner with a HEPA filter and footwear wet wiped prior to leaving the asbestos work area?

☐ Yes ☐ No

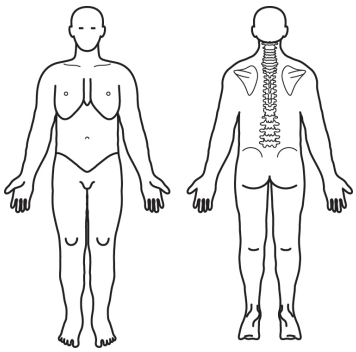
Are clothes washed separately at work in a dedicated washing machine?

☐ Yes ☐ No

10. What equipment/clothing do you use as protection against asbestos exposure?

7. MEDICAL EXAMINATION

Physical examination with emphasis on respiratory system (Mark in abnormalities)



1. Respiratory: Normal / crackles / wheeze
2. BP _____ / _____ mm Hg
3. Age: _____ years Weight: _____ kgs Height _____ cms BMI: _____

4.	Date of last volume calibration: DD/MM/YYYY
5.	Temperature: _____ °C
6.	At least 3 technically acceptable manoeuvres should be obtained with the highest and second highest FEV_1 and FVC within 0.15 L (within 0.100 L for those with an FVC of equal to or less than 1.0 L)*.

* Miller MR, Hankinson J, Brusasco V, Burgos F, Casaburi R, Coates A, Crapo R, Enright P, van der Grinten CPM, Gustafsson P, Jensen R, Johnson DC, MacIntyre N, McKay R, Navajas D, Pedersen OF, Pellegrino R, Viegi G, Wanger J, 'Standardisation of spirometry', Series "ATS/ERS Task Force: Standardisation of Lung Function Testing", Brusasco V, Crapo R, Viegi G (eds), Number 2 in this series, *Eur Respir J*, vol 26, pp 319-338, 2005.
http://www.thoracic.org/statements/resources/pfet/PFT2.pdf.

Use best result for FEV_1 and FVC, even if from different tests.

		% Predicted
FEV_1		
FVC		
FEV_1/FVC%		

☐ Normal ☐ Abnormal

Comments

8.	**RESULTS / RECOMMENDATIONS**	**(✓) all relevant boxes**	
1.	Is appropriate PPE used for all jobs?	☐ Yes	☐ No
2.	Respiratory symptoms	☐ Yes	☐ No
3.	Adequate workplace controls in place	☐ Yes	☐ No
4.	Medical counselling required	☐ Yes	☐ No
5.	Remove from exposure	☐ Yes	☐ No
6.	Follow-up medical examination	On DD/MM/YYYY	
7.	Referral to Medical Specialist Specialist's name:	On DD/MM/YYYY	
8.	☐ Control of exposure may not be adequate – recommend a review of work practices		
9.	Respiratory questionnaire completed and reviewed? Add comments/recommendations below	☐ Yes	☐ No

Additional comments and/or recommendations arising from health monitoring

Medical Practitioner (responsible for supervising health monitoring)

Name:	Signature	Date DD/MM/YYYY
Tel: ( )	Fax:	Registration Number:
Medical Practice		
Address:		
Suburb:		Postcode:

Appendix 2

Questionnaire based on the MRC (UK) Respiratory Questionnaire 1986, which has been extensively validated. This questionnaire can be completed by the worker at home. Additional questions have been added to cover clinical aspects of bronchial hyper-responsiveness validated by the Department of Occupational and Environmental Medicine, National Lung Institute[1].

The British Occupational Health Research Foundation (BOHRF)[2] concluded that in the clinical setting, questionnaires that identify symptoms of wheeze and/or shortness of breath which improve on days away from work or on holidays have a high sensitivity, but relatively low specificity for occupational asthma.

Preamble

Following are questions, mainly about your chest. Answer **yes** or **no** whenever possible.

If you are disabled from walking from any condition other than heart and lung disease, please begin questionnaire at **Question 5** and mark the adjacent box.

BREATHLESSNESS AND WHEEZING		
During the last month:		
1. Are you troubled by shortness of breath when hurrying on level ground or walking up a slight hill?	☐ Yes	☐ No
2. **If Yes to 1** - Do you get short of breath walking with other people of your age on level ground?	☐ Yes	☐ No
3. **If Yes to 2** - Do you have to stop for breath when walking at your own pace on level ground?	☐ Yes	☐ No
4. If you run, or climb stairs fast do you ever		
a. cough?	☐ Yes	☐ No
b. wheeze?	☐ Yes	☐ No
c. get tight in the chest?	☐ Yes	☐ No
5. Is your sleep ever broken		
a. by wheeze?	☐ Yes	☐ No
b. difficulty in breathing?	☐ Yes	☐ No
6. Do you ever wake up in the morning (or from your sleep if a shift worker)		
a. with wheeze?	☐ Yes	☐ No
b. difficulty with breathing?	☐ Yes	☐ No

1 Venables KM, Farrer N, Sharp L, Graneek BJ, Newman Taylor AJ, 'Respiratory Symptoms Questionnaire for Asthma Epidemiology: Validity and Reproducibility', *Thorax*, vol 48, pp 214-219, 1993.

2 The British Occupational Health Research Foundation (BOHRF), *Guidelines for Prevention, Identification and Management of Occupational Asthma: Evidence Review and Recommendations*, London 2004. www.bohrf.org.uk

7.	Do you ever wheeze		
	a. if you are in a smoky room?	☐ Yes	☐ No
	b. if you are in a very dusty place?	☐ Yes	☐ No
8.	**If Yes to either Q5, Q6, Q7** - Are your symptoms better		
	a. at weekends (or equivalent if shift worker)?	☐ Yes	☐ No
	b. when you are on holidays?	☐ Yes	☐ No
	If **Yes to Question 8**, please record details of any occupational exposure to respiratory hazards e.g. isocyanates, wood dust, aluminium pot room or asbestos, in **Additional notes** at the end of this questionnaire.		
COUGH			
9.	Do you usually cough first thing in the morning in winter?	☐ Yes	☐ No
10.	Do you usually cough during the day/ or at night / in the winter?	☐ Yes	☐ No
11.	**If Yes to Q9 or Q10** – Do you cough like this on most days for as much as three months each year?	☐ Yes	☐ No
PHLEGM			
12.	Do you usually bring up phlegm from your chest first thing in the morning in winter?	☐ Yes	☐ No
13.	Do you usually bring up any phlegm from your chest during the day / or at night / in winter?	☐ Yes	☐ No
14.	**If Yes to Q12 or Q13** – Do you bring up phlegm like this on most days for as much as three months each year?	☐ Yes	☐ No
PERIODS OF COUGH AND PHLEGM			
15.	In the past three years, have you had a period of (increased) cough and phlegm lasting for three weeks or more?	☐ Yes	☐ No
16.	**If Yes to Q15** – Have you had more than one such episode?	☐ Yes	☐ No
CHEST ILLNESSES			
17.	During the past three years, have you had any chest illness that has kept you from your usual activities for as much as a week?	☐ Yes	☐ No
18.	**If Yes to Q17** – Did you bring up more phlegm than usual in any of these illnesses?	☐ Yes	☐ No
19.	**If Yes to Q18** – Have you had more than one illness like this in the past three years?	☐ Yes	☐ No

PAST ILLNESSES			
20.	Have you ever had, or been told that you have had any of the following?		
	a. An injury, or operation affecting your chest?	☐ Yes	☐ No
	b. Heart problems?	☐ Yes	☐ No
	c. Bronchitis?	☐ Yes	☐ No
	d. Pneumonia?	☐ Yes	☐ No
	e. Pleurisy?	☐ Yes	☐ No
	f. Asthma?	☐ Yes	☐ No
	g. Other chest trouble?	☐ Yes	☐ No
	h. Hay fever?	☐ Yes	☐ No
TOBACCO SMOKING			
21.	Do you smoke?	☐ Yes	☐ No
If No to Q21			
22.	Have you ever smoked as much as one cigarette a day for as long as one year?	☐ Yes	☐ No
23.	How old were you when you started smoking regularly? _____		
24.	a. Do (did) you smoke manufactured cigarettes?	☐ Yes	☐ No
	If Yes to Q24a: How many do (did) you usually smoke per day? _____		
	b. on weekdays? _____		
	c. at weekends? _____		
25.	Do (did) you smoke any other forms of tobacco?	☐ Yes	☐ No
	If Yes to Q25, record details under Additional notes		
FOR EX-SMOKERS			
26.	When did you give up smoking altogether?	Month _____	Year _____
Additional notes:			

BENZENE

BASELINE HEALTH MONITORING BEFORE STARTING WORK IN A BENZENE PROCESS

1. **Collection of demographic data**
2. **Work history**
3. **Medical history**
4. **Physical examination**

A physical examination will be conducted only if indicated by work and medical history

5. **Investigation**

A blood sample for haematological profile will be used to test the worker's baseline exposure.

DURING EXPOSURE TO A BENZENE PROCESS

6. **Biological exposure**

S-Phenylmercapturic acid (S-PMA) and trans-trans-muconic acid (tt-MA) are two minor urinary metabolites of benzene which can be used as a measure of exposure to benzene. S-PMA is a highly specific marker of benzene exposure. It has greater specificity than other markers like phenol which is unsuitable for benzene monitoring unless at very high levels. The metabolite (tt-MA) is useful for benzene monitoring at exposures < 0.5 ppm but has the interference of sorbic acid and sorbitol found in certain foods including cheese, syrup, jelly, cake, dry fruits and soft drinks.

A spot urine test can be used to determine levels of S-PMA relative to creatinine. A spot urine test for S-PMA should be performed at the end of a work shift. S-PMA has a half-life of nine hours in urine.

A spot urine test showing a level of S-PMA of 25 µg/g creatinine indicates the worker may have been occupationally exposed to benzene[1]. If this occurs, the registered medical practitioner should consider a recommendation to remove the worker from the benzene work, taking into account other benzene exposure factors including the confounding factors described below. The person controlling the business or undertaking should also review control measures.

If spot urine testing demonstrates exposure to benzene is consistent with occupational exposure, the registered medical practitioner should also consider taking a blood sample to compare the haematological profile with the worker's baseline haematological profile determined at the beginning of the health monitoring process. For people exposed to relatively high levels of benzene, complete blood analysis can be used to monitor possible changes related to exposure.

1 The WorkCover NSW Biological Occupational Exposure Limit (BOEL) for benzene is 11.8 µmol S-PMA/mol creatinine in urine (which is equivalent to 25 µg/L in mass units), while the American Conference of Governmental Industrial Hygienists (ACGIH) biological exposure limits for benzene are 500 µg/g creatinine for tt-MA and 25 µg/g creatinine for S-PMA. WorkCover NSW BOEL and ACGIH biological exposure limit for S-PMA are about the same as there is approximately 1g creatinine/l urine.

7. Confounding factors

As tobacco smoke contains benzene, inhalation of tobacco smoke will cause elevated background values of S-PMA. The normal background level of S-PMA for a non-smoker is around 2.0 µg/g creatinine and 3.6 µg/g creatinine for a smoker.

AT TERMINATION OF WORK IN A BENZENE PROCESS

8. Final medical examination

A final medical examination will be carried out. A blood sample should be taken and results compared with the worker's baseline haematological profile. Workers with haematological abnormalities should be advised to seek continuing medical monitoring.

SUPPLEMENTARY INFORMATION ON BENZENE

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

9. 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 by filling rail tankers and top-filling road tankers with gasoline
- 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.

10. Non-work sources of exposure

- cigarettes
- vehicle exhaust
- petrol evaporation

Benzene, together with the other aromatic hydrocarbons toluene, ethylbenzene and the xylenes, is a major component of petrol. The aromatics increase the octane rating of petrol to the level needed by engines to provide acceptable fuel economy and performances. Of these aromatics, benzene is normally a minority component[2] . Benzene can escape into the air, particularly from vehicle fuel systems and from filling stations. The major source of exposure for the general population is from vehicle exhausts because in addition to the benzene actually present in petrol, it is also produced by chemical reactions during combustion in the engine.

2 Australian Government Department of Sustainability, Environment, Water, Population and Communities. www.environment.gov.au.

Cigarette smoke contains benzene and the World Health Organisation has estimated a benzene intake of 30 µg per cigarette. Passive smoking particularly indoors will also contribute to benzene intake of non-smokers.

Food and drinking water contains either no or negligible amounts of benzene.

POTENTIAL HEALTH EFFECTS FOLLOWING EXPOSURE TO BENZENE

11. Route of entry into the body

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

12. Target organ/effect

Blood/bone marrow – bone marrow depression (anaemia, leucopenia, thrombocytopaenia or pancytopaenia) and leukaemia, particularly acute myeloid leukaemia, and possibly an increased risk of non-Hodgkin's lymphoma and multiple myeloma.

CNS – acute CNS depression with drowsiness, dizziness, headaches and vomiting, and chronic solvent neurotoxicity.

Respiratory system – irritation.

Skin – irritation.

Eyes – irritation.

13. 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 arrythmias.

Central nervous system

The acute effects from exposure to high levels of benzene, that is 500-1000 ppm, 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 50-150 ppm for several hours can cause headaches, lassitude 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 pulmonary oedema, 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.

14. Chronic effects

Benzene is both haematotoxic and leukaemogenic by a range of mechanisms involving the bone marrow haematopoietic cell populations. The haematotoxic effects of benzene largely involve cytotoxic damage to the bone marrow stem cells. Chronic exposure to levels of 100-500 ppm have resulted in depression of bone marrow haemopoiesis leading to anaemia, leucopoenia, thrombocytopenia or pancytopenia.

For bone marrow depression, the lowest observed adverse effect level in humans is 7.6 parts per million (ppm) based on minimal blood count changes in otherwise healthy workers.

Metabolites of benzene are thought to be responsible for most of the toxic effects associated with benzene exposure. The molecular mechanisms underlying leukaemogenesis appear to involve clastogenic effects. Leukaemia cell populations are monoclonal (resulting from a single cell or cell type) and are found to have distinct chromosomal abnormalities thought to be due to interference by benzene metabolite with chromosomal separation during cell division.

Benzene may be present as a contaminant in mixed solvents and health effects may be due to exposure to the mixture.

The chronic effects of exposure to organic solvents include:

CNS
Neurotoxicity with symptoms including personality or mood changes, fatigue, decreased motivation, difficulty in concentration and impairment in memory.

SKIN
Almost all organic solvents are primary skin irritants as a result of defatting of the skin. Up to 20% of cases of work-related dermatitis are caused by solvents.

LIVER
Organic solvents may cause hepato-cellular damage if there is exposure in sufficient dose for sufficient duration.

KIDNEYS
There are reports of renal effects in exposed workers. Chronic exposure to a number of solvents may result in mild renal tubular dysfunction evidenced by impaired re-absorption of proteins, glucose and amino acids by the proximal tubule. A characteristic sign of renal tubular dysfunction is an increased excretion of low molecular weight proteins in the urine including β2-microglobulin.

15. Carcinogenicity

Acute myeloid leukaemia has been demonstrated to occur 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.

16. Carcinogen and germ cell mutagen classification

Benzene is classified according to the GHS as a Carcinogenicity Category 1A (May cause cancer) and Germ Cell Mutagenicity Category 1B (May cause genetic defects).

FURTHER READING

Agency for Toxic Substances and Disease Registry, *Toxicological Profile for Benzene*, Agency for Toxic Substances and Disease Registry, United States Department of Health and Human Services, Public Health Service, Atlanta, 2007.

American Conference of Governmental Industrial Hygienists (ACGIH), *Documentation of the Threshold Limit Values and Biological Exposure Indices, Benzene*, 7th edition, Cincinnati, 2011.

American Petroleum Institute, *Biological mechanistic considerations relevant to benzene induced leukemogenesis*, ACGIH TLV Committee, 1996; 9/9/96.

Institute of Petroleum, *Guidelines for Health Surveillance and Biological Monitoring for Occupational Exposure to Benzene*, Occupational and Environmental Medical Sub-committee of the Institute of Petroleum, London, 1993.

International Programme on Chemical Safety, *Environmental Health Criteria 150: Benzene*, International Programme on Chemical Safety, World Health Organization, Geneva 1993.

National Industrial Chemicals Notification and Assessment Scheme, *Benzene, Priority Existing Chemical Assessment Report No.21*, Sep 2001.

Oil Companies' European Organisation for Environmental and Health Protection, *Guidelines for the Health Surveillance of Workers Exposed to Benzene in the Petroleum Industry Report No. 93/59*, Oil Companies' European Organisation for Environmental and Health Protection, Brussels, 1993.

This health monitoring report is a confidential health record and must not be disclosed to another person except in accordance with the Work Health and Safety Regulations or with the consent of the worker.

There are two sections. Complete both sections and all questions if applicable.

Section 1 is to be forwarded to the PCBU who has engaged your services. A copy of laboratory report(s) must be attached > > > >

Section 2 may contain confidential information which may not be relevant to the health monitoring program being carried out. This section should be retained by the medical practitioner. Information which is required to be given to the PCBU should be summarised in part 7 of section 1.

SECTION 1 – THIS SECTION TO BE RETURNED TO THE PCBU			
1. PERSON CONDUCTING A BUSINESS OR UNDERTAKING			
Company / Organisation name:			
Site address:			
Suburb:		Postcode:	
Site Tel:	Site Fax:	Contact Name:	
2. OTHER BUSINESSES OR UNDERTAKINGS ENGAGING THE WORKER			
Company / Organisation name:			
Site address:			
Suburb:		Postcode:	
Site Tel:	Site Fax:	Contact Name:	
3. WORKER		(✓) all relevant boxes	
Surname:		Given names:	
Date of birth: DD/MM/YYYY	Sex:	☐ Male	☐ Female
Address:			
Suburb:		Postcode:	
Current Job:	Tel(H):	Mob:	
Date started employment : DD/MM/YYYY			
4. EMPLOYMENT IN BENZENE RISK WORK		(✓) all relevant boxes	
1. ☐ New to benzene work			
2. ☐ New worker but not new to benzene work			
3. ☐ Current worker continuing in benzene work			
4. Worked with benzene since DD/MM/YYYY			
5. Satisfactory personal hygiene (for example nail biting, frequency of hand washing)		☐ Yes	☐ No
6. Risk assessment completed		☐ Yes	☐ No

5. WORK ENVIRONMENT ASSESSMENT		(✓) all relevant boxes	
Date of assessment: DD/MM/YYYY			
Benzene Industry			
☐ Refining	**Controls:**		
☐ Chemical Industry	Wear gloves	☐ Yes	☐ No
☐ Petrol Industry	Respirator use	☐ Yes	☐ No
☐ Automotive Industry	Local exhaust ventilation	☐ Yes	☐ No
☐ Plastics/Rubber Manufacturing	Overalls / work clothing	☐ Yes	☐ No
☐ Steel Industry	Laundering by employer	☐ Yes	☐ No
☐ Emergency Services	Wash basins & showers (with hot & cold water)	☐ Yes	☐ No
☐ Other (specify):	Smoking or eating in workshop	☐ Yes	☐ No
	Personal hygiene:		
	Clean Shaven	☐ Yes	☐ No
	Shower & change into clean clothes at end of shift	☐ Yes	☐ No

6. BIOLOGICAL MONITORING RESULTS Include at least the previous two test results (if available)		
Date	**Tests performed**	**Recommended Action and/or Comment**
1. DD/MM/YYYY		
2. DD/MM/YYYY		
3. DD/MM/YYYY		
4. DD/MM/YYYY		
5. DD/MM/YYYY		
6. DD/MM/YYYY		
7. DD/MM/YYYY		
8. DD/MM/YYYY		

7. RECOMMENDATIONS (by Medical Practitioner)	(✓) all relevant boxes
1. ☐ Suitable for work with benzene	
2. ☐ Counselling required	
3. ☐ Review workplace controls	
4. ☐ Repeat health assessment in _____ month(s) / _____ week(s)	
5. ☐ Removal from work with benzene	On DD/MM/YYYY
6. ☐ Medical examination by Medical Practitioner	On DD/MM/YYYY
7. ☐ Fit to resume work	From DD/MM/YYYY
8. ☐ Referred to Medical Specialist (respiratory/ dermatology/other)	On DD/MM/YYYY
Specialist's name:	
Additional comments or recommendations arising from health monitoring:	

Medical Practitioner (responsible for supervising health monitoring)		
Name:	Signature	Date: DD/MM/YYYY
Tel:	Fax:	Registration Number:
Medical Practice:		
Address:		
Suburb:		Postcode:

SECTION 2 – THIS SECTION TO BE RETAINED BY THE MEDICAL PRACTITIONER			
1. PERSON CONDUCTING A BUSINESS OR UNDERTAKING			
Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
2. OTHER BUSINESSES OR UNDERTAKINGS ENGAGING THE WORKER			
Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
3. WORKER		(✓) all relevant boxes	
Surname:		Given names:	
Date of birth: DD/MM/YYYY	Sex:	☐ Male	☐ Female ☐ Pregnant/Breast Feeding?
Address:			
Suburb:			Postcode:
Current Job:	Tel(H):		Mob:
Date started employment : DD/MM/YYYY			
4. GENERAL HEALTH ASSESSMENT (if applicable)			
Symptoms of:	**Comments**	**Further testing?**	
Skin disorders		☐ Yes	☐ No
Headaches, dizziness		☐ Yes	☐ No
Respiratory disorders		☐ Yes	☐ No
Irritation of eyes, nose or throat		☐ Yes	☐ No
Cough		☐ Yes	☐ No
CNS		☐ Yes	☐ No
Others		☐ Yes	☐ No

Height _____cm Weight _____kg Bp _____/_____ mmHg		☐ Yes ☐ No

5. OTHER MEDICAL HISTORY, FAMILY MEDICAL HISTORY, CURRENT MEDICATION, COMMENTS, TESTS OR RECOMMENDATIONS (use separate sheet if necessary)

Medical Practitioner (responsible for supervising health monitoring)

Name:	Signature	Date: DD/MM/YYYY
Tel:	Fax:	Registration Number:
Medical Practice:		
Address:		
Suburb:		Postcode:

CADMIUM

BASELINE HEALTH MONITORING BEFORE STARTING WORK IN A CADMIUM PROCESS

Workers must be informed about the potential health effects associated with exposure to cadmium. This should include counselling on the effect of smoking on cadmium exposure.

1. **Collection of demographic data**
2. **Work history**
3. **Medical history**

Administration of a standardised respiratory questionnaire. Two examples are the International Union Against Tuberculosis' *Bronchial Symptoms Questionnaire 1986* [1] **or** the Medical Research Council's *Questionnaire on Respiratory Symptoms 1986* [2].

4. **Physical examination**

A physical examination will be conducted, with an emphasis on the respiratory system.

5. **Investigation**

The following tests will be used to test the worker's baseline exposure:

- Standardised respiratory function tests – FEV_1[1], FVC[2] and FEV_1/FVC[3]. (**Note**: the normal ranges for predictive values should be stated.)
- A spot urine for cadmium will be conducted and the results will be corrected for creatinine. Where there is 5 µg or more cadmium per gram of creatinine, repeat urine tests should be performed at the same time of the day.
- A urine $ß_2$-microglobulin test will be conducted and the results will be corrected for creatinine.

Note: There may be abnormal values for some renal markers [3] at thresholds of urinary cadmium between 3 and 5 µg/g of creatinine.

BACKGROUND INFORMATION ON CADMIUM EXPOSURE

The measurement of cadmium in urine estimates chronic exposure. However, it may provide no information on exposure during the first year of exposure. In the workplace, the lungs are the major route of absorption of aerosols, dusts and fumes containing cadmium. The main route of elimination of cadmium is renal. Renal tubular damage from cadmium or renal tubular dysfunction of other aetiologies results in increased renal elimination of cadmium.

1 Forced expiratory volume in one second

2 Forced vital capacity

3 Tiffeneau index

DURING EXPOSURE TO A CADMIUM PROCESS

6. Monitoring exposure to cadmium

COMPARISON OF RESULTS WITH BASELINE LEVEL

Work-related exposure to cadmium can be assessed by monitoring urine. Two methods can be used:

- measurement of cadmium in urine as µg/g of creatinine
- assessment of $ß_2$-microglobulin as µg/g of creatinine.

A spot urine for cadmium and urine $ß_2$-microglobulin will be conducted annually and compared against the worker's baseline levels measured at the start of the health monitoring process.

ACTION LEVEL

Where testing results show a level of a level of 3 µg cadmium or more per gram of creatinine in urine or more than 200 µg $ß_2$-microglobulin per gram creatinine:

- repeat urine tests should be conducted at the same time of the day to confirm results
- a medical examination should be performed
- the person conducting a business or undertaking must review control measures and carry out recommended remedial action
- the worker must be informed of the results of the health monitoring.

The information on **blood cadmium** is scant compared with that on urinary cadmium. Cadmium concentration in blood reflects recent exposure over months. The action level is 5 µg/L.

REMOVAL LEVEL

Where results of urine testing indicate a level of cadmium in urine above 5 µg per gram of creatinine or the level of $ß_2$-microglobulin exceeds 1000 µg per gram creatinine, the following action should be carried out:

- a medical examination should be performed and the registered medical practitioner should consider whether the worker should be removed from cadmium work
- urine tests should be repeated every six months until the level falls below 3 µg cadmium per gram creatinine and $ß_2$-microglobulin is less than 200 µg per gram creatinine
- the person conducting a business or undertaking should review control measures and carry out recommended remedial action
- the worker must be informed of the results of health monitoring.

RETURN TO WORK

The worker must not return to cadmium work until they have been assessed as medically fit to return to work by the medical practitioner supervising the health monitoring.

7. Medical examination

A medical examination should be conducted every two years and will include:

- medical history and counselling on the additional cadmium burden from smoking
- physical examination
- respiratory function tests.

AT TERMINATION OF WORK IN A CADMIUM PROCESS

8. Final medical examination

A final medical examination will be conducted and workers with a history of raised ß2-microglobulin should be advised to seek continuing medical monitoring.

SUPPLEMENTARY INFORMATION ON CADMIUM

9. 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.

10. Non-work sources

The general population may be exposed to cadmium through food like potatoes, grain cereal products and seafood.

The average intake of cadmium for Australian males is 30 µg per day and about 1 µg of cadmium is absorbed.

The use of superphosphate fertiliser has resulted in addition of cadmium to the soil in some agricultural areas. Plants take up and retain cadmium from soil; fish and shellfish take up and retain cadmium from water.

Tobacco, like other plants, takes up cadmium, which is then inhaled in the smoke. Each cigarette contains about 1 to 2 μg cadmium and about 10 per cent of this is absorbed. People in high traffic areas may have elevated levels of cadmium.

POTENTIAL HEALTH EFFECTS FOLLOWING EXPOSURE TO CADMIUM

11. Route of entry into the body

The primary route of entry of cadmium into the body is through inhalation. Respiratory absorption of cadmium fumes ranges from 25 to 50 per cent. Only small cadmium particles are absorbed by the alveoli, and these small particles are typically found in fumes and cigarette smoke. As noted above, about 10 per cent of the cadmium in cigarettes is absorbed during smoking.

There is some risk of ingestion if personal hygiene is inadequate. Gastrointestinal absorption ranges from three to seven per cent, and absorption may be increased in the presence of calcium and iron deficiency. Percutaneous absorption is not significant. Cadmium does not pass through the placenta.

12. Target organ/effect

Kidney – proximal renal tubular dysfunction, proteinuria, renal calculi.

Respiratory tract – irritation, pneumonitis, pulmonary oedema, metal fume fever, chronic obstructive airways disease.

Bones – osteomalacia and osteoporosis secondary to hypercalciuria, hyperphosphaturia and decreased 1-hydroxylation of 25-hydroxy vitamin D in tubular cells.

Stomach – acute gastroenteritis.

Liver – elevation of hepatic enzymes.

13. Acute effects [4]

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, pneumonitis, and pulmonary oedema. 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/m^3 for a period of about one hour. Relatively mild cases resemble metal fume fever.

High ingestion exposure of soluble cadmium salts causes acute gastroenteritis. The amount of cadmium absorbed is probably very limited due to vomiting and the consequential short presence of cadmium in the gastrointestinal tract.

14. Chronic effects

Long-term work-related exposure to cadmium has caused severe chronic effects, predominantly in the lungs and kidneys. Kidney effects are described as tubular dysfunction rather than florid renal disease or renal failure. Renal toxicity may be caused by chronic inhalation or chronic ingestion of cadmium. Commonly, the proximal renal tubules are affected resulting in urinary excretion of low molecular weight proteins like $ß_2$-microglobulin. Increased excretions are predictive of an acceleration of age-related decline of the glomerular filtration rate. A raised urinary excretion of $ß_2$-microglobulin is not specific to renal dysfunction induced by cadmium. The differential diagnosis includes renal disorders like diabetic nephropathy or excessive production in some cancers and autoimmune disorders.

The accumulation of cadmium in the renal cortex leads to renal tubular dysfunction. This occurs at levels of exposure much lower than previously thought. Cadmium-induced tubular proteinuria is irreversible, and continued exposure may lead to glomerular damage with decreased glomerular filtration rate. 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. The Cadmibel study randomly selected 1700 subjects in Belgium and demonstrated a 10 % probability of tubular dysfunction when cadmium excretion exceeded 2-3 µg cadmium/g creatinine.

Increased incidence of calcium containing renal calculi has occurred in work-related exposed groups. Overt bone disease, like osteomalacia and osteoporosis, is related to hypercalciuria and hyperphosphaturia. Decreased 1-hydroxylation of 25-hydroxy vitamin D in tubular cells has been recorded particularly in post-menopausal women deficient in calcium (Itai-Itai disease).

Lung changes are primarily characterised by chronic obstructive airway disease. Early minor changes in ventilatory function tests may progress with continued cadmium exposure to respiratory insufficiency.

The anaemia observed in association with cadmium exposure has been a mild reversible depression of haemoglobin.

15. Carcinogenicity

Genotoxic effects have been observed in animals exposed to cadmium chloride *in vivo* and in human cells exposed *in vitro* to cadmium chloride or cadmium sulfide [5]. There are reports [5] of increased chromosomal aberrations occurring in peripheral blood lymphocytes of workers exposed to cadmium in the metal industry. However, some of these workers also had concomitant exposure to other metals like zinc, copper and lead.

Results of studies [5] conducted in the United Kingdom, United States and Sweden on the incidence of lung cancer in cadmium-exposed workers provide consistent evidence long-term work-related exposure to cadmium may contribute to the development of lung cancer. However, in some of these studies observations from exposed workers have been difficult to interpret because of confounding factors.

A number of early studies (pre 1965) reported an increased risk for prostatic cancer among workers employed in a plant manufacturing cadmium-nickel batteries in the United Kingdom. However, results of other studies, including a later study in the same plant, a similar plant in Sweden and a United States population-based case-control study on prostate cancer, do not support the suggestion from earlier studies of a causal relationship [5].

16. Carcinogen, germ cell mutagen and reproductive toxicant classifications

The following are some cadmium-containing chemicals with GHS carcinogenicity, germ cell mutagenicity and reproductive toxicity 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.

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

The International Agency for Research on Cancer [5] classification for cadmium and cadmium compounds is Group 1; carcinogenic to humans. According to the International Agency for Research on Cancer this category is used only when there is sufficient evidence of carcinogenicity in humans.

REFERENCED DOCUMENTS

1. Respiratory Disease Committee of the International Union Against Tuberculosis, *IUAT Bronchial Symptoms Questionnaire*, International Union Against Tuberculosis, 1986.
2. Medical Research Council Committee on Research into Chronic Bronchitis, *MRC Questionnaire on Respiratory Symptoms*, Medical Research Council, 1986.
3. Lauwerys RR, Hoet P, *Industrial Chemical Exposure Guidelines for Biological Monitoring*, 3rd Ed, Lewis Publishers, Boca Raton, 2001.
4. International Programme on Chemical Safety, *Environmental Health Criteria 134: Cadmium*, International Programme on Chemical Safety, World Health Organization, Geneva, 1992.
5. International Agency for Research on Cancer, *IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans, Volume 58: Beryllium, Cadmium, Mercury and Exposures in the Glass Manufacturing Industry*, International Agency for Research on Cancer, Lyon, 1993.

FURTHER READING

Agency for Toxic Substances and Disease Registry, *Case Studies in Environmental Medicine: Cadmium Toxicity*, Agency for Toxic Substances and Disease Registry, United States Department of Health and Human Services, Public Health Service, Atlanta, 2008.

Buchet JP, Lauwerys R, Roels H, Bernard A, Bruaux P, Claeys F, Ducoffre G, De Plaen P, Staessen J, Amery A, Lijnen P, Thijs L, Rondia D, Sartor F, Saint Remy A, Nick L, 'Renal effects of cadmium body burden of the general population', *Lancet*, vol 336, pp 699-702, 1990.

Health and Safety Executive (United Kingdom), *Cadmium: Health and Safety Precautions*, Guidance Note EH 1, Health and Safety Executive, London, 1995.

Jarup L, 'Cadmium overload and toxicity', *Nephrology Dialysis Transplantation*, vol 17 (supplement 2), pp 35-39, 2002.

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There are two sections. Complete both sections and all questions if applicable.

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SECTION 1 – THIS SECTION TO BE RETURNED TO THE PCBU			
1. PERSON CONDUCTING A BUSINESS OR UNDERTAKING			
Company / Organisation name:			
Site address:			
Suburb:		Postcode:	
Site Tel:	Site Fax:	Contact Name:	
2. OTHER BUSINESSES OR UNDERTAKINGS ENGAGING THE WORKER			
Company / Organisation name:			
Site address:			
Suburb:		Postcode:	
Site Tel:	Site Fax:	Contact Name:	
3. WORKER		(✓) all relevant boxes	
Surname:	Given names:		
Date of birth: DD/MM/YYYY	Sex:	☐ Male	☐ Female
Address:			
Suburb:		Postcode:	
Current Job:	Tel(H):	Mob:	
Date started employment : DD/MM/YYYY			
4. EMPLOYMENT IN CADMIUM RISK WORK		(✓) all relevant boxes	
1. ☐ New to cadmium work			
2. ☐ New worker but not new to cadmium work			
3. ☐ Current worker continuing in cadmium work			
4. Worked with cadmium since DD/MM/YYYY			
5. Satisfactory personal hygiene (for example nail biting, frequency of hand washing)		☐ Yes	☐ No
6. Risk assessment completed		☐ Yes	☐ No

5. WORK ENVIRONMENT ASSESSMENT	(✓) all relevant boxes		
Date of assessment: DD/MM/YYYY			
Cadmium Industry	☐ Smoker ☐ Ex-smoker ☐ Non-smoker		
☐ Welding/Fabrication ☐ Electroplating ☐ Cadmium Manufacture/ Extraction ☐ Battery Construction/ Disposal ☐ Plastics/Paints ☐ Textile Industry ☐ Other (specify):	**Controls:**		
	Wear gloves	☐ Yes	☐ No
	Respirator use	☐ Yes	☐ No
	Local exhaust ventilation	☐ Yes	☐ No
	Overalls / work clothing	☐ Yes	☐ No
	Laundering by employer	☐ Yes	☐ No
	Wash basins & showers (with hot & cold water)	☐ Yes	☐ No
	Smoking or eating in workshop	☐ Yes	☐ No
	Personal hygiene:		
	Clean Shaven	☐ Yes	☐ No
	Shower & change into clean clothes at end of shift	☐ Yes	☐ No

6. BIOLOGICAL MONITORING RESULTS Include at least the previous two test results (if available)		
Date	**Tests performed**	**Recommended Action and/or Comment**
1. DD/MM/YYYY	Respiratory function test	FEV_1_____ ; FVC_____; FEV_1/FVC_____
2. DD/MM/YYYY		
3. DD/MM/YYYY		
4. DD/MM/YYYY		
5. DD/MM/YYYY		
6. DD/MM/YYYY		
7. DD/MM/YYYY		
8. DD/MM/YYYY		

7. RECOMMENDATIONS (by Medical Practitioner)	(✓) all relevant boxes
1. ☐ Suitable for work with cadmium	
2. ☐ Counselling required	
3. ☐ Review workplace controls	
4. ☐ Repeat health assessment in _____ month(s) / _____ week(s)	
5. ☐ Removal from work with cadmium	On DD/MM/YYYY
6. ☐ Medical examination by Medical Practitioner	On DD/MM/YYYY
7. ☐ Fit to resume work	From DD/MM/YYYY
8. ☐ Referred to Medical Specialist (respiratory/ dermatology/other):	On DD/MM/YYYY
Specialist's name:	

Additional comments or recommendations arising from health monitoring:

Medical Practitioner (responsible for supervising health monitoring)		
Name:	Signature	Date: DD/MM/YYYY
Tel:	Fax:	Registration Number:
Medical Practice:		
Address:		
Suburb:		Postcode:

SECTION 2 – THIS SECTION TO BE RETAINED BY THE MEDICAL PRACTITIONER			
This questionnaire also allows for recordings of a more general health assessment at the end, if applicable.			
1. PERSON CONDUCTING A BUSINESS OR UNDERTAKING			
Company / Organisation name:			
Site address:			
Suburb:		Postcode:	
Site Tel:	Site Fax:	Contact Name:	
2. OTHER BUSINESSES OR UNDERTAKINGS ENGAGING THE WORKER			
Company / Organisation name:			
Site address:			
Suburb:		Postcode:	
Site Tel:	Site Fax:	Contact Name:	
3. WORKER		(✓) all relevant boxes	
Surname:	Given names:		
Date of birth: DD/MM/YYYY	Sex:	☐Male	☐ Female
Address:			
Suburb:		Postcode:	
Current Job:	Tel(H):	Mob:	
Date started employment : DD/MM/YYYY			

This questionnaire is based on the MRC (UK) Respiratory Questionnaire 1986, which has been extensively validated. This questionnaire is intended to be completed by an interviewer rather than by the patient. Additional questions have been added to cover clinical aspects of bronchial hyper-responsiveness validated by the Department of Occupational and Environmental Medicine, National Lung Institute[1].

The British Occupational Health Research Foundation (BOHRF)[2] concluded that in the clinical setting, questionnaires that identify symptoms of wheeze and/or shortness of breath which improve on days away from work or on holidays have a high sensitivity, but relatively low specificity for occupational asthma.

Preamble

I am going to ask some questions, mainly about your chest. I would like you to answer **yes** or **no** whenever possible.

If the subject is disabled from walking from any condition other than heart and lung disease, please begin questionnaire at **Question 5** and mark the adjacent box.

1 Venables KM, Farrer N, Sharp L, Graneek BJ, Newman Taylor AJ, 'Respiratory Symptoms Questionnaire for Asthma Epidemiology: Validity and Reproducibility', *Thorax*, vol 48, pp 214-219, 1993.

2 The British Occupational Health Research Foundation (BOHRF), *Guidelines for Prevention, Identification and Management of Occupational Asthma: Evidence Review and Recommendations*, London 2004. www.bohrf.org.uk

4. BREATHLESSNESS AND WHEEZING		
During the last month:		
1. Are you troubled by shortness of breath when hurrying on level ground or walking up a slight hill?	☐ Yes	☐ No
2. **If Yes to 1** - Do you get short of breath walking with other people of your age on level ground?	☐ Yes	☐ No
3. **If Yes to 2** - Do you have to stop for breath when walking at your own pace on level ground?	☐ Yes	☐ No
4. If you run, or climb stairs fast do you ever		
a. cough?	☐ Yes	☐ No
b. wheeze?	☐ Yes	☐ No
c. get tight in the chest?	☐ Yes	☐ No
5. Is your sleep ever broken		
a. by wheeze?	☐ Yes	☐ No
b. difficulty in breathing?	☐ Yes	☐ No
6. Do you ever wake up in the morning (or from your sleep if a shift worker)		
a. with wheeze?	☐ Yes	☐ No
b. difficulty with breathing?	☐ Yes	☐ No
7. Do you ever wheeze		
a. if you are in a smoky room?	☐ Yes	☐ No
b. if you are in a very dusty place?	☐ Yes	☐ No
8. **If Yes to either Q5, Q6, Q7** - Are your symptoms better		
a. at weekends (or equivalent if shift worker)?	☐ Yes	☐ No
b. when you are on holidays?	☐ Yes	☐ No
If **Yes to Question 8**, please record details of any occupational exposure to respiratory hazards e.g. isocyanates, wood dust, aluminium pot room or asbestos, in **Additional notes**.		
5. COUGH		
9. Do you usually cough first thing in the morning in winter?	☐ Yes	☐ No
10. Do you usually cough during the day/ or at night / in the winter?	☐ Yes	☐ No
11. **If Yes to Q9 or Q10** – Do you cough like this on most days for as much as three months each year?	☐ Yes	☐ No
6. PHLEGM		
12. Do you usually bring up phlegm from your chest first thing in the morning in winter?	☐ Yes	☐ No

13. Do you usually bring up any phlegm from your chest during the day / or at night / in winter?	☐ Yes	☐ No
14. **If Yes to Q12 or Q13** – Do you bring up phlegm like this on most days for as much as three months each year?	☐ Yes	☐ No
7. PERIODS OF COUGH AND PHLEGM		
15. In the past three years, have you had a period of (increased) cough and phlegm lasting for three weeks or more?	☐ Yes	☐ No
16. **If Yes to Q15** – Have you had more than one such episode?	☐ Yes	☐ No
8. CHEST ILLNESSES		
17. During the past three years, have you had any chest illness that has kept you from your usual activities for as much as a week?	☐ Yes	☐ No
18. **If Yes to Q17** – Did you bring up more phlegm than usual in any of these illnesses?	☐ Yes	☐ No
19. **If Yes to Q18** – Have you had more than one illness like this in the past three years?	☐ Yes	☐ No
9. PAST ILLNESSES		
20. Have you ever had, or been told that you have had any of the following?		
a. An injury, or operation affecting your chest?	☐ Yes	☐ No
b. Heart problems?	☐ Yes	☐ No
c. Bronchitis?	☐ Yes	☐ No
d. Pneumonia?	☐ Yes	☐ No
e. Pleurisy?	☐ Yes	☐ No
f. Asthma?	☐ Yes	☐ No
g. Other chest trouble?	☐ Yes	☐ No
h. Hay fever?	☐ Yes	☐ No
10. TOBACCO SMOKING		
21. Do you smoke?	☐ Yes	☐ No
If No to Q21		
22. Have you ever smoked as much as one cigarette a day for as long as one year?	☐ Yes	☐ No
23. How old were you when you started smoking regularly? _____		
24. a. Do (did) you smoke manufactured cigarettes?	☐ Yes	☐ No
If Yes to Q24a: How many do (did) you usually smoke per day? _____		
b. on weekdays?_____ c. at weekends? _____		

25. Do (did) you smoke any other forms of tobacco? ☐ Yes ☐ No **If Yes to Q25, record details under Additional notes**		
11. FOR EX-SMOKERS		
26. When did you give up smoking? Month _____ Year _____		
Additional notes:		
12. GENERAL HEALTH ASSESSMENT (if applicable)		
Symptoms of:	**Comments**	**Further testing?**
Skin disorders		☐ Yes ☐ No
Respiratory disorders		☐ Yes ☐ No
Any history of kidney stones or renal disease		☐ Yes ☐ No
Irritation of eyes, nose or throat		☐ Yes ☐ No
Cough		☐ Yes ☐ No
CNS		☐ Yes ☐ No
Others		☐ Yes ☐ No
Height _____cm Weight _____kg Bp _____/_____mmHg		☐ Yes ☐ No
13. OTHER MEDICAL HISTORY, FAMILY MEDICAL HISTORY, CURRENT MEDICATION, COMMENTS, TESTS OR RECOMMENDATIONS (use separate sheet if necessary)		
Medical Practitioner (responsible for supervising health monitoring)		
Name:	Signature	Date: DD/MM/YYYY
Tel:	Fax:	Registration Number:
Medical Practice:		
Address:		
Suburb:		Postcode:

CHROMIUM (INORGANIC)

BASELINE HEALTH MONITORING BEFORE STARTING WORK IN AN INORGANIC CHROMIUM PROCESS

1. **Collection of demographic data**
2. **Work history**
3. **Medical history**
4. **Physical examination**

A physical examination will be conducted, with emphasis on the respiratory system and skin.

DURING EXPOSURE TO AN INORGANIC CHROMIUM PROCESS

5. Workplace skin care program

The medical practitioner should tell the person conducting a business or undertaking before the worker starts work with inorganic chromium, that they must ensure the worker's skin is inspected weekly by a competent person. Particular attention should be placed on the skin of the hands and forearms. Where skin abnormalities occur, the person conducting a business or undertaking must arrange for the worker to see the medical practitioner.

6. Respiratory symptoms

The PCBU should be advised that any respiratory symptoms exhibited by the worker should be reported to the registered medical practitioner.

7. Monitoring exposure to chromium

The registered medical practitioner may also choose to monitor a worker's exposure to chromium via urinary chromium level. Where urine analysis is carried out, the following values should be considered when assessing exposure to inorganic chromium:

Biological level	Source
10 µmol chromium/mol creatinine in urine = 5 µg/L	Workcover NSW Biological Occupational Exposure Limit (BOEL) Committee.
	British Health and Safety Executive Biological Guidance Value
Urine samples should be taken either: ■ pre-shift and post-shift (to measure the increase in urinary chromium during the work shift); or ■ at the end of a work shift at the end of the work week.	

Where there is an increase in the pre-shift and post-shift urinary chromium (increase during shift) of more than 10 µg/L[1] or where the end of shift at end of work week urinary chromium is more than 25µg/L, a repeat urinary chromium should be done and work practices should be reviewed. The half life for chromium in urine is 15-41 hours and should be taken into account when interpreting the results.

1 ACGIH Biological Exposure Indices 2007

These levels correspond to the TWA for Chromium VI of 0.05 mg/m^3 for an eight hour shift in workers with chronic chromium exposure. Given the same level of air exposure the concentration of chromium in newly exposed workers is expected to be lower (7 µg/L) and the increase during the workshift 5 µg/L. Note that the concentration of chromium in urine in pre-shift samples reflects past exposure, whereas the post-shift sample values reflect both past and current exposures.

These levels do not protect against acute irritation exposure (nasal chrome ulcers) where exposure is to water soluble Cr VI mist like in electroplating.

Biological monitoring results greater than the above values mean workplace exposure is not being adequately controlled and a review of control measures should be carried out immediately to reduce worker exposure. Although workers with high biological monitoring results may not be showing signs of ill health, affected workers should be removed from further potential exposure until controls are improved.

Analysis of chromium in red blood cells has been proposed by some as a specific marker for chromium (VI) exposure. However, interpretation of the test results can be problematic as variability in the analysis and inter-individual variability has been reported.

AT TERMINATION OF WORK IN A CHROMIUM (INORGANIC) PROCESS

8. Final medical examination

The final medical examination will include urinary chromium testing and a physical examination by the medical practitioner.

SUPPLEMENTARY INFORMATION ON CHROMIUM (INORGANIC)

Chromium exists in a series of oxidation states from 0 valence to 6+. The most important stable states are elemental metal (Cr^0), trivalent (Cr^{3+}) and hexavalent (Cr^{6+}).

9. 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, for example, copper chrome arsenic
- chromate use in the textile industry
- chrome pigment use, for example in paints.

10. Non-work sources

Chromium (III) is a naturally-occurring element found in rocks, plants and soil. In urban areas, chromium is in the air from fossil fuel combustion. Chromium is an essential nutrient and foods high in chromium include green beans, broccoli and high bran breakfast cereals. The average adult ingestion is 50–200 µg/day.

POTENTIAL HEALTH EFFECTS FOLLOWING EXPOSURE TO INORGANIC CHROMIUM

The adverse effects of chromium and its inorganic compounds vary according to valence state, 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.

11. Route of entry into the body

The routes of inorganic chromium entry into the body are through inhalation, ingestion and percutaneous absorption. Work-related exposure generally occurs through inhalation and dermal contact. The absorption of chromium is dependent on the valence and water-solubility of the chromium compound. Soluble forms of hexavalent chromium are readily absorbed by inhalation. Dermal absorption may also occur. Absorption of water-soluble hexavalent chromium through the gastrointestinal tract is about 10 per cent.

12. Target organ/effect

Skin irritant – irritant contact dermatitis, skin and mucous ulcerations, perforation of nasal septum.

Skin sensitivity – allergic contact dermatitis.

Respiratory tract – irritation, allergic asthma.

Gastrointestinal tract – irritation.

Kidney – renal tubule damage.

Carcinogen – for hexavalent chromium compounds there is an increase in the risk of lung cancer and case reports of sinonasal cancer.

13. Acute and chronic effects

HEXAVALENT CHROMIUM

Irritant and corrosive effects

Chromium (VI) (aerosols, dusts, liquid) 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 pulmonary oedema. Also acute oral Cr(VI) toxicity is probably a result of bleeding due to irritation and corrosion (gastroenteritis, hepatic necrosis, acute tubular necrosis with renal failure).

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 (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 Cr(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 [1]. Chromate sensitivity, once induced, may prove difficult to deal with in multiple settings and is very persistent once developed.

Cr(VI) penetrates undamaged skin (the ulcer does not seem to bear a relationship to the development of allergic sensitisation) and subsequently combines with proteins. Contact hypersensitivity due to chromium compounds is caused by a direct effect as haptene into the skin, where chromium is conjugated with autologous proteins to form a full antigen.

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. This results in generalised bronchospasm and typical asthmatic attacks occurring on subsequent low exposure levels to dusts, aerosols or welding fumes.

SYSTEMIC EFFECTS

Chronic obstructive pulmonary disease

Prolonged inhalation of Cr(VI) particulates can cause chronic respiratory irritation with hyperaemia, chronic inflammation of the lung, chronic bronchitis, bronchopneumonia, and emphysema. The effect on respiratory function could be a reduction in FEV_1[2] and maximal expiratory flow. There is a possibility of complication in the form of an infection.

Studies of welders and chromium platers have shown workers exposed to high levels of chromium show damage to renal tubules. Chronic chromium exposure results in transient renal effects. Nephrotoxicity is the primary cause of death from acute dermal exposure.

Acute chromium exposures can result in hepatic necrosis. Limited data indicate chronic exposure to chromium compounds can cause hepatic effects.

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. In animal studies, inhalation of trivalent chromium compounds has affected the respiratory system, for example in a study with rabbits there was a decrease in functional and metabolic activity of the macrophage.

14. Carcinogenicity

There is considerable epidemiological evidence exposures to hexavalent chromium compounds of sparing to high solubility in chromate production, chromium plating and zinc chromate pigment manufacture have led to a clear excess in mortality from lung cancer.

2 Forced expiratory volume in one second

The International Agency for Research on Cancer's [2] classification for hexavalent chromium compounds is Group 1. According to the International Agency for Research on Cancer, this category is used only when there is sufficient evidence of carcinogenicity in humans. Carcinogenesis may result from the formation of mutagenic oxidative DNA lesions consequential to the intracellular reduction to the trivalent form. Carcinogenicity by the oral route of exposure cannot be determined.

Metallic chromium and trivalent compounds have an International Agency for Research on Cancer classification of Group 3, and mutagenicity and epidemiological data do not rule out carcinogenic activity of trivalent compounds. There is also discussion in the literature on the carcinogenic potential of trivalent salts and insoluble chromium compounds which appear to accumulate in human lung tissue after inhalation [3].

Cases of sinonasal cancer have been reported in epidemiological studies of chromate production, chromate pigment production and chromium platers.

15. Carcinogen, germ cell mutagen and reproductive toxicant classifications

The following are some chromium-containing chemicals with GHS 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 AnnexVI: 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
- A 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.

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.

REFERENCED DOCUMENTS

1. Baruthio F, 'Toxic Effects of Chromium and its Compounds', *Biological Trace Element Research,* vol 32, pp 145-53, 1992.
2. International Agency for Research on Cancer, *IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans, Volume 49: Chromium, Nickel and Welding,* International Agency for Research on Cancer, Lyon, 1990.
3. Norseth T, 'The Carcinogenicity of Chromium and its Salts—editorial', *British Journal of Industrial Medicine,* vol 43, pp 649-51, 1986.

FURTHER READING

Agency for Toxic Substances and Disease Registry, *Toxicological Profile for Chromium*, Agency for Toxic Substances and Disease Registry, United States Department of Health and Human Services, Public Health Service, Atlanta, 2000.

American Conference of Governmental Industrial Hygienists (ACGIH), *Documentation of the Threshold Limit Values and Biological Exposure Indices*, 7th Ed, Cincinnati, 2011.

Rosenman KD, Stanbury M, 'Risk of Lung Cancer Among Former Chromium Smelter Workers', *American Journal of Industrial Medicine*, vol 29(5), pp 491-500, 1996.

This health monitoring report is a confidential health record and must not be disclosed to another person except in accordance with the Work Health and Safety Regulations or with the consent of the worker.

There are two sections. Complete both sections and all questions if applicable.

Section 1 is to be forwarded to the PCBU who has engaged your services. A copy of laboratory report(s) must be attached > > > >

Section 2 may contain confidential information which may not be relevant to the health monitoring program being carried out. This section should be retained by the medical practitioner. Information which is required to be given to the PCBU should be summarised in part 7 of section 1.

SECTION 1 – THIS SECTION TO BE RETURNED TO THE PCBU			
1. PERSON CONDUCTING A BUSINESS OR UNDERTAKING			
Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
2. OTHER BUSINESSES OR UNDERTAKINGS ENGAGING THE WORKER			
Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
3. WORKER		(✓) all relevant boxes	
Surname:		Given names:	
Date of birth: DD/MM/YYYY	Sex:	☐ Male	☐ Female
Address:			
Suburb:			Postcode:
Current Job:	Tel(H):		Mob:
Date started employment : DD/MM/YYYY			
4. EMPLOYMENT IN CHROMIUM (INORGANIC) RISK WORK		(✓) all relevant boxes	
1. ☐ New to chromium (inorganic) work			
2. ☐ New worker but not new to chromium (inorganic) work			
3. ☐ Current worker continuing in chromium (inorganic) work			
4. Worked with chromium (inorganic) since DD/MM/YYYY			
5. Satisfactory personal hygiene (for example nail biting, frequency of hand washing)		☐ Yes	☐ No
6. Risk assessment completed		☐ Yes	☐ No

5. WORK ENVIRONMENT ASSESSMENT		(✓) all relevant boxes	
Date of assessment: DD/MM/YYYY			
Chromium (Inorganic) Industry			
☐ Welding/Fabrication	**Controls:**		
☐ Chrome plating	Wear gloves	☐ Yes	☐ No
☐ Refractory Production	Respirator use	☐ Yes	☐ No
☐ Concreting	Local exhaust ventilation	☐ Yes	☐ No
☐ Leather Industry	Overalls / work clothing	☐ Yes	☐ No
☐ Timber preservation	Laundering by employer	☐ Yes	☐ No
☐ Textile Industry	Wash basins & showers (with hot & cold water)	☐ Yes	☐ No
☐ Chrome Pigment Manufacture/Use (e.g. in paints)	Smoking or eating in workshop	☐ Yes	☐ No
☐ Other (specify):	Weekly inspection of skin on hands/arms	☐ Yes	☐ No
	Personal hygiene:		
	Clean Shaven	☐ Yes	☐ No
	Shower & change into clean clothes at end of shift	☐ Yes	☐ No

6. BIOLOGICAL MONITORING RESULTS Include at least the previous two test results (if available)		
Date	**Tests performed**	**Recommended Action and/or Comment**
1. DD/MM/YYYY		
2. DD/MM/YYYY		
3. DD/MM/YYYY		
4. DD/MM/YYYY		
5. DD/MM/YYYY		
6. DD/MM/YYYY		
7. DD/MM/YYYY		
8. DD/MM/YYYY		

7. RECOMMENDATIONS (by Medical Practitioner) (✓) all relevant boxes		
1. ☐ Suitable for work with chromium (inorganic)		
2. ☐ Counselling required		
3. ☐ Review workplace controls		
4. ☐ Repeat health assessment in _____ month(s) / _____ week(s)		
5. ☐ Removal from work with chromium (inorganic)	On DD/MM/YYYY	
6. ☐ Medical examination by Medical Practitioner	On DD/MM/YYYY	
7. ☐ Fit to resume work	From DD/MM/YYYY	
8. ☐ Referred to Medical Specialist (respiratory/dermatology/other):	On DD/MM/YYYY	
Specialist's name:		
Additional comments or recommendations arising from health monitoring:		
Medical Practitioner (responsible for supervising health monitoring)		
Name:	Signature	Date: DD/MM/YYYY
Tel:	Fax:	Registration Number:
Medical Practice:		
Address:		
Suburb:		Postcode:

SECTION 2 – THIS SECTION TO BE RETAINED BY THE MEDICAL PRACTITIONER			
1. PERSON CONDUCTING A BUSINESS OR UNDERTAKING			
Company / Organisation name:			
Site address:			
Suburb:		Postcode:	
Site Tel:	Site Fax:	Contact Name:	
2. OTHER BUSINESSES OR UNDERTAKINGS ENGAGING THE WORKER			
Company / Organisation name:			
Site address:			
Suburb:		Postcode:	
Site Tel:	Site Fax:	Contact Name:	
3. WORKER		(✓) all relevant boxes	
Surname:		Given names:	
Date of birth: DD/MM/YYYY	Sex:	☐ Male	☐ Female ☐ Pregnant/Breast Feeding?
Address:			
Suburb:		Postcode:	
Current Job:	Tel(H):	Mob:	
Date started employment : DD/MM/YYYY			
4. GENERAL HEALTH ASSESSMENT (if applicable)			
Symptoms of:	**Comments**	**Further testing?**	
Skin disorders		☐ Yes	☐ No
Headaches, dizziness		☐ Yes	☐ No
Respiratory tract/GIT		☐ Yes	☐ No
Irritation of eyes, nose or throat		☐ Yes	☐ No
CNS		☐ Yes	☐ No
Others		☐ Yes	☐ No
Height _____cm Weight _____kg Bp ____/____ mmHg		☐ Yes	☐ No

5. OTHER MEDICAL HISTORY, FAMILY MEDICAL HISTORY, CURRENT MEDICATION, COMMENTS, TESTS OR RECOMMENDATIONS (use separate sheet if necessary)

Medical Practitioner (responsible for supervising health monitoring)		
Name:	Signature	Date: DD/MM/YYYY

Tel:	Fax:	Registration Number:
Medical Practice:		
Address:		
Suburb:		Postcode:

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CREOSOTE

BASELINE HEALTH MONITORING BEFORE STARTING WORK IN A CREOSOTE PROCESS

Workers must be informed of the potential health effects associated with exposure to creosote. In particular, workers should be aware of the occurrence and recognition of photosensitivity and skin changes, and the need to report them to the registered medical practitioner as soon as possible, even if they occur between regular monitoring.

1. **Collection of demographic data**
2. **Work history**
3. **Medical history**
4. **Physical examination**

A physical examination will be conducted, with emphasis on the neurological system. A thorough examination of all skin will also be conducted, including the scrotum, noting any abnormal lesions, in particular, squamous cell carcinoma and hyperkeratosis. These should be recorded on a body outline form showing both front and back views and noting size.

DURING EXPOSURE TO A CREOSOTE PROCESS

5. **Photosensitivity**

Photosensitivity is a known symptom of exposure to creosote. Where workers report photosensitivity, an appointment should be arranged with the medical practitioner and workers should receive additional counselling on the potential health effects of creosote on the skin.

Where a worker is diagnosed with photosensitivity or other health effects related to exposure, the health monitoring report should recommend that the person conducting a business or undertaking must review control measures and carry out recommended remedial action.

6. **Physical examination**

A physical examination will be conducted annually with emphasis on the neurological system and skin. Evidence of skin sensitisation will be noted.

7. **Data for inclusion in health records**

Records of photosensitivity which a worker has had, indicating specific processes involved should be included in the worker's health monitoring report.

8. **Assessing exposure to creosote [1]**

The assessment of work-related exposure to creosote is difficult because workers are exposed to a mixture of compounds. However, polycyclic aromatic hydrocarbons (PAH) and alkylated PAHs are a significant proportion of creosote and the registered medical practitioner may choose to assess exposure to PAH through urine analysis. The metabolite of pyrene, 1-hydroxypyrene (1-HP) in urine, is most often used as the biomarker for PAH exposure as pyrene is a very thermodynamically stable compound and therefore most abundant in a PAH mixture.

Where urine analysis is performed, the following values should be considered when assessing exposure to PAH:

Biological level	Source
1 µg 1-HP/L urine	Workcover NSW Biological Occupational Exposure Limit (BOEL) Committee. *Note: this value is under review.*
< 0.3 µg 1-HP/L urine – unexposed non-smokers 0.5 µg 1-HP/L urine – median for non-exposed smokers 1 µg 1-HP/L urine (benchmark value)	American Conference of Governmental Industrial Hygienists (ACGIH)
4 µmol 1-HP/mol creatinine in urine (benchmark guidance value)	Health and Safety Executive (UK)

Where results of urine testing indicate there may be high workplace exposure to PAH (creosote), the registered medical practitioner should consider recommending:

- the worker should be removed from creosote work
- the PCBU should review control measures and carry out recommended remedial actions
- the worker must be informed of the results of the health monitoring.

Other information

Measurement of airborne levels of PAH fails to take into account the potential pathway of skin absorption, which can contribute significantly to the total internal dose. The levels of 1-hydroxypyrene in the urine can increase during the course of a workday, reaching maximum values three to nine hours after the end of exposure. If the contribution of dermal exposure is important, post-shift 1-hydroxypyrene excretion can be lower than pre-shift levels when the worker has been exposed to PAH on the day prior to sampling. The difference between beginning and end of workweek excretion gives an indication of the average exposure over the work week.

Note: Other hydroxylated metabolites of PAH have been proposed as markers of PAH exposure, however, currently correlation between metabolite levels and exposure have not been determined.

AT TERMINATION OF WORK IN A CREOSOTE PROCESS

9. Final medical examination

A final medical examination will be conducted and will include a physical examination with emphasis on the neurological system and skin, noting abnormal lesions and evidence of skin sensitisation.

10. Continuing medical monitoring

Workers with a history of skin disease due to contact with creosote should be advised to seek continuing medical monitoring.

SUPPLEMENTARY INFORMATION ON CREOSOTE

11. What is creosote?

Creosote is the name used for a variety of products that are mixtures of many chemicals. Creosotes are created by high-temperature treatment of beech and other woods (beechwood creosote) or coal (coal tar creosote). Creosote prepared from coal tar is the most common form of creosote in the workplace. Creosote is a mixture of several hundred chemicals but only a limited number are present in amounts of more than one per cent. There are six major classes of compounds in creosote: aromatic hydrocarbons, including polycyclic aromatic hydrocarbons (PAHs) and alkylated PAHs (which can constitute up to 90 per cent of creosote); tar acids/phenolics; tar bases/nitrogen-containing heterocycles; aromatic amines; sulphur-containing heterocycles; and oxygen-containing heterocycles, including dibenzofurans. Generally, phenolic compounds, low-molecular-weight PAHs, and some heterocycles tend to be predominantly in the gaseous phase. Creosote constituents may also occur in the atmosphere as particulate matter.

Coal tars are by-products of the high temperature treatment of coal to produce coke or natural gas. Coal tar creosote is a distillation product of coal tar and is a thick, oily liquid that is typically amber to black in colour. Coal tar pitch is a residue produced during the distillation of coal tar and is usually thick, black or dark brown liquid or semisolid with a smoky or aromatic odour. Coal tar pitch volatiles are compounds given off from coal tar pitch when it is heated. Coal tar creosote, coal tar and coal tar pitch are mixtures of similar compounds and are rarely formed in nature.

12. 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. Most data are available for wood-preserving workers. 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.

13. Non-work sources

Coal tar shampoos for psoriasis and anti-dandruff therapy, coal tar ointments for treatment of eczematous dermatitis and contaminated groundwater near creosote waste sites. Aquatic invertebrates and fish bioaccumulate creosote components. Transfer to the human food supply is possible via contaminated seafood.

POTENTIAL HEALTH EFFECTS FOLLOWING EXPOSURE TO CREOSOTE

14. Route of entry into the body

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

15. Target organ/effect

Skin – irritation, blistering, hyperpigmentation, warts, photosensitivity, cancer.

CNS – depression, weakness, headache, vertigo, nausea, confusion, convulsions.

Respiratory tract – irritation.

Eyes – irritation, chemical burns, corneal damage.

16. Photosensitivity

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 [2].

Photosensitisation of the skin and eyes can be caused by exposure to specific industrial chemicals. The skin can be affected by dermal exposure or 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 [2].

17. Acute effects

Creosote has been involved in incidental or accidental poisoning incidents, mainly due to its use as a pesticide. Deaths occurred following ingestion of about 1 to 2 g (children) or about 7 g (adults). Symptoms included salivation, vomiting, respiratory difficulties, vertigo, headache, loss of pupillary reflexes, hypothermia, cyanosis, convulsion accompanied by oropharyngeal, intestinal, pericardial, liver and kidney damage [3].

Contact with creosote or creosote vapour may cause irritation of the skin. The skin may become red, papular, vesicular 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.

One or more of the following effects may be evident on short-term exposure to high concentrations of creosote:

- **systemic** – nausea and vomiting, diarrhoea, anorexia and difficulty in swallowing, salivation, abdominal discomfort, respiratory distress, cyanosis, pupillary changes, convulsive movements, rapid pulse or vascular collapse
- **neurological** – headaches, fainting, vertigo and mental disturbances.

18. Chronic exposure

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

19. Carcinogenicity [3]

Increased risks of developing lip and skin cancers have been observed in cohort studies of Swedish and Norwegian wood impregnators and in Finnish round timber workers. A cohort study examining 922 Swedish and Norwegian wood impregnators from 13 plants (for example railroad cross ties and telegraph poles) found a standardized incidence ratio (SIR) of 250 for lip cancers and an SIR of 237 for non-melanoma skin cancer.

The risk increased with the latency; analysis by duration of exposure was not provided. According to the authors, the significantly elevated risk for lip and skin cancer could probably be attributed to the combination of exposure to creosote and sunlight [4]. In a population-based record linkage study in Finland, elevated risks for lip cancer, SIR = 306, and non-melanoma skin cancer, SIR = 464, were found for round-timber workers [5]; the mortality for cancer of the scrotum was elevated among brick makers exposed to creosote. Prolonged skin exposure to soot and coal tar creosote has been associated with cancer of the scrotum in chimney sweeps.

Single epidemiological studies suggested a possible risk for bladder cancer, multiple myeloma, and lung cancer due to exposure to creosote. Two case-control studies suggested an increased risk of brain tumours and neuroblastoma among offspring of male workers with possible creosote exposure.

All of the epidemiological studies were based on qualitative estimations of exposure rather than on measurements. There is consistent evidence from human studies that creosote causes skin cancer, but the studies do not allow dose-response analysis.

20. Carcinogen classification

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

REFERENCED DOCUMENTS

1. Lauwerys RR, Hoet P, *Industrial Chemical Exposure Guidelines for Biological Monitoring*, 3rd edition, Lewis Publishers, Boca Raton, 2001.
2. Australian Safety and Compensation Council, *Guidance Note for the Protection of Workers from the Ultraviolet Radiation in Sunlight*, Australian Safety and Compensation Council, Canberra, 2008.
3. World Health Organisation/International Program on Chemical Safety, *Concise International Chemical Assessment Documents* (CICAD) 62, WHO Geneva, 2004. www.inchem.org
4. Karlehagen S, Andersen A, Ohlson C, 'Cancer incidence among creosote-exposed workers', *Scandinavian Journal of Work, Environment and Health*, vol 18, pp 26–29, 1992.
5. Pukkala E, *Cancer Risk by Social Class and Occupation: A Survey of 109,000 Cancer Cases among Finns of Working Age*, Karger, Basel, 1995.

FURTHER READING

Agency for Toxic Substances and Disease Registry, *Toxicological Profile for Creosote*, Agency for Toxic Substances and Disease Registry, US Department of Health & Human Services, Public Health Service, Atlanta, 2002.

International Agency for Research on Cancer, *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Volume 92: Some Non-heterocyclic Polycyclic Aromatic Hydrocarbons and Some Related Exposures*, International Agency for Research on Cancer, Lyon, 2010.

Worksafe Australia, *Occupational Diseases of the Skin*, Australian Government Publishing Service, Canberra, 1990.

This health monitoring report is a confidential health record and must not be disclosed to another person except in accordance with the Work Health and Safety Regulations or with the consent of the worker.

There are two sections. Complete both sections and all questions if applicable.

Section 1 is to be forwarded to the PCBU who has engaged your services. A copy of laboratory report(s) must be attached > > > >

Section 2 may contain confidential information which may not be relevant to the health monitoring program being carried out. This section should be retained by the medical practitioner. Information which is required to be given to the PCBU should be summarised in part 7 of section 1.

SECTION 1 – THIS SECTION TO BE RETURNED TO THE PCBU			
1. PERSON CONDUCTING A BUSINESS OR UNDERTAKING			
Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
2. OTHER BUSINESSES OR UNDERTAKINGS ENGAGING THE WORKER			
Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
3. WORKER		(✓) all relevant boxes	
Surname:		Given names:	
Date of birth: DD/MM/YYYY	Sex:	☐ Male	☐ Female
Address:			
Suburb:			Postcode:
Current Job:	Tel(H):		Mob:
Date started employment : DD/MM/YYYY			
4. EMPLOYMENT IN CREOSOTE RISK WORK		(✓) all relevant boxes	
1. ☐ New to creosote work			
2. ☐ New worker but not new to creosote work			
3. ☐ Current worker continuing in creosote work			
4. Worked with creosote since DD/MM/YYYY			
5. Satisfactory personal hygiene (for example nail biting, frequency of hand washing)		☐ Yes	☐ No
6. Risk assessment completed		☐ Yes	☐ No

5. WORK ENVIRONMENT ASSESSMENT		(✓) all relevant boxes	
Date of assessment: DD/MM/YYYY			
Creosote Industry			
☐ Coal Distillation	**Controls:**		
☐ Timber Preservation	Wear gloves	☐ Yes	☐ No
☐ Rubber/Tyre Industry	Respirator use	☐ Yes	☐ No
☐ Pesticide/Insecticide/ Fungicides	Local exhaust ventilation	☐ Yes	☐ No
☐ Marine Piling Construction	Overalls / work clothing	☐ Yes	☐ No
☐ Other (specify):	Laundering by employer	☐ Yes	☐ No
	Wash basins & showers (with hot & cold water)	☐ Yes	☐ No
	Smoking or eating in workshop	☐ Yes	☐ No
	Personal hygiene:		
	Clean Shaven	☐ Yes	☐ No
	Shower & change into clean clothes at end of shift	☐ Yes	☐ No

6. BIOLOGICAL MONITORING RESULTS Include at least the previous two test results (if available)		
Date	**Tests performed**	**Recommended Action and/or Comment**
1. DD/MM/YYYY		
2. DD/MM/YYYY		
3. DD/MM/YYYY		
4. DD/MM/YYYY		
5. DD/MM/YYYY		
6. DD/MM/YYYY		
7. DD/MM/YYYY		
8. DD/MM/YYYY		

7. RECOMMENDATIONS (by Medical Practitioner) (✓) all relevant boxes
1. ☐ Suitable for work with creosote
2. ☐ Counselling required
3. ☐ Review workplace controls
4. ☐ Repeat health assessment in _____ month(s) / _____ week(s)

5. ☐ Removal from work with creosote	On DD/MM/YYYY
6. ☐ Medical examination by Medical Practitioner	On DD/MM/YYYY
7. ☐ Fit to resume work	From DD/MM/YYYY
8. ☐ Referred to Medical Specialist (respiratory/dermatology/other):	On DD/MM/YYYY
Specialist's name:	
Additional comments or recommendations arising from health monitoring:	

Medical Practitioner (responsible for supervising health monitoring)		
Name:	Signature	Date: DD/MM/YYYY
Tel:	Fax:	Registration Number:
Medical Practice:		
Address:		
Suburb:		Postcode:

SECTION 2 – THIS SECTION TO BE RETAINED BY THE MEDICAL PRACTITIONER			
1. PERSON CONDUCTING A BUSINESS OR UNDERTAKING			
Company / Organisation name:			
Site address:			
Suburb:		Postcode:	
Site Tel:	Site Fax:	Contact Name:	
2. OTHER BUSINESSES OR UNDERTAKINGS ENGAGING THE WORKER			
Company / Organisation name:			
Site address:			
Suburb:		Postcode:	
Site Tel:	Site Fax:	Contact Name:	
3. WORKER		(✓) all relevant boxes	
Surname:		Given names:	
Date of birth: DD/MM/YYYY	Sex: ☐ Male	☐ Female	☐ Pregnant/Breast Feeding?
Address:			
Suburb:		Postcode:	
Current Job:	Tel(H):	Mob:	
Date started employment : DD/MM/YYYY			
4. GENERAL HEALTH ASSESSMENT (if applicable)			
Symptoms of:	**Comments**	**Further testing?**	
Skin disorders		☐ Yes	☐ No
Headaches, dizziness		☐ Yes	☐ No
Respiratory disorders		☐ Yes	☐ No
Irritation of eyes, nose or throat		☐ Yes	☐ No
Cough		☐ Yes	☐ No
CNS		☐ Yes	☐ No
Others		☐ Yes	☐ No
Height _____cm Weight _____kg Bp _____/_____ mmHg		☐ Yes	☐ No

5. OTHER MEDICAL HISTORY, FAMILY MEDICAL HISTORY, CURRENT MEDICATION, COMMENTS, TESTS OR RECOMMENDATIONS (use separate sheet if necessary)		
Medical Practitioner (responsible for supervising health monitoring)		
Name:	Signature	Date: DD/MM/YYYY
Tel:	Fax:	Registration Number:
Medical Practice:		
Address:		
Suburb:		Postcode:

CRYSTALLINE SILICA

BASELINE HEALTH MONITORING BEFORE STARTING WORK IN A CRYSTALLINE SILICA PROCESS

1. **Collection of demographic data**
2. **Work history**
3. **Medical history**

Administration of a standardised respiratory questionnaire. Two examples are the international Union Against Tuberculosis' *Bronchial Symptoms Questionnaire 1986* [1] **or** the Medical Research Council's *Questionnaire on Respiratory Symptoms 1986* [2].

4. **Physical examination**

A physical examination will be conducted with emphasis on the respiratory system.

5. **Investigation**

The following tests will be used to test the worker's baseline exposure:

- standardised respiratory function tests* to be performed. The tests are FEV_1[1], FVC[2] and FEV_1/FVC[3]. The norms for predictive values should be stated.
- chest X-ray, full size PA view. Report to be recorded according to current International Labour Organisation classification.

Note: In order to reduce radiation exposure the frequency of chest X-ray should be minimised. There is potential for excessive X-rays with a workforce that changes employers frequently. Protocols have been reviewed recently by the United Kingdom HSE, see http://www.hse.gov.uk/research/rrpdf/rr827.pdf, and there is a general consensus for annual assessment with respiratory questionnaire and lung function tests to look for lung function changes over time.

DURING EXPOSURE TO A CRYSTALLINE SILICA PROCESS

6. **Monitoring exposure to crystalline silica**

A medical examination should be conducted annually and will include:

- work history
- medical history
- physical examination
- lung function investigation consisting of standardised respiratory function tests and, if required, a chest X-ray.

* Spirometry equipment should be calibrated regularly according to a standard protocol.

1. Forced expiratory volume in one second

2. Forced vital capacity

3. Tiffeneau index

The Michigan State University have developed the following protocol [3]:

- **Chest X-ray**. Every 5-10 years in first 20 years of work unless the air levels are above the exposure standard. In order to reduce radiation exposure, the frequency of chest X-rays should be minimised. (Note: be aware of the potential for excessive X-rays where the worker has worked for multiple employers, particularly in the construction industry). An abnormal X-ray or 20-years exposure or more warrants X-rays on a more frequent basis.
- **Pulmonary function tests**. Performed as a baseline and annually. Individuals with progressive decreases in pulmonary function beyond that normally associated with age [4,5] should be closely followed up about the aetiology of the pulmonary function decrement.

The Australian Institute of Occupational Hygienists (AIOH) [6] recommends "where there is a continued likelihood of 50 per cent of the exposure standard being exceeded, exposure monitoring and health surveillance should apply. To overcome limitations in analytical sensitivity, full shift monitoring and the use of a NATA (National Association of Testing Authorities) registered laboratory is recommended."

AT TERMINATION OF WORK IN A CRYSTALLINE SILICA PROCESS

7. Final medical examination

A final medical examination will be conducted and will include:

- medical history
- physical examination
- investigation.

SUPPLEMENTARY INFORMATION ON CRYSTALLINE SILICA

8. Work activities that may represent a high risk exposure

Silica is silicon dioxide, a naturally occurring widely abundant mineral that forms the major component of most rocks and soils. There are non-crystalline and crystalline forms of silicon dioxide. Crystalline silica is also known as free silica. Crystalline silica dust particles which are small enough to penetrate deep into the lung are termed respirable. Respirable crystalline silica may cause lung damage. The non-crystalline form of silica does not cause this kind of lung damage.

The main form of crystalline silica is quartz. Granite contains 25 per cent to 40 per cent quartz, shales average 22 per cent and sandstones average 67 per cent quartz. Quartz is the major component of sand in locations like stream beds, beaches and deserts. Other polymorphs of silicon dioxide, like cristobalite and tridymite are less common. 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.

POTENTIAL HEALTH EFFECTS FOLLOWING EXPOSURE TO CRYSTALLINE SILICA

9. Route of entry into the body

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

10. Target organ/effect

Lungs – silicosis, International Agency for Research on Cancer (IARC) Group 1 Carcinogen for lung cancer, chronic obstructive pulmonary disease.

Kidneys – epidemiologic data emerging that silica causes renal disease.

Airborne crystalline silica can bio-accumulate in the lungs and cause disease of the respiratory system.

Large bio-accumulated loads of crystalline silica in the lung substance (or lung parenchyma) can cause a build up of connective tissue, which is termed silicosis, a specific form of pneumoconiosis. Silicosis is an irreversible and progressive condition. Early silicosis may have no untoward effects. However, severe forms can result in poor gas exchange, difficulty in breathing and death. Evidence suggests crystalline silica interacts with other respiratory hazards, like tobacco smoke, to cause airway diseases.

Silicosis virtually always requires prolonged exposure to substantial airborne quantities of respirable crystalline free silica. Four clinical patterns of diffuse lung disease may be seen with silicosis: simple nodular silicosis, progressive massive fibrosis, accelerated silicosis, and acute silicosis or silicoproteinosis.

The AIOH supports the workplace exposure standard of 0.1 mg/m^3 for respirable crystalline silica.

However, a “no observable adverse effects level” (NOAEL) cannot be demonstrated. Risks to health are occurring at levels previously thought to be acceptable. Limitations in technology make it difficult to determine a NOAEL if it occurs at very low levels of exposure [6].

Factors thought to influence the potential for respirable crystalline silica to cause silicosis include the following [6]:

- polymorphic type of crystalline silica with cristobalite, tridymite and quartz appearing more reactive and cytotoxic than coesite and shishovite
- presence of other minerals, for example aluminium containing materials reduces the toxic effect of quartz, however, this may only be a temporary effect
- total surface area which is related to particle number, size and surface area of individual particles. Smaller particle size fractions would be expected to cause more lung damage than larger size fractions

- freshly fractured versus 'aged' surfaces. Increased cytotoxicity occurs when crystalline silica particles are cleaved into smaller fragments with reactive free radical species forming on the surface of the particles. There is an 'aging' process where free radical activity decays with time. This occurs slowly in air but rapidly in water.

11. Acute effects

Acute silicosis occurs after a short exposure to very high levels of silica and the alveolar spaces fill with a lipid and proteinaceous exudate. This could occur in exposure in confined spaces where respiratory protection is not worn. The condition causes rapidly progressive dyspnoea and death, usually within months of onset. Workers with acute silicosis may be expected to have a largely restrictive functional abnormality with gas exchange abnormalities.

12. Chronic effects

Simple silicosis is the most common pattern with a profusion of small rounded opacities less than 1 cm in diameter throughout the lung fields but predominantly in the upper lobes of the lung. Hilar lymph nodes may be prominent and calcification can be seen. Pulmonary function is usually well preserved. As silicosis progresses, the individual nodules enlarge and coalesce in a transition to progressive massive fibrosis.

Progressive massive fibrosis occurs as individual lesions conglomerate to form larger masses and emphysema develops in lung tissue as the conglomerate shrinks through fibrosis. Substantial impairment of pulmonary function occurs.

Accelerated silicosis is rare but can develop within two to five years with intense exposure to free silica [7].

Exposure to silica at levels that appear not to cause overt silicosis can cause chronic bronchitis and chronic obstructive airways disease. An increased susceptibility to tuberculosis occurs in workers with established silicosis. Epidemiological studies have revealed an excess prevalence of autoimmune disease like scleroderma, rheumatoid arthritis and systemic lupus erythematosus.

In the last 10 years several studies have linked crystalline silica with renal disease, particularly glomerulonephritis.

13. Carcinogenicity

Several work-related exposure studies indicate the crystalline silica is a potential human carcinogen, but provide 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 [6]. A number of epidemiologic studies from around the world have shown an increased risk for lung cancer among workers exposed to silica. In 1997, the International Agency for Research on Cancer (IARC) made the following evaluation: crystalline silica inhaled in the form of quartz or cristobalite from work-related sources is carcinogenic to humans (Group 1). IARC also noted that not all studies were consistent, and the carcinogenic potential of silica might be affected by the physical properties of the silica particles [8].

According to the IARC, this category is used when there is sufficient evidence of carcinogenicity in humans. In some circumstances, an agent may be placed in this category when evidence in humans is less than sufficient but there is sufficient evidence of carcinogenicity in experimental animals and strong evidence in exposed humans that the agent acts through a relevant mechanism of carcinogenicity.

The National Toxicology Program also concluded silica was a definite lung carcinogen in 2000 [9].

REFERENCED DOCUMENTS

1. Respiratory Disease Committee of the International Union Against Tuberculosis, *IUAT Bronchial Symptoms Questionnaire*, International Union Against Tuberculosis, 1986.
2. Medical Research Council Committee on Research into Chronic Bronchitis, *MRC Questionnaire on Respiratory Symptoms*, Medical Research Council, 1986.
3. Michigan State University, *Recommended Screening Protocol for Silica Exposed Workers*. http://www.oem.msu.edu/userfiles/file/Resources/Silica%20Screen%20Protocol.pdf
4. American College of Occupational and Environmental Medicine http://www.acoem.org/Guidelines.aspx search for *Spirometry in the Occupational Health Setting - 2011 Update*.
5. Fishwick D, Naylor S, 'COPD and the workplace. Is it really possible to detect early cases?', *Occupational Medicine*, vol 57, pp 82-84, 2007.
6. Australian Institute of Occupational Hygienists Inc., *AIOH Position Paper: Respirable Crystalline Silica and Occupational Health Issues,* AIOH, Feb 2009.
7. Centers for Disease Control and Prevention, Department of Health and Human Services, 'Current Trends Silicosis: Cluster in Sandblasters – Texas', *Morbidity and Mortality Weekly Review (MMWR)*, vol 39(25), pp 433-437, 1990.
8. International Agency for Research on Cancer, *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Volume 68: Silica, Some Silicates, Coal Dust and para-Aramid Fibrils*, International Agency for Research on Cancer, Lyon, 1997.
9. National Toxicology Program, *Silica, Crystalline (Respirable Size)*, in 9th Report on Carcinogens, United States Department of Health and Human Services, Public Health Service, North Carolina, 2000.

FURTHER READING

Health and Safety Executive (UK), *Health surveillance in Silica Exposed Workers*, Health and Safety Executive, London, 2010.

Hendrick D, Burge P, Beckett W, Churg A, *Occupational Disorders of the Lung: Recognition, Management, and Prevention,* WB Saunders, London, 2002.

Steenland K, 'One Agent, Many diseases: Exposure-Response Data and Comparative Risks of Different Outcomes Following Silica Exposure', *American Journal of Industrial Medicine*, vol 48(1), pp 16-23, 2005.

World Health Organisation/International Program on Chemical Safety, *Concise International Chemical Assessment Documents 24: Crystalline Silica, Quartz*, WHO, Geneva, 2000.

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SECTION 1 – THIS SECTION TO BE RETURNED TO THE PCBU			
1. PERSON CONDUCTING A BUSINESS OR UNDERTAKING			
Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
2. OTHER BUSINESSES OR UNDERTAKINGS ENGAGING THE WORKER			
Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
3. WORKER		(✓) all relevant boxes	
Surname:		Given names:	
Date of birth: DD/MM/YYYY	Sex:	☐Male	☐ Female
Address:			
Suburb:			Postcode:
Current Job:	Tel(H):		Mob:
Date started employment : DD/MM/YYYY			
4. EMPLOYMENT IN CRYSTALLINE SILICA RISK WORK (✓) all relevant boxes			
1. ☐ New to crystalline silica work			
2. ☐ New worker but not new to crystalline silica work			
3. ☐ Current worker continuing in crystalline silica work			
4. Worked with crystalline silica since DD/MM/YYYY			
5. Satisfactory personal hygiene (for example nail biting, frequency of hand washing)		☐ Yes	☐ No
6. Risk assessment completed		☐ Yes	☐ No

5. WORK ENVIRONMENT ASSESSMENT	(✓) all relevant boxes		
Date of assessment: DD/MM/YYYY			
Crystalline silica Industry			
☐ Excavation/Earth Moving	**Controls:**		
☐ Drilling Plant	Respirator use	☐ Yes	☐ No
☐ Clay/Stone Processing	Local exhaust ventilation (if indoors)	☐ Yes	☐ No
☐ Paving/Surfacing	Overalls / work clothing	☐ Yes	☐ No
☐ Mining	Laundering by employer	☐ Yes	☐ No
☐ Construction	Wash basins & showers (with hot & cold water)	☐ Yes	☐ No
☐ Abrasive Blasting	Wet handling methods used where possible	☐ Yes	☐ No
☐ Foundry Casting	**Personal hygiene:**		
☐ Other (specify):	Clean Shaven	☐ Yes	☐ No
	Shower & change into clean clothes at end of shift	☐ Yes	☐ No

6. BIOLOGICAL MONITORING RESULTS Include at least the previous two test results (if available)		
Date	**Tests performed**	**Recommended Action and/or Comment**
1. DD/MM/YYYY	**Last Chest X-Ray** DD/MM/YYYY	**Results: Normal / Abnormal** _____
2. DD/MM/YYYY	**Spirometry**	**Results:** FEV_1 _____; FVC _____; FEV_1/FVC _____
3. DD/MM/YYYY		
4. DD/MM/YYYY		
5. DD/MM/YYYY		
6. DD/MM/YYYY		
7. DD/MM/YYYY		
8. DD/MM/YYYY		

7. RECOMMENDATIONS (by Medical Practitioner) (✓) all relevant boxes		
1. ☐ Suitable for work with crystalline silica		
2. ☐ Counselling required		
3. ☐ Review workplace controls		
4. ☐ Repeat health assessment (including any tests) in _____ month(s) / _____ week(s) Specify tests to be repeated: _____		
5. ☐ Removal from work with crystalline silica		On DD/MM/YYYY
6. ☐ Medical examination by Medical Practitioner		On DD/MM/YYYY
7. ☐ Fit to resume work		From DD/MM/YYYY
8. ☐ Referred to Medical Specialist (respiratory/dermatology/other):		On DD/MM/YYYY
Specialist's name:		
Additional comments or recommendations arising from health monitoring:		

Medical Practitioner (responsible for supervising health monitoring)		
Name:	Signature	Date: DD/MM/YYYY
Tel:	Fax:	Registration Number:
Medical Practice:		
Address:		
Suburb:		Postcode:

SECTION 2 – THIS SECTION TO BE RETAINED BY THE MEDICAL PRACTITIONER			
This questionnaire also allows for recordings of a more general health assessment at the end, if applicable.			
1. PERSON CONDUCTING A BUSINESS OR UNDERTAKING			
Company / Organisation name:			
Site address:			
Suburb:		Postcode:	
Site Tel:	Site Fax:	Contact Name:	
2. OTHER BUSINESSES OR UNDERTAKINGS ENGAGING THE WORKER			
Company / Organisation name:			
Site address:			
Suburb:		Postcode:	
Site Tel:	Site Fax:	Contact Name:	
3. WORKER		(✓) all relevant boxes	
Surname:		Given names:	
Date of birth: DD/MM/YYYY	Sex:	☐ Male	☐ Female
Address:			
Suburb:		Postcode:	
Current Job:	Tel(H):	Mob:	
Date started employment : DD/MM/YYYY			

This questionnaire is based on the MRC (UK) Respiratory Questionnaire 1986, which has been extensively validated. This questionnaire is intended to be completed by an interviewer rather than by the patient. Additional questions have been added to cover clinical aspects of bronchial hyper-responsiveness validated by the Department of Occupational and Environmental Medicine, National Lung Institute[1].

The British Occupational Health Research Foundation (BOHRF)[2] concluded that in the clinical setting, questionnaires that identify symptoms of wheeze and/or shortness of breath which improve on days away from work or on holidays have a high sensitivity, but relatively low specificity for occupational asthma.

Preamble

I am going to ask some questions, mainly about your chest. I would like you to answer **yes** or **no** whenever possible.

If the subject is disabled from walking from a condition other than heart and lung disease, please begin questionnaire at **Question 5** and mark the adjacent box.

1 Venables KM, Farrer N, Sharp L, Graneek BJ, Newman Taylor AJ, 'Respiratory Symptoms Questionnaire for Asthma Epidemiology: Validity and Reproducibility', *Thorax*, vol 48, pp 214-219, 1993.

2 The British Occupational Health Research Foundation (BOHRF), *Guidelines for Prevention, Identification and Management of Occupational Asthma: Evidence Review and Recommendations*, London 2004. www.bohrf.org.uk

4. BREATHLESSNESS AND WHEEZING		
During the last month:		
1. Are you troubled by shortness of breath when hurrying on level ground or walking up a slight hill?	☐ Yes	☐ No
2. **If Yes to 1** - Do you get short of breath walking with other people of your age on level ground?	☐ Yes	☐ No
3. **If Yes to 2** - Do you have to stop for breath when walking at your own pace on level ground?	☐ Yes	☐ No
4. If you run, or climb stairs fast do you ever		
a. cough?	☐ Yes	☐ No
b. wheeze?	☐ Yes	☐ No
c. get tight in the chest?	☐ Yes	☐ No
5. Is your sleep ever broken		
a. by wheeze?	☐ Yes	☐ No
b. difficulty in breathing?	☐ Yes	☐ No
6. Do you ever wake up in the morning (or from your sleep if a shift worker)		
a. with wheeze?	☐ Yes	☐ No
b. difficulty with breathing?	☐ Yes	☐ No
7. Do you ever wheeze		
a. if you are in a smoky room?	☐ Yes	☐ No
b. if you are in a very dusty place?	☐ Yes	☐ No
8. **If Yes to either Q5, Q6, Q7** - Are your symptoms better		
a. at weekends (or equivalent if shift worker)?	☐ Yes	☐ No
b. when you are on holidays?	☐ Yes	☐ No
If **Yes to Question 8**, please record details of any occupational exposure to respiratory hazards e.g. isocyanates, wood dust, aluminium pot room or asbestos, in **Additional notes**.		
5. COUGH		
9. Do you usually cough first thing in the morning in winter?	☐ Yes	☐ No
10. Do you usually cough during the day/ or at night / in the winter?	☐ Yes	☐ No
11. **If Yes to Q9 or Q10** – Do you cough like this on most days for as much as three months each year?	☐ Yes	☐ No
6. PHLEGM		
12. Do you usually bring up phlegm from your chest first thing in the morning in winter?	☐ Yes	☐ No

13. Do you usually bring up any phlegm from your chest during the day / or at night / in winter?	☐ Yes	☐ No
14. **If Yes to Q12 or Q13** – Do you bring up phlegm like this on most days for as much as three months each year?	☐ Yes	☐ No
7. PERIODS OF COUGH AND PHLEGM		
15. In the past three years, have you had a period of (increased) cough and phlegm lasting for three weeks or more?	☐ Yes	☐ No
16. **If Yes to Q15** – Have you had more than one such episode?	☐ Yes	☐ No
8. CHEST ILLNESSES		
17. During the past three years, have you had any chest illness that has kept you from your usual activities for as much as a week?	☐ Yes	☐ No
18. **If Yes to Q17** – Did you bring up more phlegm than usual in any of these illnesses?	☐ Yes	☐ No
19. **If Yes to Q18** – Have you had more than one illness like this in the past three years?	☐ Yes	☐ No
9. PAST ILLNESSES		
20. Have you ever had, or been told that you have had any of the following?		
a. An injury, or operation affecting your chest?	☐ Yes	☐ No
b. Heart problems?	☐ Yes	☐ No
c. Bronchitis?	☐ Yes	☐ No
d. Pneumonia?	☐ Yes	☐ No
e. Pleurisy?	☐ Yes	☐ No
f. Asthma?	☐ Yes	☐ No
g. Other chest trouble?	☐ Yes	☐ No
h. Hay fever?	☐ Yes	☐ No
10. TOBACCO SMOKING		
21. Do you smoke?	☐ Yes	☐ No
If No to Q21		
22. Have you ever smoked as much as one cigarette a day for as long as one year?	☐ Yes	☐ No
23. How old were you when you started smoking regularly? _____		
24. a. Do (did) you smoke manufactured cigarettes?	☐ Yes	☐ No
If Yes to Q24a: How many do (did) you usually smoke per day? _____		
b. on weekdays? _____ c. at weekends? _____		

25. Do (did) you smoke any other forms of tobacco? ☐ Yes ☐ No **If Yes to Q25, record details under Additional notes**		
11. FOR EX-SMOKERS		
26. When did you give up smoking? Month _____ Year _____		
Additional notes:		
12. GENERAL HEALTH ASSESSMENT (if applicable)		
Symptoms of:	**Comments**	**Further testing?**
Skin disorders		☐ Yes ☐ No
Headaches, dizziness		☐ Yes ☐ No
Respiratory disorders		☐ Yes ☐ No
Irritation of eyes, nose or throat		☐ Yes ☐ No
Cough		☐ Yes ☐ No
CNS		☐ Yes ☐ No
Others		☐ Yes ☐ No
Height _____cm Weight _____kg Bp ____/___mmHg		☐ Yes ☐ No
13. OTHER MEDICAL HISTORY, FAMILY MEDICAL HISTORY, CURRENT MEDICATION, COMMENTS, TESTS OR RECOMMENDATIONS (use separate sheet if necessary)		
Medical Practitioner (responsible for supervising health monitoring)		
Name:	Signature	Date: DD/MM/YYYY
Tel:	Fax:	Registration Number:
Medical Practice:		
Address:		
Suburb:		Postcode:

ISOCYANATES

BASELINE HEALTH MONITORING BEFORE STARTING WORK IN AN ISOCYANATE PROCESS

Note: People with a history of asthma, atopic 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 a history of atopy or asthma does not preclude working with isocyanates. However, exposure to isocyanates is likely to cause respiratory irritation and may aggravate pre-existing asthma.

1. **Collection of demographic data**
2. **Work history**
3. **Medical history**

Administration of a standardised respiratory questionnaire. Two examples are the International Union Against Tuberculosis' *Bronchial Symptoms Questionnaire 1986* [1] **or** the Medical Research Council's *Questionnaire on Respiratory Symptoms 1986* [2].

4. **Physical examination**

A physical examination will be conducted, with emphasis on the respiratory system and skin.

5. **Investigation**

Standardised respiratory function tests* will be performed. The tests are FEV_1[1], FVC[2] and FEV_1/FVC[3]. The normal ranges for predicted values should be stated. A physical examination for work-related dermatitis should also be performed.

DURING EXPOSURE TO AN ISOCYANATE PROCESS

6. **Medical examination**

A medical examination should be performed at six weeks and then at six monthly intervals during continued exposure. Where monitoring after 12 months shows no adverse health effects the medical practitioner may choose to carry out annual monitoring. The medical examination will include:

- physical examination for work-related dermatitis
- standardised respiratory function tests.

There is no existing evidence pre- and post-shift changes in lung function are either sensitive or specific for the validation or exclusion of work-related asthma [3].

Note: the United Kingdom Health and Safety Executive (HSE) provides guidance for working with isocyanate paints in motor vehicle repair. For spray painters who are new workers, lung-function testing and a questionnaire are recommended at the beginning of work, after six weeks, twelve weeks and then yearly [4]. Also, skin checks for dermatitis should be conducted. Biological monitoring is recommended at least yearly and for new workers during the first few months as well as a check on control measures and working practices.

- * Spirometry equipment should be calibrated regularly according to a standard protocol.
- 1 Forced expiratory volume in one second
- 2 Forced vital capacity
- 3 Tiffeneau index

7. Assessing exposure to isocyanates

The registered medical practitioner may choose to assess isocyanate exposure by a urinary isocyanate metabolite level test. The urine sample should be taken following the isocyanate task.

Where urine analysis is performed, the following values should be used as a guide for assessing exposure to PAH:

Biological Level	Source
1 µmol of isocyanate-derived diamine/mol creatinine in urine	NSW Workcover Biological Occupational Exposure Limit
10 µg methylenediamine (MDA)/L (~4 µmol MDA/mol creatinine) in urine	German Commission for the Investigation of Health Hazards of Chemical Compounds in the Work Area Biologischer Leit-Wert (BLW) value

Note: The absorbed isocyanates are metabolised and excreted in urine as the corresponding diamine and conjugates. The half-lives are usually short (two to four hours) so samples only reflect recent exposure.

AT TERMINATION OF WORK IN AN ISOCYANATE PROCESS

8. Final medical examination

A final medical examination will be conducted and will include:

- physical examination for work-related dermatitis
- standardised respiratory function tests.

9. Health advice

Workers sensitised to isocyanates should be strongly advised against further exposure.

SUPPLEMENTARY INFORMATION ON ISOCYANATES

10. Work activities that may represent a high risk exposure

Isocyanates are compounds containing one or more -N=C=O groups which can combine with other compounds containing alcohol groups. 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, like in vehicle paints
- processes where heat decomposition of polyurethane products occurs, like welding, heat removal of electrical insulating varnishes and hot wire cutting of foam.

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

POTENTIAL HEALTH EFFECTS FOLLOWING EXPOSURE TO ISOCYANATES

11. Route of entry into the body

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

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 isocyanates like HDI have been partially polymerised into the form of pre-polymers so they are less volatile, however, the spray painting process creates a mist of easily inhaled fine particles.

12. Target organ/effect

Respiratory tract – irritation, sensitisation with work-related asthma.

Eyes – irritation.

Skin – irritation, sensitisation.

CNS – headache, loss of consciousness, coma.

13. 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.

In mild cases there may be slight irritation of the nose and throat. Headaches can also occur from inhalation of low concentrations of isocyanates. With higher exposure there may be acute bronchial irritation with coughing, shortness of breath and bronchospasm, abdominal distress, nausea and vomiting, chemical pneumonitis and pulmonary oedema. 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. Evidence is emerging that RADS can be seen as one end of a spectrum of irritant effects on the airways.

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

Oral toxicity appears to be low.

14. Chronic effects

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

Diisocyanates 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.

There is growing evidence skin exposure can induce isocyanate respiratory sensitisation although this is still under debate. Skin exposure may be especially important with less volatile diisocyanates like polyisocyanates and MDI where skin exposure may be the main route of exposure [5].

The estimated prevalence of work-related asthma in the isocyanate exposed workforce has most commonly been reported in the range five to 10 per cent. There is no evidence atopy influences susceptibility. Smoking has been identified as increasing the risk of work-related asthma in workers exposed to isocyanates.

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. There is a latent (sensitising) period of exposure to isocyanates that is highly variable: from several weeks, often less than two years but in 20 per cent 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 chronicity of work-related asthma (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, a granulomatous inflammatory reaction in terminal airways, alveoli and surrounding interstitium. Symptoms are dyspnoea, malaise and fever occurring several hours after work with isocyanates. Diagnosis is confirmed by restrictive ventilatory patterns, reticular or nodular lung patterns on chest X-ray.

Other health effects may include liver and kidney dysfunction. Interstitial pulmonary fibrosis has been reported as a long-term hazard.

Adverse health effects resulting from exposure to isocyanates normally arise during the ordinary working period, soon after contact occurs. Occasionally, as with hypersensitivity pneumonitis, symptoms may not appear for several hours following exposure. 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 adverse health effects and they should report adverse health effects which they think may be related to isocyanate exposure so the root-cause can be investigated.

15. Carcinogenicity

The International Agency for Research on Cancer concluded there is sufficient evidence TDI is carcinogenic in experimental animals and there is limited evidence for a carcinogenic effect of MDI in animals. Increased incidence of lung tumours in rats resulted from inhalation of a mixture of monomeric and polymeric 4,4'-MDI. Inhalation of freshly generated polyurethane dust has been reported to generate lung tumours in rats [6].

16. Carcinogen classifications

The following isocyanates are classified according to the GHS 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).

REFERENCED DOCUMENTS

1. Respiratory Disease Committee of the International Union Against Tuberculosis, *IUAT Bronchial Symptoms Questionnaire*, International Union Against Tuberculosis, 1986.
2. Medical Research Council Committee on Research into Chronic Bronchitis, *MRC Questionnaire on Respiratory Symptoms*, Medical Research Council, 1986.
3. BOHRF, *Occupational Asthma: Evidence Review*, British Occupational Health Research Foundation, London 2010.
4. Health and Safety Executive (UK), *Safety in Motor Vehicle Repair, Working with Isocyanate Paints*, Leaflet INDG388(rev1), Health and Safety Executive, London, Dec, 2009
5. Bello D, Woskie SR, Streicher RP, Liu Y, Stowe MH, Eisen EA, Ellenbecker MJ, Sparer J, Youngs F, Cullen MR, Redlich CA, 'Polyisocyanates in Occupational Environments: A Critical Review of Exposure Limits and Metrics', *American Journal of Industrial Medicine*, vol 46, pp 480-491, 2004.
6. Mikoczy Z, Welinder H, Tinnerberg H, Hagmar L, 'Cancer Incidence and Mortality of Isocyanate Exposed Workers from the Swedish Polyurethane Foam Industry: Updated Findings 1959-98', *Occupational and Environmental Medicine*, vol 61, pp 432-437, 2004.

FURTHER READING

Allport D, Gilbert D, Outterside S, *MDI and TDI: Safety, Health and the Environment: A Source Book and Practical Guide*, John Wiley and Sons, New York, 2003.

American Conference of Governmental Industrial Hygienists (ACGIH), *Documentation of the Threshold Limit Values for Chemical Substances*, 7th Ed, Cincinnati, 2011.

Bernstein DI, Jolly A, 'Current Diagnostic Methods for Diisocyanate Induced Occupational Asthma', *American Journal of Industrial Medicine*, vol 36, pp 459-468, 1999.

Burge S, 'Respiratory Symptoms', *Occupational Medicine*, vol 47, pp 55-56, 1997.

HSE (UK), *COSHH Essentials: General Guidance, G408, Urine sampling for isocyanate exposure measurement*, Health and Safety Executive, London.

Lauwerys RR, Hoet P, *Industrial Chemical Exposure Guidelines for Biological Monitoring*, 3rd Ed, Lewis Publishers, Boca Raton, 2001.

Occupational asthma in Australia, Australian Institute of Health and Welfare Bulletin 59, April 2008 www.aihw.gov.au

WorkCover NSW, *Chemical Analysis Branch Handbook*, 8th edition. Available at www.testsafe.com.au.

This health monitoring report is a confidential health record and must not be disclosed to another person except in accordance with the Work Health and Safety Regulations or with the consent of the worker.

There are two sections. Complete both sections and all questions if applicable.

Section 1 is to be forwarded to the PCBU who has engaged your services. A copy of laboratory report(s) must be attached > > > >

Section 2 may contain confidential information which may not be relevant to the health monitoring program being carried out. This section should be retained by the medical practitioner. Information which is required to be given to the PCBU should be summarised in part 7 of section 1.

SECTION 1 – THIS SECTION TO BE RETURNED TO THE PCBU			
1. PERSON CONDUCTING A BUSINESS OR UNDERTAKING			
Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
2. OTHER BUSINESSES OR UNDERTAKINGS ENGAGING THE WORKER			
Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
3. WORKER		(✓) all relevant boxes	
Surname:		Given names:	
Date of birth: DD/MM/YYYY	Sex:	☐Male	☐ Female
Address:			
Suburb:			Postcode:
Current Job:	Tel(H):		Mob:
Date started employment : DD/MM/YYYY			
4. EMPLOYMENT IN ISOCYANATE RISK WORK		(✓) all relevant boxes	
1. ☐ New to isocyanate work			
2. ☐ New worker but not new to isocyanate work			
3. ☐ Current worker continuing in isocyanate work			
4. Worked with isocyanate since DD/MM/YYYY			
5. Satisfactory personal hygiene (for example nail biting, frequency of hand washing)		☐ Yes	☐ No
6. Risk assessment completed		☐ Yes	☐ No

5. WORK ENVIRONMENT ASSESSMENT		(✓) all relevant boxes	
Date of assessment: DD/MM/YYYY			
Isocyanate Industry			
☐ Isocyanate Manufacture	**Controls:**		
☐ Foam Manufacture	Eye protection	☐ Yes	☐ No
☐ Spray Painting	Wear gloves	☐ Yes	☐ No
☐ Welding/Fabrication	Respirator use	☐ Yes	☐ No
☐ Automotive Industry	Local exhaust ventilation	☐ Yes	☐ No
☐ Furniture Industry	Overalls / work clothing	☐ Yes	☐ No
☐ Flooring Industry	Laundering by employer	☐ Yes	☐ No
☐ Other (specify):	Wash basins & showers (with hot & cold water)	☐ Yes	☐ No
	Smoking or eating in workshop	☐ Yes	☐ No
	Personal hygiene:		
	Clean Shaven	☐ Yes	☐ No
	Clean hands with thinners	☐ Yes	☐ No
	Shower & change into clean clothes at end of shift	☐ Yes	☐ No

6. BIOLOGICAL MONITORING RESULTS Include at least the previous two test results (if available)		
Date	**Tests performed**	**Recommended Action and/or Comment**
1. DD/MM/YYYY	Spirometry	Results: FEV_1 _____; FVC_____; FEV_1/FVC_____
2. DD/MM/YYYY		
3. DD/MM/YYYY		
4. DD/MM/YYYY		
5. DD/MM/YYYY		
6. DD/MM/YYYY		
7. DD/MM/YYYY		
8. DD/MM/YYYY		

7. RECOMMENDATIONS (by Medical Practitioner) (✓) all relevant boxes		
1. ☐ Suitable for work with isocyanates		
2. ☐ Counselling required		
3. ☐ Review workplace controls		
4. ☐ Repeat health assessment (including any tests) in _____ month(s) / ____ week(s) Specify tests to be repeated: _____		
5. ☐ Removal from work with isocyanates	On DD/MM/YYYY	
6. ☐ Medical examination by Medical Practitioner	On DD/MM/YYYY	
7. ☐ Fit to resume work with isocyanates	From DD/MM/YYYY	
8. ☐ Referred to Medical Specialist (respiratory/dermatology/other):	On DD/MM/YYYY	
Specialist's name:		
Additional comments or recommendations arising from health monitoring:		

Medical Practitioner (responsible for supervising health monitoring)		
Name:	Signature	Date: DD/MM/YYYY
Tel:	Fax:	Registration Number:
Medical Practice:		
Address:		
Suburb:		Postcode:

SECTION 2 – THIS SECTION TO BE RETAINED BY THE MEDICAL PRACTITIONER			
This questionnaire also allows for recordings of a more general health assessment at the end, if applicable.			
1. PERSON CONDUCTING A BUSINESS OR UNDERTAKING			
Company / Organisation name:			
Site address:			
Suburb:		Postcode:	
Site Tel:	Site Fax:	Contact Name:	
2. OTHER BUSINESSES OR UNDERTAKINGS ENGAGING THE WORKER			
Company / Organisation name:			
Site address:			
Suburb:		Postcode:	
Site Tel:	Site Fax:	Contact Name:	
3. WORKER		(✓) all relevant boxes	
Surname:	Given names:		
Date of birth: DD/MM/YYYY	Sex:	☐ Male	☐ Female
Address:			
Suburb:		Postcode:	
Current Job:	Tel(H):	Mob:	
Date started employment : DD/MM/YYYY			

This questionnaire is based on the MRC (UK) Respiratory Questionnaire 1986, which has been extensively validated. This questionnaire is intended to be completed by an interviewer rather than by the patient. Additional questions have been added to cover clinical aspects of bronchial hyper-responsiveness validated by the Department of Occupational and Environmental Medicine, National Lung Institute[1].

The British Occupational Health Research Foundation (BOHRF)[2] concluded that in the clinical setting, questionnaires that identify symptoms of wheeze and/or shortness of breath which improve on days away from work or on holidays have a high sensitivity, but relatively low specificity for occupational asthma.

Preamble

I am going to ask some questions, mainly about your chest. I would like you to answer **yes** or **no** whenever possible.

If the subject is disabled from walking from any condition other than heart and lung disease, please begin questionnaire at **Question 5** and mark the adjacent box.

1 Venables KM, Farrer N, Sharp L, Graneek BJ, Newman Taylor AJ, 'Respiratory Symptoms Questionnaire for Asthma Epidemiology: Validity and Reproducibility', *Thorax*, vol 48, pp 214-219, 1993.

2 The British Occupational Health Research Foundation (BOHRF), *Guidelines for Prevention, Identification and Management of Occupational Asthma: Evidence Review and Recommendations*, London 2004. www.bohrf.org.uk

4. BREATHLESSNESS AND WHEEZING		
During the last month:		
1. Are you troubled by shortness of breath when hurrying on level ground or walking up a slight hill?	☐ Yes	☐ No
2. **If Yes to 1** - Do you get short of breath walking with other people of your age on level ground?	☐ Yes	☐ No
3. **If Yes to 2** - Do you have to stop for breath when walking at your own pace on level ground?	☐ Yes	☐ No
4. If you run, or climb stairs fast do you ever		
a. cough?	☐ Yes	☐ No
b. wheeze?	☐ Yes	☐ No
c. get tight in the chest?	☐ Yes	☐ No
5. Is your sleep ever broken		
a. by wheeze?	☐ Yes	☐ No
b. difficulty in breathing?	☐ Yes	☐ No
6. Do you ever wake up in the morning (or from your sleep if a shift worker)		
a. with wheeze?	☐ Yes	☐ No
b. difficulty with breathing?	☐ Yes	☐ No
7. Do you ever wheeze		
a. if you are in a smoky room?	☐ Yes	☐ No
b. if you are in a very dusty place?	☐ Yes	☐ No
8. **If Yes to either Q5, Q6, Q7** - Are your symptoms better		
a. at weekends (or equivalent if shift worker)?	☐ Yes	☐ No
b. when you are on holidays?	☐ Yes	☐ No
If **Yes to Question 8**, please record details of any occupational exposure to respiratory hazards e.g. isocyanates, wood dust, aluminium pot room or asbestos, in **Additional notes**.		
5. COUGH		
9. Do you usually cough first thing in the morning in winter?	☐ Yes	☐ No
10. Do you usually cough during the day/ or at night / in the winter?	☐ Yes	☐ No
11. **If Yes to Q9 or Q10** – Do you cough like this on most days for as much as three months each year?	☐ Yes	☐ No
6. PHLEGM		
12. Do you usually bring up phlegm from your chest first thing in the morning in winter?	☐ Yes	☐ No

13. Do you usually bring up any phlegm from your chest during the day / or at night / in winter?	☐ Yes	☐ No
14. **If Yes to Q12 or Q13** – Do you bring up phlegm like this on most days for as much as three months each year?	☐ Yes	☐ No
7. PERIODS OF COUGH AND PHLEGM		
15. In the past three years, have you had a period of (increased) cough and phlegm lasting for three weeks or more?	☐ Yes	☐ No
16. **If Yes to Q15** – Have you had more than one such episode?	☐ Yes	☐ No
8. CHEST ILLNESSES		
17. During the past three years, have you had any chest illness that has kept you from your usual activities for as much as a week?	☐ Yes	☐ No
18. **If Yes to Q17** – Did you bring up more phlegm than usual in any of these illnesses?	☐ Yes	☐ No
19. **If Yes to Q18** – Have you had more than one illness like this in the past three years?	☐ Yes	☐ No
9. PAST ILLNESSES		
20. Have you ever had, or been told that you have had any of the following?		
a. An injury, or operation affecting your chest?	☐ Yes	☐ No
b. Heart problems?	☐ Yes	☐ No
c. Bronchitis?	☐ Yes	☐ No
d. Pneumonia?	☐ Yes	☐ No
e. Pleurisy?	☐ Yes	☐ No
f. Asthma?	☐ Yes	☐ No
g. Other chest trouble?	☐ Yes	☐ No
h. Hay fever?	☐ Yes	☐ No
10. TOBACCO SMOKING		
21. Do you smoke?	☐ Yes	☐ No
If No to Q21		
22. Have you ever smoked as much as one cigarette a day for as long as one year?	☐ Yes	☐ No
23. How old were you when you started smoking regularly? _____		
24. a. Do (did) you smoke manufactured cigarettes?	☐ Yes	☐ No
If Yes to Q24a: How many do (did) you usually smoke per day? _____		
b. on weekdays? _____ c. at weekends? _____		

25. Do (did) you smoke any other forms of tobacco? ☐ Yes ☐ No **If Yes to Q25, record details under Additional notes**			
11. FOR EX-SMOKERS			
26. When did you give up smoking? Month _____ Year _____			
Additional notes:			
12. GENERAL HEALTH ASSESSMENT (if applicable)			
Symptoms of:	**Comments**	**Further testing?**	
Skin disorders		☐ Yes	☐ No
Headaches, dizziness		☐ Yes	☐ No
Respiratory disorders (asthma, wheezing, etc)		☐ Yes	☐ No
Irritation of eyes, nose or throat		☐ Yes	☐ No
Cough		☐ Yes	☐ No
CNS		☐ Yes	☐ No
Others		☐ Yes	☐ No
Height _____cm Weight _____kg Bp _____/___mmHg		☐ Yes	☐ No

13. OTHER MEDICAL HISTORY, FAMILY MEDICAL HISTORY, CURRENT MEDICATION, COMMENTS, TESTS OR RECOMMENDATIONS (use separate sheet if necessary)			
Medical Practitioner (responsible for supervising health monitoring)			
Name:	Signature		Date: DD/MM/YYYY
Tel:	Fax:	Registration Number:	
Medical Practice:			
Address:			
Suburb:		Postcode:	

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LEAD (INORGANIC)

DEFINITIONS

The following definitions apply to this Guideline.

blood lead level monitoring means the testing of the venous or capillary blood of a person by a laboratory accredited by the National Association of Testing Authorities (NATA), under the supervision of a registered medical practitioner, to determine the blood lead level.

blood lead level means the concentration of lead in whole blood expressed in micromoles per litre (μmol/L) or micrograms per decilitre (μg/dL).

biological monitoring means:

a. the measurement and evaluation of a substance, or its metabolites, in the body tissue, fluids or exhaled air of a person exposed to the substance; or

b. blood lead level monitoring.

female of reproductive capacity means a female other than a female who provides information stating that she is not of reproductive capacity.

lead means lead metal, lead alloys, inorganic lead compounds and lead salts of organic acids.

lead risk work means work carried out in a lead process that is likely to cause the blood lead level of a worker carrying out the work to be more than:

a. for a female of reproductive capacity—10μg/dL (0.48μmol/L); or

b. in any other case—30μg/dL (1.45μmol/L).

Note: examples of lead processes can be found at Part 11.

lead process area means a workplace or part of a workplace where a lead process is carried out.

BASELINE HEALTH MONITORING BEFORE STARTING WORK IN AN INORGANIC LEAD PROCESS

1. Baseline health monitoring

Baseline health monitoring of the worker is required:

- before the worker first starts lead risk work
- one month after the worker first starts lead risk work.

If work is identified as lead risk work after a worker starts the work, health monitoring of the worker must be provided:

- as soon as practicable after the lead risk work is identified
- one month after the first monitoring of the worker under paragraph (a).

2. Collection of demographic data

3. Work History

4. Medical history

The following details about the worker's medical history will be collected by the medical practitioner:

- presence of symptoms with an emphasis on reproductive history including current pregnancy or breast feeding, neuropsychologic problems, haematological disorders and renal disorders
- prior history of non-work-related lead exposure e.g. hobbies like shooting (exposure to gun powder) and fishing (exposure to lead sinkers)
- history of medication or medical treatment including recent chelating agent therapy e.g. EDTA
- smoking history.

5. Physical Examination

A physical examination will be conducted, with an emphasis on the gastrointestinal, haematopoietic, renal, cardiovascular, reproductive and neurological systems.

Assessment of the pulmonary status is also warranted in cases where respiratory protective equipment is likely to be needed. Worker should be counselled that respirator fit can be poor and protection ineffective if they have a beard or facial hair.

6. Investigation

The following tests may be conducted to test the worker's baseline exposure:

- full blood examination
- blood lead in whole blood or packed red cells
- serum creatinine
- routine urinalysis
- pulmonary function test in cases where respiratory protection is likely to be required.

7. Counselling

The registered medical practitioner supervising the health monitoring should take into consideration whether medical counselling is required for the worker. If medical counselling is required, the level of counselling and recommended timeframe/level of urgency should be recorded in the Health Monitoring Report, see Appendix 1. For further information about counselling see Appendix 2.

Counselling for lead risk work should include the following health and personal hygiene advice.

Health effects of lead	Workers should be informed of the potential health effects associated with exposure to inorganic lead including the different risks to men and women and people of younger age (<18).
Family planning	Workers who consider they have not completed their family should be counselled on the health effects of lead on male and female reproduction, as appropriate.
Pregnancy	Workers who are pregnant or breastfeeding should be advised to seek alternative work during that period from their PCBU which does not involve lead risk work.

Personal hygiene

Workers should be encouraged to use changing rooms and washing, showering and toilet facilities at the workplace in order to minimise secondary lead exposure from contaminated clothing; minimise ingestion of lead; and avoid the spread of lead contamination.

Workers who bite their nails should be counselled on the increased risk it places on lead intake.

Eating, drinking and smoking

Workers should be reminded:

- they are not permitted to smoke, carry smoking materials, eat, chew gum or drink in a lead process area
- the importance of removing lead contaminated clothing and equipment and to wash their hands and faces before entering areas provided for eating and drinking.

  A full explanation of the reasons for these restrictions and the benefits to be gained by compliance should be given.

Those workers with smoking history should be counselled on the possible additional lead burden from smoking.

DURING EXPOSURE TO AN INORGANIC LEAD PROCESS

8. Monitoring exposure to inorganic lead

Biological monitoring must be arranged for each worker who carries out lead risk work at the following times:

<u>For females not of reproductive capacity and males</u>

- six months after the last biological monitoring of the worker if the last monitoring shows a blood lead level of less than 30µg/dL (1.45µmol/L); **or**
- three months after the last biological monitoring of the worker if the last monitoring shows a blood lead level of 30µg/dL (1.45µmol/L) or more but less than 40µg/dL (1.93µmol/L); **or**
- six weeks after the last biological monitoring of the worker if the last monitoring shows a blood lead level of 40µg/dL (1.93µmol/L) or more.

<u>For females of reproductive capacity</u>

- three months after the last biological monitoring of the worker if the last monitoring shows a blood lead level of less than 10µg/dL (0.48µmol/L); **or**
- six weeks after the last biological monitoring of the worker if the last monitoring shows a blood lead level of 10µg/dL (0.48µmol/L) or more.

The frequency of biological monitoring must be increased if the worker carries out an activity that is likely to significantly change the nature or increase the duration or frequency of the worker's lead exposure. If the above biological exposure limits are breached, workplace practices and controls should be immediately reviewed as this suggests current controls are not performing satisfactorily.

9. Removal of a worker from a lead risk work

A worker must be immediately removed from carrying out lead risk work if:

1. biological monitoring of the worker shows that the worker's blood lead level is, or is more than:
   - for females not of reproductive capacity and males—50µg/dL (2.42µmol/L); **or**
   - for females of reproductive capacity—20µg/dL (0.97µmol/L); **or**
   - for females who are pregnant or breastfeeding—15µg/dL (0.72µmol/L); **or**
2. following a medical examination of the worker, the medical practitioner who supervised the health monitoring recommends that the worker must be removed from carrying out the lead risk work; **or**
3. there is an indication that a risk control measure has failed and as a result, the worker's blood lead level is likely to reach the relevant level for the worker mentioned above.

If a worker's blood lead level is above the prescribed removal level, the Health Monitoring Report should advise immediate removal to alternative duties. A second medical examination should be conducted within seven days after the day the worker is removed from lead risk work.

10. Return to work

The frequency of repeat blood lead level tests after removal from lead risk work is at the discretion of the medical practitioner supervising the health monitoring, but should be done at least every three to six weeks until the appropriate fall in blood lead levels has occurred.

The worker should be examined periodically to determine whether the worker is suitable to return to carrying out lead risk work.

A worker must not return to lead risk work until the worker's blood lead level is less than:

- for females not of reproductive capacity and males—40µg/dL (1.93µmol/L); **or**
- for females of reproductive capacity—10µg/dL (0.48µmol/L); **AND**

they have been assessed as medically fit to return to lead risk work by the medical practitioner supervising the health monitoring.

SUPPLEMENTARY INFORMATION ON INORGANIC LEAD

11. Work activities that may represent a high risk exposure (lead processes)

It is a requirement of the regulations that a PCBU determines whether a job is a lead risk job requiring health monitoring. The following **lead processes** may involve significant exposures to lead:

a. work that exposes a person to lead dust or lead fumes arising from the manufacture or handling of dry lead compounds

b. 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

c. 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

d. spraying molten lead metal or alloys containing more than five per cent by weight of lead metal

e. 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

f. recovering lead from its ores, oxides or other compounds by thermal reduction process

g. dry machine grinding, discing, buffing or cutting by power tools alloys containing more than 5 per cent by weight of lead metal

h. machine sanding or buffing surfaces coated with paint containing more than one per cent by dry weight of lead

i. 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

j. radiator repairs that may cause exposure to lead dust or lead fumes

k. fire assays if lead, lead compounds or lead alloys are used

l. hand grinding and finishing lead or alloys containing more than 50 per cent by dry weight of lead

m. spray painting with lead paint containing more than one per cent by dry weight of lead;

n. 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

o. 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

p. a process that exposes a person to lead dust or lead fumes arising from manufacturing or testing detonators or other explosives that contain lead

q. a process that exposes a person to lead dust or lead fumes arising from firing weapons at an indoor firing range

r. 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

s. a process decided by the regulator to be a lead process under regulation 393.

12. Observed health effects and blood lead levels

Lead affects people of all ages, but the effects of lead are considered most serious in young children. Inorganic lead uptake occurs as a result of ingestion or inhalation of inorganic lead particles. Not only are particulates in air, like dusts and fumes, important sources of exposure in the workplace, but also from 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. When inhaled, most inorganic forms of lead deposited in the alveolar regions appear to be almost completely absorbed, although it is possible lead compounds of low solubility like lead sulphide may accumulate to some extent in the lung. Absorption of inhaled lead is affected by various factors including personal characteristics, physical activity, particle size and solubility of the airborne lead.

In 2007, Kosnett et al published *Recommendations for Medical Management of Adult Lead Exposure*, which shows a summary of the adverse health risks associated with different blood lead concentrations and presents corresponding medical management recommendations that range from discussion of risks and reductions of lead exposure at low levels to removal from lead exposure accompanied by probable chelation therapy at the highest levels, see Appendix 3.

The publication notes that research conducted in recent years has increased concern about the toxicity of lead at low blood lead levels and supports a reappraisal of the levels of lead exposure that may be safely tolerated in the workplace. Consistent with the American Conference of Governmental Industrial Hygienists (ACGIH) recommendations, it recommends individuals be removed from work lead exposure if a single blood lead measurement exceeds 30μg/dL.

It focuses on four categories of health effects – hypertension, renal function, cognitive dysfunction, and adverse reproductive outcome; however, it does not mention carcinogenicity. Since there is no dose-response relationship for cancer, the risk of this disease applies to all blood lead level bands. The designation of risks as either "short-term" or "long-term," depending on whether the risks are associated with exposure lasting less than or more than one year, reflects a qualitative understanding of the duration of lead exposure that may be required to elicit certain adverse health effects of lead. The categorisation of risks in Appendix 3 by discrete bands of blood lead concentration is a qualitative assessment.

Inhibition of the mitochondrial enzyme, ferrocheletase, which is the next most sensitive enzyme, results in accumulation of free erythrocyte protoporphyrin (FEP) in the red blood cells primarily as zinc protoporphyrin (ZPP) and increased urinary excretion of coproporphyrin. Because ZPP remains in the erythrocyte for the average lifespan of the red blood cell, the blood ZPP level reflects averaged exposure over a three-month period.

Blood ZPP levels can therefore be used as a measures of lead exposure. There is a lot of individual variability in the protoporphyrin response to lead absorption and it is suggested results are compared with previous results from the same individual. Monitor the individual response rather than interpret a particular level. The protoporphyrin response lags behind the current blood lead level as an increase only becomes measurable in the peripheral blood as affected erythrocytes mature and are released from the bone marrow. The lag is around two to three months. It is recommended the testing for ZPP as a measure of lead exposure only be considered once removal limits have been reached[1]. Continued removal from lead work is recommended until levels return to satisfactory levels.

13. Inorganic lead toxicity

One of the main targets of inorganic lead toxicity in adults is the nervous system—central and peripheral. Severe exposures may cause encephalopathy that is progressive degeneration of certain parts of the brain, coma or death. Historically, high, chronic workplace exposure to lead damages the peripheral nervous system, 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.

1 Wooller, KK (2003) Occupational Medicine Handbook (Eleventh Edition), Information for WorkCover Authority of NSW Authorised Medical Practitioners.

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 electrocardiographic abnormalities.

When inorganic lead enters the body it does not undergo biological transformation. Lead is a cumulative poison. This 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.

Adults have an approximate 94 per cent body burden, that is more is stored in the body than circulated in the blood. Once in the body, lead is transported in the bloodstream, entering all body tissues. Only two to five per cent of the total body lead is found in red blood cells.

Lead is preferentially stored in the skeleton and in regions undergoing the most active calcification at the time of exposure—cortical and trabecular. Acute lead poisoning is uncommon today in work settings.

Distribution of lead to various organs has variable elimination rates. Soft tissue is fast whereas skeletal is slow. Blood lead clearance shortly after exposure changes is approximately 20-35 days - red blood cells have a half life of 120 days. Redistribution from bone, however, is much slower and takes approximately three to 30 years.

Body recovery is slower each time exposure occurs and body burden builds up over a lifetime. Clinical treatment using chelation therapy to reduce lead levels may decrease total lead body burden but not the risk of cognitive effects.

14. Carcinogen and reproductive toxicant classifications

The following are examples of lead chemicals with GHS 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, [≥ 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

FURTHER READING

Association of Occupational and Environmental Clinics (AOEC), *Medical Management Guidelines for Lead-Exposed Adults* Revised 04/24/2007.

Sourced AOEC - http://www.aoec.org/principles.htm

Australian Institute of Occupational Hygienists Position Paper, *Inorganic Lead and Occupational Health Issues*, March 2009.

Cherrie JW, Semple S, Christopher Y, Saleem A, Hughson GW, Phillips A, 'How Important is Inadvertent Ingestion of Hazardous Substances at Work?', *Annals of Occupational Hygiene*, vol 50(7): pp 693-704, 2006. http://annhyg.oxfordjournals.org/cgi/content/full/50/7/693

Lauwerys RR, Hoet P, *Industrial Chemical Exposure Guidelines for Biological Monitoring*, 3rd Ed, Lewis Publishers, Boca Raton, 2001.

Lead Development Association International (LDAI), *Voluntary Risk Assessment Report on Lead and Some Lead Compounds*, Human Health Section, Interim Revised Draft, March 2008, prepared by the ILZRO and EBRC consulting under contract to the LDAI Lead Risk Assessment Working Group.

Lundströrom N-G, Nordberg G, Englyst V, Gerhardsson L, Hagmar L, Jin T, Rylander L, Wall S, 'Cumulative Lead Exposure In Relation to Mortality and Lung Cancer Morbidity in a Cohort of Primary Smelter Workers', *Scand J Work Environ Health*, vol 23(1); pp 24-30, 1997.

Skerfving S, *Criteria Document for Swedish Occupational Standards: Inorganic Lead – an update 1991–2004*, The Swedish Group for Occupational Standards, Department of Occupational and Environmental Medicine, Lund, Sweden, 2005. https://gupea.ub.gu.se/dspace/handle/2077/4356

APPENDIX 1

This health monitoring report is a confidential health record and must not be disclosed to another person except in accordance with the Work Health and Safety Regulations or with the consent of the worker.

There are two sections. Complete both sections and all questions if applicable.

Section 1 is to be forwarded to the PCBU who has engaged your services. A copy of laboratory report(s) must be attached > > > >

Section 2 may contain confidential information which may not be relevant to the health monitoring program being carried out. This section should be retained by the medical practitioner. Information which is required to be given to the PCBU should be summarised in part 7 of section 1.

SECTION 1 – THIS SECTION TO BE RETURNED TO THE PCBU		
1. PERSON CONDUCTING A BUSINESS OR UNDERTAKING		
Company / Organisation name:		
Site address:		
Suburb:		Postcode:
Site Tel:	Site Fax:	Contact Name:
2. OTHER BUSINESSES OR UNDERTAKINGS ENGAGING THE WORKER		
Company / Organisation name:		
Site address:		
Suburb:		Postcode:
Site Tel:	Site Fax:	Contact Name:
3. WORKER		(✓) all relevant boxes
Surname:		Given names:
Date of birth: DD/MM/YYYY	Sex: ☐ Male	☐ Female
Address:		
Suburb:		Postcode:
Current Job:	Tel(H):	Mob:
Date started employment : DD/MM/YYYY		
4. EMPLOYMENT IN LEAD RISK WORK		(✓) all relevant boxes
1. ☐ New to lead work		
2. ☐ New worker but not new to lead work		
3. ☐ Current worker continuing in lead work		
4. Worked with lead since DD/MM/YYYY		

5. Satisfactory personal hygiene (for example nail biting, frequency of hand washing)	☐ Yes ☐ No
6. Risk assessment completed	☐ Yes ☐ No

5. WORK ENVIRONMENT ASSESSMENT (✓) all relevant boxes

Date of assessment: DD/MM/YYYY

Lead Industry	☐ Smoker ☐ Ex-smoker ☐ Non-smoker		
☐ Fire Assay	**Controls:**		
☐ Foundry	Wear gloves	☐ Yes	☐ No
☐ Lead Battery – Maintenance	Respirator use	☐ Yes	☐ No
☐ Lead Burning	Local exhaust ventilation	☐ Yes	☐ No
☐ Lead Flux – Manufacture	Overalls / work clothing	☐ Yes	☐ No
☐ Leadlight Work	Laundering by employer	☐ Yes	☐ No
☐ Lead Paint – Manufacture	Wash basins & showers (with hot & cold water)	☐ Yes	☐ No
☐ Lead Paint – Painting	Smoking or eating in workshop	☐ Yes	☐ No
☐ Lead Paint – Stripping/ Cleaning	Dry sweeping	☐ Yes	☐ No
☐ Lead Sinker – Manufacture	**Personal hygiene:**		
☐ Metal Recycling	Clean Shaven	☐ Yes	☐ No
☐ Monumental Work	Shower & change into clean clothes at end of shift	☐ Yes	☐ No
☐ Radiator Repair			
☐ Firing Range			
☐ Other (specify):			

6. BIOLOGICAL MONITORING RESULTS Include at least the previous two test results (if available)

Date	**Blood lead level** (µg/dL or µmol/L)	**Recommended Action and/or Comment**
1. DD/MM/YYYY		Insert baseline or last known result and date
2. DD/MM/YYYY		
3. DD/MM/YYYY		
4. DD/MM/YYYY		

5. DD/MM/YYYY		
6. DD/MM/YYYY		

7. RECOMMENDATIONS (by Medical Practitioner) (✓) all relevant boxes

1. ☐ Suitable for work with lead
2. ☐ Counselling required
3. ☐ Review workplace controls
4. ☐ Repeat health assessment in _____ month(s) / _____ week(s)
5. ☐ Removal from work with lead On DD/MM/YYYY
6. ☐ Medical examination by Medical Practitioner On DD/MM/YYYY
7. ☐ Fit to resume lead risk work From DD/MM/YYYY
8. ☐ Referred to Medical Specialist (respiratory/dermatology/other): On DD/MM/YYYY

Specialist's name:

Additional comments or recommendations arising from health monitoring:

Medical Practitioner (responsible for supervising health monitoring)

Name:	Signature	Date: DD/MM/YYYY
Tel:	Fax:	Registration Number:
Medical Practice:		
Address:		
Suburb:		Postcode:

SECTION 2 – THIS SECTION TO BE RETAINED BY THE MEDICAL PRACTITIONER			
1. PERSON CONDUCTING A BUSINESS OR UNDERTAKING			
Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
2. OTHER BUSINESSES OR UNDERTAKINGS ENGAGING THE WORKER			
Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
3. WORKER			(✓) all relevant boxes
Surname:		Given names:	
Date of birth: DD/MM/YYYY	Sex:	☐ Male	☐ Female ☐ Pregnant/Breast Feeding?
Address:			
Suburb:			Postcode:
Current Job:		Tel(H):	Mob:
Date started employment : DD/MM/YYYY			

4. GENERAL HEALTH ASSESSMENT (if applicable)

Symptoms of:	**Comments**	**Further testing?**
Skin disorders		☐ Yes ☐ No
Headaches, dizziness		☐ Yes ☐ No
Respiratory disorders		☐ Yes ☐ No
Irritation of eyes, nose or throat		☐ Yes ☐ No
Cough		☐ Yes ☐ No
CNS		☐ Yes ☐ No
Peripheral nervous system symptoms		☐ Yes ☐ No
Others: Pregnant Breastfeeding Smoker		☐ Yes ☐ No ☐ Yes ☐ No ☐ Yes ☐ No ☐ Yes ☐ No

Height _____cm Weight _____kg Bp _____/_____ mmHg		

5. OTHER MEDICAL HISTORY, FAMILY MEDICAL HISTORY, CURRENT MEDICATION, COMMENTS, TESTS OR RECOMMENDATIONS (use separate sheet if necessary)

Medical Practitioner (responsible for supervising health monitoring)

Name:	Signature	Date: DD/MM/YYYY
Tel:	Fax:	Registration Number:
Medical Practice:		
Address:		
Suburb:		Postcode:

APPENDIX 2
COUNSELLING

Counselling is a process of dialogue between an individual worker and the various parties involved in the management of work exposure to lead.

Workers who are to start work in lead risk jobs or who work in lead risk jobs must be counselled on the health effects of lead. Workers excluded from working in lead risk jobs should also be counselled.

Counselling will usually be an informal discussion about a workplace or workstation, work practice, personal hygiene practice, and about the health effects of lead, between the worker and the medical practitioner at the time of attendance for biological monitoring. More formal discussion about these matters should take place during a medical examination carried out by the medical practitioner. If the worker is to be removed from work exposure to lead then there should be an emphasis on the health effects of lead and actions to prevent a recurrence of removal.

Workers who consider they have not completed their family should be counselled in particular on the effects of lead on male and female reproduction, as appropriate. Female workers working in lead-risk jobs should be counselled on the effects of lead on foetal and childhood development, in particular cognitive development. The level of counselling should be such that the worker can make an informed decision in regard to the risk to their own health and to a future foetus. Male workers should be told exposure to lead may adversely affect reproductive function. Female workers should be told exposure to lead during pregnancy may be associated with pregnancy complications and may pose a risk to the development of the foetus or eventual child.

Counselling may cover the following topics:

Physical maturity. As a guide people under the age of 16 should not be employed in lead processes.

Medical conditions. Individuals with certain medical conditions, for example impaired renal function and anaemia, haemoglobinopathies, neuropathies and reproductive problems may be more susceptible to adverse health effects of lead.

Lead accumulation in the body, particularly in bones. This can be mobilised in some circumstances including pregnancy and old age.

Females of reproductive capacity should be informed about the reproductive hazards where blood lead level may exceed 10µg/dL (0.48µmol/L). It is highly recommended that in order to give maximum protection to the foetus, women who are planning a pregnancy should endeavour to limit lead to a level **well below** 10µg/dL (0.48µmol/L) for a period of at least a year prior to pregnancy.

Statistics show one in four pregnancies in Australia is unplanned, and because there is limited information on bone-lead mobility during pregnancy it is prudent to maintain blood lead levels for females who may later become pregnant below 20µg/dL (0.97 µmol/L).

It is for these reasons females of reproductive capacity should endeavour not to seek employment in lead risk jobs.

In certain circumstances, conception methods like *in vitro* fertilisation may need to be considered in assessing reproductive capacity.

Infants are more susceptible to the health effects of lead than adults. A breast feeding worker should keep her blood lead level below 10µg/dL (0.48 µmol/L) and as low as possible.

APPENDIX 3

Health-based management recommendations for lead-exposed adults*

Blood lead level (µg/dL)	Short-term risks	Long-term risks	
	(lead exposure < 1 year)	**(lead exposure ≥1 year)**	**Medical Management Recommended**
< 5	None documented	None documented	None indicated
5–9	Possible spontaneous abortion	Possible spontaneous abortion	Discuss health risks
	Possible postnatal developmental delay	Possible postnatal developmental delay	Reduce lead exposure for women who are or may become pregnant
		Possible hypertension and kidney dysfunction	
10–19	Possible spontaneous abortion	Possible spontaneous abortion	As above for BLL 5–9 µg/dL, plus:
	Possible postnatal developmental delay	Reduced birth weight	Decrease lead exposure
	Reduced birth weight	Possible postnatal developmental delay	Increase biological monitoring
		Hypertension and kidney dysfunction	Consider removal from lead exposure to avoid long-term risks if exposure control over an extended period does not decrease BLL < 10 µg/dL, or
		Possible subclinical neurocognitive deficits	if medical condition present that increases risk with continued exposure[a]
20–29	Possible spontaneous abortion	Possible spontaneous abortion	Remove from lead exposure if repeat BLL measured in 4 weeks remains ≥20 µg/dL
	Possible postnatal developmental delay	Possible postnatal developmental delay	
	Reduced birth weight	Reduced birth weight	
		Hypertension and kidney dysfunction	
		Possible subclinical neurocognitive deficits	
30–39	Spontaneous abortion	Spontaneous abortion	Remove from lead exposure
	Possible postnatal developmental delay	Reduced birth weight	
	Reduced birth weight	Possible postnatal developmental delay	

* Table is reproduced from: Recommendations for Medical Management of Adult Lead Exposure, Michael J Kosnett, Richard P Wedeen, Stephen J Rothenberg, Karen L Hipkins, Barbara L Materna, Brian S Schwartz, Howard Hu, and Alan Woolf. Environmental Health Perspectives, Volume 115, Number 3, March 2007.

Blood lead level (μg/dL)	Short-term risks (lead exposure < 1 year)	Long-term risks (lead exposure ≥1 year)	Medical Management Recommended
		Hypertension and kidney dysfunction	
		Possible neurocognitive deficits Possible nonspecific symptoms[b]	
40–79	Spontaneous abortion	Spontaneous abortion	Remove from lead exposure
	Reduced birth weight	Reduced birth weight	Refer for prompt medical evaluation
	Possible postnatal developmental delay	Possible postnatal developmental delay	Consider chelation therapy for BLL > 50 μg/dL with significant symptoms or signs of lead toxicity
	Nonspecific symptoms[b]	Nonspecific symptoms[b]	
	Neurocognitive deficits	Hypertension	
	Sperm abnormalities	Kidney dysfunction/nephropathy	
		Subclinical peripheral neuropathy	
		Neurocognitive deficits	
		Sperm abnormalities	
		Anemia	
		Colic	
		Possible gout	
≥80	Spontaneous abortion	Spontaneous abortion	Remove from lead exposure
	Reduced birth weight	Reduced birth weight	Refer for immediate/urgent medical evaluation
	Possible postnatal developmental delay	Possible postnatal developmental delay	Probable chelation therapy
	Nonspecific symptoms[b]	Nonspecific symptoms[b]	
	Neurocognitive deficits	Hypertension	
	Encephalopathy	Nephropathy	
	Sperm abnormalities	Peripheral neuropathy	
	Anemia	Neurocognitive deficits	
	Colic	Sperm abnormalities	
		Anemia	
		Colic	
		Gout	

BLL = blood lead level.

a. Medical conditions that may increase the risk of continued exposure include chronic renal dysfunction (serum creatinine > 1.5 mg/dL for men and > 1.3 mg/dL for women, or proteinuria), hypertension, neurologic disorders, and cognitive dysfunction.

b Non-specific symptoms may include headache, fatigue, sleep disturbance, anorexia, constipation, arthralgia, myalgia, and decreased libido.

MERCURY (INORGANIC)

BASELINE HEALTH MONITORING BEFORE STARTING WORK IN AN INORGANIC MERCURY PROCESS

1. **Collection of Demographic Data**
2. **Work History**
3. **Medical History**
4. **Physical Examination**

A physical examination will be conducted with an emphasis on the dermatological, gastrointestinal, neurological and renal systems.

5. **Investigation**

Spot urine for inorganic mercury will be used to test the worker's baseline exposure. The result is corrected for creatinine, for example mercury concentration in micrograms per gram of creatinine. Where there is 50 µg of inorganic mercury or more per gram of creatinine, a repeat spot urine test should be performed at the same time of the day.

BACKGROUND INFORMATION ON MERCURY EXPOSURE

Concentrations of inorganic mercury in urine correlate with long-lasting exposures reflecting average exposure over the previous few months in those chronically exposed, while levels in blood reflect only the recent exposure. Recent visits to the dentist for amalgam fillings, use of disinfectants containing organomercury, and recent treatment with penicillin type antibiotics should be recorded as they can affect results.

Urine total mercury in the non-work exposed population is generally < 20µg/L[1].

DURING EXPOSURE TO INORGANIC MERCURY

6. **Monitoring exposure to inorganic mercury**

Urinary mercury is an indicator of average exposure during the past month rather than exposure at the time of urine collection. Throughout the day, the level of mercury in urine can vary. Occasional high levels of mercury in the urine should not be cause for immediate alarm and new samples should be taken. Acute exposure to mercury, for example through an accidental spill should be measured via blood testing.

It is recommended testing should occur after one month, three months, six months and thereafter at regular intervals at the discretion of the supervising medical practitioner, depending on the test results obtained. More frequent testing may be necessary where symptoms of exposure are evident or where test results indicate significant concern exists or workplace controls are not working.

However, a six-monthly testing pattern is recommended where the level is <25 µg inorganic mercury per gram of creatinine.

Sampling prior to a work shift assists in minimising the risk of external contamination.

1 To convert µg/L to µmol mercury/mol creatinine multiply by $\frac{\text{MW creatinine}}{\text{MW mercury}} = \frac{113.12}{200.59}$

For example 20 µg/L urine total mercury = 11.3 µmol mercury/mol creatinine.

ACTION LEVEL

Where testing shows a level of 25 µg inorganic mercury or more per gram of creatinine in urine, this may indicate the worker has been exposed[2]. In these instances:

- a repeat spot urine test should be performed at the same time of the day
- a medical examination should be conducted, with emphasis on the neurological, gastrointestinal, renal and dermatological systems
- the person conducting a business or undertaking should review control measures and carry out recommended remedial action
- the worker must be informed of the results of the health monitoring
- repeat spot urine for inorganic mercury tests should be conducted every six weeks until the level of inorganic mercury per gram of creatinine in urine falls below 25 µg inorganic mercury or more per gram of creatinine in urine.

Blood mercury should only be measured if an acute exposure, like during an accidental spill, has occurred within the last three to 20 days. The total inorganic mercury in blood at end of the shift at end of work week should be less than 15 µg/L. Eating seafood can interfere with the blood test results.

REMOVAL LEVEL

Although no significant sign of adverse health effects have been observed at urinary concentrations below 100 µg of mercury per gram of creatinine, altered psychological performance and renal enzyme activities have been reported between 35-100 µg mercury per gram of creatinine. On this basis it is recommended the worker is removed from mercury work when the level of mercury in urine is greater than 35 µg per gram of creatinine.

Where a worker shows signs of mercury poisoning or the level of inorganic mercury in urine is greater than 35 µg per gram of creatinine, the worker should immediately be removed from mercury work.

The worker's health should be monitored every 30 days until the level falls below 35 µg inorganic mercury per gram of creatinine on two successive occasions. Testing should occur every six weeks until the level of inorganic mercury in urine is less than 25 µg per gram of creatinine.

RETURN TO WORK

The worker must not return to mercury work until they have been assessed as medically fit to return to work by the medical practitioner supervising the health monitoring.

AT TERMINATION OF WORK IN AN INORGANIC MERCURY PROCESS

7. Final physical examination

A medical examination should be conducted to determine whether the worker has neurological or renal dysfunction due to exposure to inorganic mercury.

2 The American Conference of Governmental Industrial Hygienists (ACGIH) recommended biological exposure index (BEI) for a pre-shift end of work week is 35 µg/g creatinine (= 20 µmol/mol creatinine). The WorkCover NSW Biological Occupational Exposure Limit (BOEL) Committee also recommends this value.

SUPPLEMENTARY INFORMATION ON MERCURY (INORGANIC)

8. Work activities that may represent a high risk exposure

Mercury exists in three forms: liquid and vapour states (Hg^{o}) 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
- 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.

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

9. Non-work sources

A significant amount of organic mercury can be ingested in seafood as methyl mercury—the average intake for adults is seven micrograms per day. Release of mercury from dental amalgam fillings is about 3.5 micrograms per day [1]. The major source of temporarily increased levels of inorganic mercury in blood and urine is fresh dental amalgams. Urine and blood mercury concentrations transiently increase over a period of a week or more when dental restoration involving mercury amalgams is performed.

Inorganic mercury excretion may also be increased after exposure to disinfectants, for example mouthwash, and paints containing inorganic mercury. Mercury compounds are used in water-based latex paint to prevent mildew after the paint has been applied and as a preservative for paint in storage. Recent penicillin type antibiotics can increase urinary excretion of mercury, as the main degradation product of penicillin, penicillamine, enhances this process. Some skin lightening creams contain mercury. Mercury is ubiquitous in the environment, for example in ash or vapour from coal combustion.

POTENTIAL HEALTH EFFECTS FOLLOWING EXPOSURE TO INORGANIC MERCURY

10. 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 through the intestinal tract—less than one per cent—and skin. About 75-80 per cent of inhaled mercury vapour is absorbed across alveolar membranes into the bloodstream. Percutaneous absorption of mercury vapour is minimal. 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. Percutaneous absorption of ionic mercury salts can cause toxicity. Generally, mercuric salts are more soluble and more toxic than mercurous salts.

11. Target organ/effect

Nervous system – muscular tremor, increased excitability, memory loss, emotional ability, peripheral neuropathy.

Kidney – renal tubular necrosis.

Respiratory tract – irritation, pneumonitis, metal fume fever, dyspnoea, pulmonary fibrosis.

Gastrointestinal tract – acute gastroenteritis, stomatitis, metallic taste, excess salivation, ulceration or bleeding of gums.

Skin – corrosive blisters, contact dermatitis.

Hearing – potential ototoxin.

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, dyspnoea, fever, nausea, vomiting, diarrhoea and a metallic taste in the mouth. Stomatitis, colitis, nephrotic syndrome and salivation may occur. High concentrations cause corrosive bronchitis and interstitial pneumonitis. The uptake of mercury vapour into the central nervous system produces tremor and increased excitability. In milder cases the patient will recover in one to two weeks. 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 and ulcerative gingivitis may appear within 24 to 36 hours. Renal tubular necrosis may progress from transient polyuria to anuria.

In some people, metallic mercury has been shown to cause an allergic skin reaction.

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. No significant sign of adverse health effects have been observed at urinary concentrations of mercury below 100 µg/g creatinine. Occasionally, altered psychological performance and renal enzyme activities are reported at concentrations between 35 and 100 µg/g creatinine [2].

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 performs a defined action like writing, which may become so disordered by the tremor that it is illegible. The neurological disturbance or mercurial erethism 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.

Kidney dysfunction sometimes develops, especially in workers exposed to elemental mercury. However, the development of kidney dysfunction is not clearly linked with the intensity of exposure. After inorganic salts are ingested, a large amount of mercury may accumulate in the kidneys, producing a generalised increase in the permeability of the tubular epithelium.

Soluble inorganic mercury salts, like mercuric chloride, will devitalise tissue by denaturation and precipitation of the proteins present. 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 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.

12. Germ cell mutagen and reproductive toxicant classifications

Mercury is classified according to the GHS 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).

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There are two sections. Complete both sections and all questions if applicable.

Section 1 is to be forwarded to the PCBU who has engaged your services. A copy of laboratory report(s) must be attached > > > >

Section 2 may contain confidential information which may not be relevant to the health monitoring program being carried out. This section should be retained by the medical practitioner. Information which is required to be given to the PCBU should be summarised in part 7 of section 1.

SECTION 1 – THIS SECTION TO BE RETURNED TO THE PCBU			
1. PERSON CONDUCTING A BUSINESS OR UNDERTAKING			
Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
2. OTHER BUSINESSES OR UNDERTAKINGS ENGAGING THE WORKER			
Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
3. WORKER		(✓) all relevant boxes	
Surname:		Given names:	
Date of birth: DD/MM/YYYY	Sex:	☐ Male	☐ Female
Address:			
Suburb:			Postcode:
Current Job:	Tel(H):		Mob:
Date started employment : DD/MM/YYYY			
4. EMPLOYMENT IN MERCURY (INORGANIC) RISK WORK		(✓) all relevant boxes	
1. ☐ New to mercury (inorganic) work			
2. ☐ New worker but not new to mercury (inorganic) work			
3. ☐ Current worker continuing in mercury (inorganic) work			
4. Worked with mercury (inorganic) since DD/MM/YYYY			

5. Satisfactory personal hygiene (for example nail biting, frequency of hand washing)		☐ Yes	☐ No
6. Risk assessment completed		☐ Yes	☐ No
5. WORK ENVIRONMENT ASSESSMENT	(✓) all relevant boxes		
Date of assessment: DD/MM/YYYY			
Mercury (inorganic) Industry			
☐ Manufacture of Amalgams	**Controls:**		
☐ Dental Work	Wear gloves	☐ Yes	☐ No
☐ Manufacture of Paints	Respirator use	☐ Yes	☐ No
☐ Gold/Silver Extraction	Local exhaust ventilation	☐ Yes	☐ No
☐ Laboratory Work	Overalls / work clothing	☐ Yes	☐ No
☐ Fungicides	Laundering by employer	☐ Yes	☐ No
☐ Natural Gas Industry	Wash basins & showers (with hot & cold water)	☐ Yes	☐ No
☐ Other (specify):	Smoking or eating in workshop	☐ Yes	☐ No
	Personal hygiene:		
	Clean Shaven	☐ Yes	☐ No
	Shower & change into clean clothes at end of shift	☐ Yes	☐ No

6. BIOLOGICAL MONITORING RESULTS Include at least the previous two test results (if available)

Date	Tests performed	Recommended Action and/or Comment
1. DD/MM/YYYY		
2. DD/MM/YYYY		
3. DD/MM/YYYY		
4. DD/MM/YYYY		
5. DD/MM/YYYY		
6. DD/MM/YYYY		
7. DD/MM/YYYY		

7. RECOMMENDATIONS (by Medical Practitioner) (✓) all relevant boxes		
1. ☐ Suitable for work with mercury (inorganic)		
2. ☐ Counselling required		
3. ☐ Review workplace controls		
4. ☐ Repeat health assessment in _____ month(s) / _____ week(s)		
5. ☐ Removal from work with mercury (inorganic)	On DD/MM/YYYY	
6. ☐ Medical examination by Medical Practitioner	On DD/MM/YYYY	
7. ☐ Fit to resume mecury (inorganic) risk work	From DD/MM/YYYY	
8. ☐ Referred to Medical Specialist (respiratory/dermatology/other):	On DD/MM/YYYY	
Specialist's name:		
Additional comments or recommendations arising from health monitoring:		
Medical Practitioner (responsible for supervising health monitoring)		
Name:	Signature	Date: DD/MM/YYYY
Tel:	Fax:	Registration Number:
Medical Practice:		
Address:		
Suburb:		Postcode:

SECTION 2 – THIS SECTION TO BE RETAINED BY THE MEDICAL PRACTITIONER			
1. PERSON CONDUCTING A BUSINESS OR UNDERTAKING			
Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
2. OTHER BUSINESSES OR UNDERTAKINGS ENGAGING THE WORKER			
Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
3. WORKER		(✓) all relevant boxes	
Surname:		Given names:	
Date of birth: DD/MM/YYYY	Sex:	☐ Male	☐ Female ☐ Pregnant/Breast Feeding?
Address:			
Suburb:			Postcode:
Current Job:	Tel(H):		Mob:
Date started employment : DD/MM/YYYY			
4. GENERAL HEALTH ASSESSMENT (if applicable)			
Symptoms of:	**Comments**	**Further testing?**	
Skin disorders		☐ Yes	☐ No
Headaches, dizziness		☐ Yes	☐ No
Respiratory tract/GIT		☐ Yes	☐ No
Irritation of eyes, nose or throat		☐ Yes	☐ No
Cough		☐ Yes	☐ No
CNS		☐ Yes	☐ No
Others		☐ Yes	☐ No
Height _____cm Weight _____kg Bp ____/____ mmHg		☐ Yes	☐ No

5. OTHER MEDICAL HISTORY, FAMILY MEDICAL HISTORY, CURRENT MEDICATION, COMMENTS, TESTS OR RECOMMENDATIONS (use separate sheet if necessary)			
Medical Practitioner (responsible for supervising health monitoring)			
Name:	Signature		Date: DD/MM/YYYY
Tel:	Fax:	Registration Number:	
Medical Practice:			
Address:			
Suburb:		Postcode:	

4,4'-METHYLENE BIS(2CHLOROANILINE) [MOCA]

BASELINE HEALTH MONITORING BEFORE STARTING WORK IN A MOCA PROCESS

Workers must be informed of the potential health effects associated with exposure to MOCA. In particular, workers should be made aware of potential for MOCA to be absorbed readily through the skin and the need to maintain a high standard of personal hygiene and housekeeping practices.

1. **Collection of demographic data**
2. **Work history**
3. **Medical history**
4. **Physical examination**

A physical exam will be conducted only if indicated by work and medical history.

5. **Investigation**

The following tests will be used to test the worker's baseline exposure:

- dipstick urinalysis for haematuria
- urine cytology[1] may be required depending on the medical history and previous exposure.

DURING EXPOSURE TO A MOCA PROCESS

6. **Monitoring exposure to MOCA**

The following tests will be conducted twice annually at the time of peak exposure/use:

- urinary total MOCA
- spot creatinine corrected urine for total MOCA
- dipstick urinalysis for haematuria.

Dipstick urinalysis results will be compared with the worker's baseline dipstick urinalysis. Urine cytology will also be conducted annually.

The following values should be considered when assessing exposure to MOCA:

Biological level	Source
15 µmol MOCA/mol creatinine in urine	Workcover NSW Biological Occupational Exposure Limit (BOEL) Committee.
15 µmol MOCA/mol creatinine in urine	Health and Safety Executive (UK) Biological Monitoring Guidance Value (BMGV)[2]

- 1 See 'Biomarkers of Exposure and Effect' in Section 2- Supplementary Information on MOCA - of this Guideline for further information
- 2 BMGVs are not health based. They are practicable, achievable levels. A BMGV represents the 90th percentile of biological monitoring results from a representative sample of workplaces with good work hygiene practices. If a result is greater than the BMGV it does not necessarily mean that ill health will occur but does indicate that control of exposure may not be adequate.

Where testing shows a level of 15 μmol MOCA or more per mol of creatinine in urine, this may indicate the worker has been exposed. In these instances:

- repeat tests should be performed at the same time of the day
- a medical examination should be conducted and the medical practitioner supervising the health monitoring should consider removing the worker from MOCA work
- the person conducting a business or undertaking should review control measures and carry out recommended remedial action
- the worker must be informed of the results of the health monitoring.

Other information

Urine samples should be collected at end of a work shift at end of the work week. Samples should be collected in plastic containers and analysed without delay.

Care should be taken to prevent contamination of the urine samples. Samples should be collected in a clean room, following the removal of contaminated clothing and washing the hands.

AT TERMINATION OF WORK IN A MOCA PROCESS

7. Final medical examination

A final medical examination will be conducted and will include:

- urine cytology for haematuria
- dipstick urinalysis
- a medical review of health monitoring records.

8. Continuing medical monitoring

The worker should be reminded of the need for continuing urine cytology and dipstick urinalysis. Where practicable, the person who is conducting a business or undertaking should provide this service and remind workers of its availability.

SUPPLEMENTARY INFORMATION ON MOCA

9. Work activities that may represent a high risk exposure

MOCA is used as a curing agent in the production of hardened isocyanate-based polyurethane products. More specifically, MOCA is utilised in the manufacturing of wear-resistant industrial products like polyurethane gears, gaskets, belts, rollers, sport boots and roller skate wheels. The compound is commonly used as a coating to set other glues, plastics and adhesives.

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.

10. Non-work sources

Not known to occur in nature therefore non-work exposure is rare. Exposure has been reported from contact with contaminated soils.

POTENTIAL HEALTH EFFECTS FOLLOWING EXPOSURE TO MOCA

11. 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—visible MOCA granules on floors and work benches
- poor personal hygiene practices
- inadequate personal protection.

12. Target organ/effect

Blood – methaemoglobinaemia causing hypoxia and cyanosis.

Respiratory tract – irritation.

Eyes – irritation.

Kidney – haematuria, proteinuria.

Bladder – GHS Carcinogenicity Category 1B (presumed to have carcinogenic potential for humans).

13. Systemic effects

DERMAL/OCULAR EFFECTS

In a case report, a worker complained of burning face and eyes shortly after accidentally been sprayed in the face with molten MOCA [1]. In another case of accidental exposure, a worker described a burning sensation of the skin [2].

RENAL EFFECTS

In the case report [1] described above, protein was detected in the worker's urine within the first five hours after exposure, suggesting damage to the renal tubules had occurred.

In a retrospective bladder cancer incidence study conducted among 532 workers exposed to MOCA from 1968 to 1979 and 20 workers who were first employed in 1980 and 1981, 385 participated in a urine screening test [3]. Exfoliative cytology revealed 21 urine samples contained atypical cells. No cytology readings were positive for cancer. Sixteen urine samples were positive for haeme. In the course of the screening, one of the participants who had negative cytology and was negative for the presence of haeme in urine was diagnosed with a tumour of the urinary bladder.

14. Carcinogenicity

MOCA is an aromatic amine which is structurally similar to benzidine, a known human bladder carcinogen. MOCA has been shown to cause hepatomas in mice and rats, lung and mammary carcinomas in rats and bladder cancer in dogs [4,5,6].

A case study has described bladder cancer in two non-smoking males, under the age of 30, who were exposed to MOCA during its manufacture [7]. This is the only report of humans with a history of MOCA exposure developing bladder cancer.

15. Carcinogen classification

MOCA is classified according to the GHS as Carcinogen Category 1B (May cause cancer).

16. Biomarkers of exposure and effect

EXPOSURE

The best currently available indicator of absorption of MOCA is urinary total MOCA estimations based on spot creatinine corrected urines. Although this method is not without limitations, that is unmetabolised MOCA is measured and dose-response curves are lacking, its use is a reasonable means of monitoring the effectiveness of engineering controls, personal protective equipment and work practices including education. This has been demonstrated in Western Australia where urinary monitoring showed a decrease in MOCA levels after educating the workforce on safe-handling practices of MOCA [8].

MOCA levels are usually higher at the end of the shift and reflect exposure over the preceding two to three days. The biological half-life of MOCA in urine is approximately 23 hours [2].

EFFECT

Three techniques have been used to detect bladder cancer: urinalysis for haematuria; urine cytology and cystoscopy. All three methods can be utilised for early detection of the disease. Since 1995, a latex agglutination assay for the qualitative detection of bladder tumour antigen in urine has been available. The antigen is composed of basement membrane complexes that have been isolated and characterised from the urine of patients with bladder cancer. The test, apparently has a high specificity and sensitivity in high risk groups [9,10,11]. However, it has not been trialled as a screening test in a normal population.

Dipstick urinalysis has been reported to have a sensitivity of 91 per cent to 100 per cent for haematuria, variably defined as greater than two to greater than five erythrocytes per high powered field in the sediment of spun urine [12]. Haematuria in an asymptomatic person can be an indication of bladder cancer. In current population based studies, between zero and two per cent of adults with confirmed haematuria have definite significant disease [12]. Therefore, current recommendations are not to screen the general public for haematuria because the risks of the subsequent work-up do not justify screening [10]. However, as pointed out in subsequent correspondence to this observation, individuals in high risk groups should be considered for screening since bladder cancer is potentially treatable if detected at an early stage [13]. It should be noted that haematuria may be intermittent. A person with persistent haematuria requires the cause of the haematuria to be followed up.

Urine cytology has been used as a screening technique for work-related bladder cancer in the U.K. since the 1950's [14]. It is a technique which is easily reproducible when the laboratory personnel are adequately trained in preparation and interpretation of smears. Among fourteen groups of investigators the correlation of positive cytology with clinical findings ranged from 100 per cent to 26 per cent with a mean sensitivity of 71.6 per cent [14]. The false positive rates ranged from 11.3 per cent to 11.9 per cent. In both MOCA exposed bladder cancer cases reported by Ward et al 1988, urine cytology was negative. Urinary cytology screening is felt to be of little value in detecting low-grade papillary neoplasms [15]. However, urine cytology has had a favourable impact on patients whose disease is uncovered by this method with screened cases living 3.6 years longer than unscreened cases [16]. Therefore, screening urine cytology for groups at high risk for bladder cancer may provide early detection of the disease.

Finally, cystoscopy is another method which may be employed for bladder cancer screening. Cystoscopy is an invasive procedure which allows visualisation of the urinary bladder along with the ability to perform biopsies. However, this procedure lends itself to significant risk including the risks of anaesthesia. Therefore, cystoscopy will not be utilised as a screening procedure. The general population would reject having this test performed on a routine basis. However, if urinalysis for haematuria or urine cytology indicate a result where bladder cancer is considered then cystoscopy should be utilised to confirm the diagnosis.

Urinary measurement of total MOCA and the urinary bladder cancer detection technique of urinalysis for haematuria are the methods best suited for health monitoring of MOCA. Urine cytology, the antigen test and cystoscopy are useful as case management tools but not as routine screening tests. Cystoscopy has too many risks to consider as a mass screening procedure.

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FURTHER READING

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American Conference of Governmental Industrial Hygienists (ACGIH), *Documentation of the Threshold Limit Values for Chemical Substances*, 7th Ed, Cincinnati, 2011.

Cocker J, Boobis AR, Wilson HK and Gompertz D, 'Evidence that a ß-N-Glucuronide of 4,4'-Methylenebis (2-chloroaniline) (MbOCA) is a Major Urinary Metabolite in Man: Implications for Biological Monitoring', *British Journal of Industrial Medicine*, vol 47, pp 154-161, 1990.

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Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
2. OTHER BUSINESSES OR UNDERTAKINGS ENGAGING THE WORKER			
Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
3. WORKER		(✓) all relevant boxes	
Surname:		Given names:	
Date of birth: DD/MM/YYYY	Sex:	☐ Male	☐ Female
Address:			
Suburb:			Postcode:
Current Job:	Tel(H):		Mob:
Date started employment : DD/MM/YYYY			
4. EMPLOYMENT IN MOCA RISK WORK		(✓) all relevant boxes	
1. ☐ New to MOCA work			
2. ☐ New worker but not new to MOCA work			
3. ☐ Current worker continuing in MOCA work			
4. Worked with MOCA since DD/MM/YYYY			
5. Satisfactory personal hygiene (for example nail biting, frequency of hand washing)		☐ Yes	☐ No
6. Risk assessment completed		☐ Yes	☐ No

5. WORK ENVIRONMENT ASSESSMENT	(✓) all relevant boxes		
Date of assessment: DD/MM/YYYY			
MOCA Industry			
☐ Polyurethane Production ☐ Other (specify):	**Controls:**		
	Wear gloves	☐ Yes	☐ No
	Respirator use	☐ Yes	☐ No
	Process enclosed	☐ Yes	☐ No
	Worker isolated from exposure	☐ Yes	☐ No
	Local exhaust ventilation	☐ Yes	☐ No
	Overalls / work clothing	☐ Yes	☐ No
	Laundering by employer	☐ Yes	☐ No
	Wash basins & showers (with hot & cold water)	☐ Yes	☐ No
	Smoking or eating in workshop	☐ Yes	☐ No
	Personal hygiene:		
	Clean Shaven	☐ Yes	☐ No
	Shower & change into clean clothes at end of shift	☐ Yes	☐ No

6. BIOLOGICAL MONITORING RESULTS Include at least the previous two test results (if available)		
Date	**Tests performed**	**Recommended Action and/or Comment**
1. DD/MM/YYYY		
2. DD/MM/YYYY		
3. DD/MM/YYYY		
4. DD/MM/YYYY		
5. DD/MM/YYYY		
6. DD/MM/YYYY		
7. DD/MM/YYYY		

7. RECOMMENDATIONS (by Medical Practitioner)	(✓) all relevant boxes
1. ☐ Suitable for work with MOCA	
2. ☐ Counselling required	
3. ☐ Review workplace controls	
4. ☐ Repeat health assessment in _____ month(s) / _____ week(s)	
5. ☐ Removal from work with MOCA	On DD/MM/YYYY
6. ☐ Medical examination by Medical Practitioner	On DD/MM/YYYY
7. ☐ Fit to resume MOCA risk work	From DD/MM/YYYY
8. ☐ Referred to Medical Specialist (respiratory/dermatology/other):	On DD/MM/YYYY
Specialist's name:	
Additional comments or recommendations arising from health monitoring:	

Medical Practitioner (responsible for supervising health monitoring)		
Name:	Signature	Date: DD/MM/YYYY
Tel:	Fax:	Registration Number:
Medical Practice:		
Address:		
Suburb:		Postcode:

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1. PERSON CONDUCTING A BUSINESS OR UNDERTAKING			
Company / Organisation name:			
Site address:			
Suburb:		Postcode:	
Site Tel:	Site Fax:	Contact Name:	
2. OTHER BUSINESSES OR UNDERTAKINGS ENGAGING THE WORKER			
Company / Organisation name:			
Site address:			
Suburb:		Postcode:	
Site Tel:	Site Fax:	Contact Name:	
3. WORKER		(✓) all relevant boxes	
Surname:		Given names:	
Date of birth: DD/MM/YYYY	Sex: ☐ Male	☐ Female	☐ Pregnant/Breast Feeding?
Address:			
Suburb:		Postcode:	
Current Job:	Tel(H):	Mob:	
Date started employment : DD/MM/YYYY			
4. GENERAL HEALTH ASSESSMENT (if applicable)			
Symptoms of:	**Comments**	**Further testing?**	
Skin disorders		☐ Yes	☐ No
Headaches, dizziness		☐ Yes	☐ No
Respiratory tract		☐ Yes	☐ No
Eyes		☐ Yes	☐ No
Cough		☐ Yes	☐ No
CNS		☐ Yes	☐ No
Haematuria/proteinuria		☐ Yes	☐ No
Hypoxia/cyanosis		☐ Yes	☐ No
Others		☐ Yes	☐ No
Height _____cm Weight _____kg Bp _____/_____ mmHg		☐ Yes	☐ No

5. OTHER MEDICAL HISTORY, FAMILY MEDICAL HISTORY, CURRENT MEDICATION, COMMENTS, TESTS OR RECOMMENDATIONS (use separate sheet if necessary)		
Medical Practitioner (responsible for supervising health monitoring)		
Name:	Signature	Date: DD/MM/YYYY
Tel:	Fax:	Registration Number:
Medical Practice:		
Address:		
Suburb:		Postcode:

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ORGANOPHOSPHATE PESTICIDES

BASELINE HEALTH MONITORING BEFORE STARTING WORK IN AN ORGANOPHOSPHATE PESTICIDES PROCESS

1. **Collection of demographic data**
2. **Work history**
3. **Medical history**
4. **Physical examination**

A physical examination will be conducted only if work and medical history indicates this is necessary, for example by the presence of relevant symptoms.

5. **Investigation**

The following tests will be used to test the worker's baseline exposure:

- Estimation of red cell and plasma cholinesterase activity levels by the Ellman method. A venous blood sample is recommended. At least one, and ideally two, pre-exposure tests should be performed at least three days apart and the baseline obtained by averaging these tests. The results of these tests should be within 15 per cent to be regarded as reliable.

Note: If the worker has had previous exposure, then it is desirable a period of four weeks of no exposure should occur before the base-line level is established—see section 21, Baseline levels of serum and erythrocyte cholinesterase activity.

DURING EXPOSURE TO ORGANOPHOSPHATE PESTICIDES

6. **Monitoring exposure to organophosphate pesticides**

Periodic testing of workers during organophosphate pesticides use is desirable. The medical examination will include:

- work history
- medical history
- physical examination including look for evidence of dermatitis on the hands and forearms—this may indicate advice is required on work practices
- estimation of red cell and plasma cholinesterase activity levels by the Ellman method. It is preferable the estimation be done in the latter half of the working day when organophosphate pesticides are used. If a 20 per cent depression of cholinesterase activity is seen the worker should be re-tested.

The medical practitioner supervising the health monitoring program may also use the Urine Alkyl Phosphate Metabolites test (urine test) [1]. The urine test is a sensitive indicator of exposure to organophosphate pesticides and may indicate lower level exposures that would not typically result in a shift in blood cholinesterase levels. The urine test includes the metabolites dimethylphosphate (DMP), diethylphosphate (DEP), dimethylthiophosphate (DMTP), diethylthiophosphate (DETP), dimethyldithiophosphate (DMDTP) and diethyldithiophosphate (DEDTP).

7. Other information

The frequency of ongoing monitoring should be determined based on an assessment of the type of work, pattern of exposure, the pesticide(s) being handled or used and work practices in consultation with the medical practitioner supervising the health monitoring, see Table 1 for further information on pattern of use and action required.

Sample collection should occur at the end of the work shift at the end of the working week at the time of peak exposure.

Advantages of urine metabolite testing:

- worker can collect urine
- non-invasive test compared to blood sampling
- the analytical test is sensitive. It detects low level exposures when no shift in cholinesterase activity can be measured
- a baseline pre-exposure level is not required.

Disadvantages:

- no biological exposure limits currently exist.

8. Guidelines for interpreting results

Where urine analysis is performed, the following values should be considered when assessing exposure to organophosphate pesticides.

Biological level	Source
Levels of dialkyl phosphates in urine approaching 100 µmol/mol creatinine indicate low occupational exposure and is equivalent to high non-occupational exposure.	WorkCover NSW Chemical Analysis Branch Handbook 8th edition
Levels of dialkyl phosphates in urine between 100 and 1000 µmol/mol creatinine indicate medium occupational exposure.	
Levels of dialkyl phosphates in urine above 1000 µmol/mol creatinine indicate high occupational exposure and may be associated with a drop in the blood cholinesterase level.	
For workers with chronic exposure to organophosphates, the dialkyl phosphate level in urine may also be associated with a drop in the blood cholinesterase level.	

Where testing indicates high occupational exposure, the following action should be taken:

- A repeat urine test should be performed at the same time of the day.
- Estimation of red cell and plasma cholinesterase activity levels by the Ellman method and results compared with the worker's baseline test results.
- A medical examination should be conducted.
- The person conducting a business or undertaking should review control measures and carry out recommended remedial action.
- The worker must be informed of the results of the health monitoring.

9. Removal from organophosphate pesticide exposure

If there is a fall in cholinesterase activity by 40 per cent or more the worker should be removed from further exposure to the organophosphate pesticides until the level returns to baseline levels.

The worker can be moved to another area or can use other classes of pesticides, except pyrethroids (like permethrin) and carbamates.

AT TERMINATION OF WORK IN AN ORGANOPHOSPHATE PESTICIDES PROCESS

10. Final medical examination

A final medical examination will be conducted.

SUPPLEMENTARY INFORMATION ON ORGANOPHOSPHATE PESTICIDES

11. Work activities that may represent a high risk exposure

Organophosphorous compounds are derived from phosphoric and thiophosphoric acids. Individual organophosphate pesticides vary widely in acute toxicity but collectively they are among the most acutely toxic of all pesticides to mammals. The organophosphorous class of compound consists of organophosphates and also organophosphorodithiolates, organophosphorothiolates and organophosphorothionates which contain sulphur as well as phosphorus.

Most organophosphorous compounds are insecticides, although there are also a number of related herbicide and fungicide compounds. A list of the registered organophosphorous pesticides in use in Australia is provided at Appendix 2.

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. Less toxic pyrethrums and synthetic pyrethroids are replacing many of the currently used organophosphates.

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

- pest control operators who use OP every day in their work
- manufacture and packaging
- transport, storage and distribution
- handling used containers, for example in scrap recovery
- agricultural and horticultural activities like mixing, loading and applications 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.

12. Non-work sources

Many of these products are commonly used in home and garden products. For example, diazinon and chlorpyrifos have been widely used by consumers who may contaminate themselves or their food by not understanding the precautions necessary for safe use. Cases have been reported of acetylcholinesterase inhibition by a wide variety of drugs, alkyl sulphates and sulphonates, for example neostigmine, physostigmine, pyridostigmine, pethidine, some immunosuppressants and various cytostatic agents.

POTENTIAL HEALTH EFFECTS FOLLOWING EXPOSURE TO ORGANOPHOSPHATE PESTICIDES

13. Mode of action – Toxic effects

Organophosphorous compounds owe their toxic effect to the inhibition of cholinesterase enzyme activity in the nervous tissue. There are different types of cholinesterases in the human body, which differ in their location in tissues, substrate affinity and physiological function. The principal ones are acetylcholinesterase (AChE), which besides nervous tissues is also present in red blood cells, and serum cholinesterases which are a group of enzymes present in glial cells, plasma and liver. The effects induced by OP compounds in the organism are due to the inhibition of AChE; serum cholinesterase is inhibited as well, but with no apparent functional impairment. Acetylcholinesterase, under normal physiological conditions, performs the breakdown of acetylcholine, which is the chemical mediator responsible for physiological transmission of nerve impulses at different sites. In the presence of OP, AChE is phosphorylated and is no longer able to break down acetylcholine into choline and acetic acid. The resulting accumulation of acetylcholine in the parasympathetic nerve synapses (muscarinic-like action), the motor end-plate (nicotine-like action) and in the central nervous system is responsible for all typical symptoms occurring after acute poisoning with OP.

14. Route of entry into the body

For most OP, dermal 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 [2]. If the concentrate of any of the more toxic OP is splashed into the eye, absorption may be very rapid.

15. Target organ/effect

Central nervous system – headache, anxiety, restlessness, confusion, slurred speech, convulsions, coma and depression of the respiratory and circulatory centres.

Peripheral nervous system – muscarinic receptors in exocrine glands, smooth muscles and heart; nicotinic receptors in motor nerves to skeletal muscle and autonomic ganglia.

16. Acute toxicity

Typical symptoms of acute exposure to OP include excessive sweating, slurred speech and blurred vision.

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. The symptoms of intoxication can be divided into muscarine-like and nicotine-like effects, as well as effects on the central nervous system, see Table 2.

Local effects at the site of exposure may occur without symptoms and signs of systemic absorption. A splash in the eye may cause blurred vision due to spasm of accommodation. Inhalation may cause bronchoconstriction 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.

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.

17. Chronic toxicity

Symptoms of poisoning usually do not occur until 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. The erythrocyte cholinesterase is not reactivated. Its regeneration depends upon the replacement of erythrocytes in the peripheral blood which only occurs at the rate of about one per cent per day.

Continual exposure may cause persistent anorexia, weakness and malaise. Certain neurobehavioural effects may be seen.

Delayed polyneuropathy can occur from inhibition of another nervous tissue esterase called neuropathy target esterase. This mechanism appears to be related to protein changes occurring in this inhibited enzyme over time. The interval between acute exposure and the onset of neuropathy may be up to four weeks. Initial symptoms are often sensory and consist of tingling and burning sensations in the hands and feet followed by weakness in the lower limbs and ataxia. In severe cases the upper limbs may be affected. There is no specific treatment for this disorder although physiotherapy may limit the muscle wasting which follows denervation.

Combined toxicological data from epidemiology studies and from bioassays demonstrate the potential for organophosphates to produce a wide range of ophthalmological effects [2].

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

18. GHS carcinogen, germ cell mutagen and reproductive toxicant classifications

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

DEFINITIONS OF PATTERNS OF USE

Even though all OP compounds have a common mechanism of action, their effectiveness as inhibitors of AChE varies widely. Further, OP compounds can be classified as direct or indirect inhibitors of AChE. Direct inhibitors are effective without further metabolic modification after absorption into the body. Indirect inhibitors need to be transformed in the body to be effective. Thiono OP, that is those containing a P=S bond (mainly the phosphorothioates and phosphorodithioates) are not active inhibitors of AChE, but require activation by oxidation of the P=S to the P=O group. The practical importance of this classification is that direct inhibitors cause symptoms and signs to appear quickly during or after exposure, providing an early warning; whereas in the case of indirect inhibitors symptoms and signs appear later and the effects last longer after cessation of exposure. The insecticide dichlorvos is an example of a direct inhibitor while malathion and parathion are indirect inhibitors.

The organophosphorylated enzyme complex is relatively stable so that acetylcholinesterase inhibition tends to be prolonged. However, the rate of acetylcholinesterase reactivation is variable and can occur overnight in many cases of minor exposure. Over time dealkylation occurs in the inhibited enzyme making it more resistant to reactivation by oxime antidotes. This process is known as ageing.

Organophosphorous compounds also inhibit tissue carboxyesterases. Although this does not result in direct toxicity, it may increase the toxicity of other pesticides like most pyrethroids that are detoxified by carboxyesterases (i.e. toxicity is reduced through metabolism involving carboxyesterases).

Table 1 Definition of pattern of use and action required

Definition of pattern of use	Action required
Baseline	■ Baseline measurement should be carried out – two are desirable – at a time when there has been at least four weeks without exposure.
Very occasional use If use of organophosphate pesticides is only half a day every month or less, then this is *very occasional use*.	■ 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.
Intermittent use 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 *intermittent use.*	■ 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.
Seasonal use If use of organophosphate pesticides is say four days a week, and extends over a long season then, this is *seasonal use*.	■ 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 then be made if necessary. Workers should be advised of their results (percentage depression of cholinesterase from their baseline values). ■ The timing of further tests, should be based on the nature of the work and 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.

Table 2 Signs and symptoms of organophosphate poisoning

Nervous tissue and receptors affected	Site affected	Manifestations
Parasympathetic autonomic (muscarinic receptors) post ganglionic nerve fibres	Exocrine glands	Increased salivation, lacrimation, perspiration.
	Eyes	Miosis (pinpoint and non reactive) ptosis, blurring of vision, conjunctival injection, 'bloody-tears'.
	Gastrointestinal tract	Nausea, vomiting, abdominal tightness, swelling and cramps, diarrhoea, tenesmus, faecal incontinence.
	Respiratory tract	Excessive bronchial secretions, rhinorrhoea, wheezing, oedema, tightness in chest, bronchospasms, bronchoconstriction, cough, bradypnoea, dyspnoea.
	Cardiovascular system	Bradycardia, decrease in blood pressure.
	Bladder	Urinary frequency and incontinence.
Parasympathetic and sympathetic autonomic fibres (nicotinic receptors)	Cardiovascular system	Tachycardia, pallor, increase in blood pressure.
Somatic motor nerve fibres (nicotine receptors)	Skeletal muscles	Muscle fasciculations (eyelids, fine facial muscles), cramps, diminished tendon reflexes, generalised muscle weakness in peripheral and respiratory muscles, paralysis, flaccid or rigid tone.
		Restlessness, generalised motor activity, reaction to acoustic stimuli, tremulousness, emotional lability, ataxia.
Brain (acetylcholine receptors)	Central nervous system	Drowsiness, lethargy, fatigue, mental confusion, inability to concentrate, headache, pressure in head, generalised weakness.
		Coma with absence of reflexes, tremors, Cheyne-Stokes respiration, dyspnoea, convulsions, depression of respiratory centres, cyanosis.

BIOMARKER OF EFFECT

19. Erythrocyte cholinesterase

Erythrocyte cholinesterase is the same enzyme (acetyl cholinesterase) that is involved in the transmission of nerve impulses across the nerve synapses and neuromuscular junction. Measurement of erythrocyte cholinesterase is an indirect measure of the enzyme activity that exists in nerve tissue. Erythrocyte cholinesterase shows no difference in activity between sexes when the sex-related difference in red-cell packed volume is taken into consideration. Increased values may be found in polycythaemia and in thalassaemia or other congenital blood dyscrasias. Low values of erythrocyte cholinesterase not related to OP exposure have been observed in subjects affected with leukaemias or other neoplasms.

20. Serum cholinesterase

Serum cholinesterase is synthesised in the liver. Serum cholinesterase shows normal values 10-15 per cent greater in males than in females. Low values of serum cholinesterase activity not related to OP exposure may be found in liver diseases or drugs affecting the liver, uraemia, cancer, heart failure, allergic reactions, certain collagen diseases, acute infections, chronic anaemia and genetic variants which have a lower activity (suxamethonium sensitive individuals). In females lower values are also measured during pregnancy and menstruation. Serum cholinesterase activity can be increased in genetic variants and sometimes when the patient has obesity, hypertension, psoriasis, thyrotoxicosis or asthma.

Measurement of blood cholinesterase activity is an accepted method for biological effect monitoring of worker exposure to OP. This approach measures the common effect of this class of pesticide on certain enzyme activities. The number of organophosphorous pesticides in current use is very large (see Appendix 2) and it is unlikely it would be viable to directly analyse more than a few of the most widely used pesticides.

Red blood cell cholinesterase levels reflect exposure within the previous 120 days, that is the life of the red blood cell. In acute poisoning, a high inhibition of red blood cell cholinesterase is usually detected [3]. Mild symptoms of poisoning like nausea, vomiting, diarrhoea, salivation, lacrimation, sweating, wheeze, slow pulse, headache and dizziness are said to occur at a red blood cell cholinesterase inhibition of 50 per cent [3].

Plasma cholinesterase reflects exposure in the preceding two to three weeks and is affected by recent acute exposure. When comparing cholinesterase levels, red cell cholinesterase results should be compared with other red cell cholinesterase results, and plasma cholinesterase results with plasma cholinesterase results. It is not valid to compare red cell cholinesterase with plasma cholinesterase results.

21. Baseline levels of serum and erythrocyte cholinesterase activity [4]

It is essential to establish a baseline level for both serum and erythrocyte cholinesterase activity in each worker prior to initial exposure. If the worker has had previous exposure, then a period of four weeks of no exposure should occur before attempting to measure a baseline level. If possible, two attempts at measuring pre-exposure cholinesterase activity should be made. If the values obtained agree within 10 per cent then the individual baseline can be regarded as reliable. In practice it has been found that many new workers who have done no direct spraying have nevertheless been indirectly exposed by being in the vicinity of other workers who are handling or spraying the insecticide. Thus in the usual situation where a new worker is being trained, a cholinesterase activity result can only be accepted as a baseline level if it is established the training technician has not used this class of pesticides during instruction.

There are two important reasons to establish an individual baseline level of cholinesterase activity. Firstly, the reference range for cholinesterase activity is quite wide. Thus an individual may have an initial baseline cholinesterase activity in the higher part of this range. If this individual has work-related exposure then there may be a significant fall in that individual's cholinesterase activity, yet the result may still be within the reference range. Commonly, many workers have had mild chronic depression of cholinesterase activities reported as 'normal' for years simply because a comparison with baseline activity has never been made. Thus there is a danger of not recognising chronic low-level work-related exposure if attention is only focused on whether or not the cholinesterase result lies within the reference range. It is recommended an individual's current cholinesterase activity always be compared with their baseline cholinesterase activity.

Secondly, some individuals are born with a genetic deficiency in cholinesterase activity. Thus when doing initial screening to establish baseline levels it can be anticipated about three per cent of individuals will have this deficiency to some degree and hence will have lower than average cholinesterase activities. It does not appear such individuals are more at risk than those that do not have this deficiency. Thus they can be permitted to start using anticholinesterase pesticides. This deficiency should be confirmed by measuring either the dibucaine or fluoride numbers which bear a relationship to the serum cholinesterase genotype.

It is inappropriate to remove a person from further work exposure if the cholinesterase activity reflects a genetic deficiency rather than current work exposure. However, if a baseline cholinesterase activity has not been established in these individuals then they may be removed from further exposure and retested a number of times before it becomes apparent the worker is probably genetically deficient rather than work exposed. At this time the dicubaine or fluoride numbers should be determined to confirm this suspicion. However, this diagnosis is made typically when several months of restricted productivity on the part of the worker has occurred during the busiest part of the season. Therefore, it is more efficient to establish baseline cholinesterase activities.

BLOOD SAMPLE COLLECTION ARRANGEMENTS

Rural communities have to overcome special difficulties with collection, transport and storage of blood samples. An arrangement or plan could be negotiated with a local hospital or authorised doctor, so that blood tests suitable for monitoring organophosphate pesticide exposure would be conducted at a particular seasonal time and then at an appropriate time of the day or week, for occasional or intermittent users. This arrangement would cover the majority of situations. Emergencies require emergency protocols.

Specimens of whole blood should be collected in heparinised tubes and forwarded without delay to a laboratory equipped for cholinesterase determinations. In hot weather, and for long journeys, samples should be iced, not dry ice or frozen.

Plasma samples may not give a true indication of the cholinesterase level if sample collection is delayed after the last exposure has occurred. In the case of minor poisoning, if there has been a delay in collecting the sample of say 48 hours, then the subject's serum cholinesterase may have regenerated to its normal level. However, the erythrocyte cholinesterase activity would still be inhibited and this is the activity which should be measured.

Regeneration of serum cholinesterase will also occur, but more slowly if there is a delay in specimen transport. Specimens should be transported to the laboratory as quickly as possible and certainly within five days.

Normally a heparinised whole blood sample is submitted for analysis so that both plasma and erythrocyte cholinesterase levels can be determined. However, if the sample is haemolysed, only whole blood cholinesterase is reported.

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FURTHER READING

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APPENDIX 1

This health monitoring report is a confidential health record and must not be disclosed to another person except in accordance with the Work Health and Safety Regulations or with the consent of the worker.

There are two sections. Complete both sections and all questions if applicable.

Section 1 is to be forwarded to the PCBU who has engaged your services. A copy of laboratory report(s) must be attached > > > >

Section 2 may contain confidential information which may not be relevant to the health monitoring program being carried out. This section should be retained by the medical practitioner. Information which is required to be given to the PCBU should be summarised in part 7 of section 1.

SECTION 1 – THIS SECTION TO BE RETURNED TO THE PCBU			
1. PERSON CONDUCTING A BUSINESS OR UNDERTAKING			
Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
2. OTHER BUSINESSES OR UNDERTAKINGS ENGAGING THE WORKER			
Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
3. WORKER		(✓) all relevant boxes	
Surname:		Given names:	
Date of birth: DD/MM/YYYY	Sex:	☐ Male	☐ Female
Address:			
Suburb:			Postcode:
Current Job:		Tel(H):	Mob:
Date started employment : DD/MM/YYYY			
4. EMPLOYMENT IN ORGANOPHOSPHATE PESTICIDES RISK WORK (✓) all relevant boxes			
1. ☐ New to organophosphate pesticides work			
2. ☐ New worker but not new to organophosphate pesticides work			
3. ☐ Current worker continuing in organophosphate pesticides work			
4. Worked with organophosphate pesticides since DD/MM/YYYY			
5. Which organophosphate pesticides have you used?			

6. When were they last used?			
7. Satisfactory personal hygiene (for example nail biting, frequency of hand washing)	☐ Yes	☐ No	
8. Risk assessment completed	☐ Yes	☐ No	
Pattern of exposure	☐ Frequent (daily; 5 or more days in a work week)		
	☐ Regular (2-3 days in a work week)		
	☐ Occasional (2-3 days in a work month)		
	☐ Infrequent (1 day or less in a work month)		
	☐ Seasonal (several days a week for a season)		
Duration of exposure	☐ Long – 6 or more hours in a day		
	☐ Short – 1-5 hours in a day		
	☐ Brief – less than 1 hour a day		
	☐ Minimal – describe		
5. WORK ENVIRONMENT ASSESSMENT	(✓) all relevant boxes		
Date of assessment: DD/MM/YYYY			
Organophosphate pesticides being used:			
Organophophate Pesticide Industry			
☐ Pest Control	**Controls:**		
☐ Manufacture & Packaging	Wear gloves	☐ Yes	☐ No
☐ Transport/Storage/Distribution	Safety goggles / face shield	☐ Yes	☐ No
☐ Agricultural Industry	Respirator use	☐ Yes	☐ No
☐ Arial Crop Spraying	Local exhaust ventilation (if indoors)	☐ Yes	☐ No
☐ Horticultural Industry	Overalls / work clothing	☐ Yes	☐ No
☐ Veterinary/Farming	Laundering by employer	☐ Yes	☐ No
☐ Seasonal Field Work	Wash basins & showers (with hot & cold water)	☐ Yes	☐ No
☐ Laboratory Work	Smoking or eating in workshop	☐ Yes	☐ No
☐ Other (specify):	**Personal hygiene:**		
	Clean Shaven	☐ Yes	☐ No
	Shower & change into clean clothes at end of shift	☐ Yes	☐ No

6. BIOLOGICAL MONITORING RESULTS (Use either 6A or 6B) include at least the previous two test results (if available)

6A – Blood cholinesterase test (Ellman method)

Test date	RBC Cholinesterase (kU/L)	Plasma Cholinesterase (kU/L)	Timing	Comment % fall in cholinesterase
DD/MM/YYYY			Baseline 1 (B1)	Test when exposure-free for 30 days
DD/MM/YYYY			Baseline 2 (B2)	Re-test 3-14 days later, exposure-free
DD/MM/YYYY			Baseline 3 (B3)	Re-test only if (B1)-(B2) exceeds 20%
DD/MM/YYYY			**Average of Baseline results**	**Compare test results to Baseline results**
Test date	**RBC Cholinesterase (kU/L)**	**Plasma Cholinesterase (kU/L)**	**Timing**	**Comment % fall in cholinesterase**
DD/MM/YYYY			☐ Pre-shift ☐ Post-shift	
DD/MM/YYYY			☐ Pre-shift ☐ Post-shift	
DD/MM/YYYY			☐ Pre-shift ☐ Post-shift	

Note:
1. Baseline – Ideally two pre-exposure tests should be performed at least three days apart, no sooner than 30 days after prior exposure. Reliability is indicated by the two tests being within 10% of each other.
2. Periodic testing should occur during the period of organophosphate pesticide use (latter half of work day).
3. ≥ 20% fall in cholinesterase – Re-test
4. ≥ 40% fall in cholinesterase – Remove from exposure until test results return to baseline level.

6B – Urinary metabolite test (dialkyl phosphate (DAP) metabolites)

Test date	Urinary DAP metabolites (µmol/mol creatinine)	Timing	Comment
DD/MM/YYYY		☐ Pre-shift ☐ Post-shift ☐ Next day	
DD/MM/YYYY		☐ Pre-shift ☐ Post-shift ☐ Next day	
DD/MM/YYYY		☐ Pre-shift ☐ Post-shift ☐ Next day	

Note:
1. <100 µmol/mol creatinine – considered low work exposure.
2. 100-1000 µmol/mol creatinine – indicates work exposure – review workplace controls to reduce exposure levels.
3. >1000 µmol/mol creatinine – indicates high work exposure; may be associated with a fall in blood cholinesterase levels.

7. RECOMMENDATIONS (by Medical Practitioner)	(✓) all relevant boxes
1. ☐ Suitable for work with organophosphate pesticides	
2. ☐ Counselling required	
3. ☐ Review workplace controls	
4. ☐ Repeat health assessment in _____ month(s) / _____ week(s)	
5. ☐ Removal from work with organophosphate pesticides	On DD/MM/YYYY
6. ☐ Medical examination by Medical Practitioner	On DD/MM/YYYY
7. ☐ Fit to resume organophosphate pesticides risk work	From DD/MM/YYYY
8. ☐ Referred to Medical Specialist (respiratory/dermatology/other):	On DD/MM/YYYY
Specialist's name:	
Additional comments or recommendations arising from health monitoring:	

Medical Practitioner (responsible for supervising health monitoring)		
Name:	Signature	Date: DD/MM/YYYY
Tel:	Fax:	Registration Number:
Medical Practice:		
Address:		
Suburb:		Postcode:

SECTION 2 – THIS SECTION TO BE RETAINED BY THE MEDICAL PRACTITIONER			
1. PERSON CONDUCTING A BUSINESS OR UNDERTAKING			
Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
2. OTHER BUSINESSES OR UNDERTAKINGS ENGAGING THE WORKER			
Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
3. WORKER		(✓) all relevant boxes	
Surname:		Given names:	
Date of birth: DD/MM/YYYY	Sex:	☐ Male	☐ Female ☐ Pregnant/Breast Feeding?
Address:			
Suburb:			Postcode:
Current Job:		Tel(H):	Mob:
Date started employment : DD/MM/YYYY			
4. GENERAL HEALTH ASSESSMENT (if applicable)			
Symptoms of:	**Comments**	**Further testing?**	
Skin disorders		☐ Yes	☐ No
Headaches, dizziness		☐ Yes	☐ No
Respiratory tract		☐ Yes	☐ No
Eyes		☐ Yes	☐ No
Cough		☐ Yes	☐ No
CNS		☐ Yes	☐ No
Peripheral nervous system		☐ Yes	☐ No
Others		☐ Yes	☐ No

Height _____cm Weight _____kg Bp ____/____ mmHg		☐ Yes ☐ No

5. OTHER MEDICAL HISTORY, FAMILY MEDICAL HISTORY, CURRENT MEDICATION, COMMENTS, TESTS OR RECOMMENDATIONS (use separate sheet if necessary)

Any previous symptoms associated with organophosphate pesticides?	☐ Yes	☐ No

Other relevant conditions					
Pregnant	☐ Yes	☐ No	Cancer	☐ Yes	☐ No
Liver disease	☐ Yes	☐ No	Thyroid disease	☐ Yes	☐ No
Kidney disease	☐ Yes	☐ No	Heart disease	☐ Yes	☐ No
Add more details below if necessary			Crohn's Disease	☐ Yes	☐ No
Medications currently taken that may affect blood test results:					
Oral contraceptive pill	☐ Yes	☐ No	Prednisone/cortisone	☐ Yes	☐ No
Lithium	☐ Yes	☐ No	Propanolol	☐ Yes	☐ No

Medical Practitioner (responsible for supervising health monitoring)

Name:	Signature	Date: DD/MM/YYYY
Tel:	Fax:	Registration Number:
Medical Practice:		
Address:		
Suburb:		Postcode:

APPENDIX 2 LIST OF ORGANOPHOSPHATE PESTICIDES

The following list of cholinesterase-inhibiting organophosphate chemicals are approved for use in Australia, as of March 2012)*.

To be legally used in Australia products must be registered by the Australian Pesticides and Veterinary Medicines Authority (APVMA). Since product registrations change over time, the person conducting the business or undertaking should check the product label and safety data sheet to determine whether the chemical products used contain cholinesterase inhibiting (anti-cholinesterase) compounds.

Organophosphate pesticides

Acephate
Azamethiphos
Azinphos-methyl
Cadusafos
Carbofuran
Chlorfenvinphos
Chlorpyrifos
Chlorpyrifos-methyl
Coumaphos
Cythioate
Diazinon
Dichlorvos
Dimethoate
Disulfoton
Ethion
Fenamiphos
Fenitrothion
Fenthion
Maldison (Malathion)
Methamidophos
Methidathion
Mevinphos
Naled
Naphthalophos
Omethoate
Oxydemeton-methyl
Parathion
Parathion-methyl
Phorate
Phosmet
Pirimiphos-methyl
Profenofos
Propetamphos
Prothiofos
Temephos
Terbufos
Tetrachlorvinphos
Thiometon
Trichlorfon

* The Australian Pesticides and Veterinary Medicines Authority (APVMA) is the national agency responsible for assessing and registering agricultural and veterinary chemical products and controlling them up to the point of retail sale. The States and Territories are responsible for control-of-use aspects, like licensing of pest control operators and aerial sprayers. All agricultural and veterinary chemical products containing approved active ingredients are required to bear approved labels stating what the active ingredient is and its percent concentration in that product.

POLYCYCLIC AROMATIC HYDROCARBONS (PAH)

BASELINE HEALTH MONITORING BEFORE STARTING WORK IN A PAH PROCESS

1. **Work history**
2. **Medical history**

The following details about the worker's medical history will be collected by the medical practitioner:

- records of personal exposure, including photosensitivity
- presence of symptoms, see supplementary information in this Guide
- smoking history.

3. **Physical examination**

A physical examination will be conducted if indicated by work and medical history.

DURING EXPOSURE TO A PAH PROCESS

4. **Photosensitivity**

Photosensitivity is a known symptom of exposure to PAH. Where workers report photosensitivity, an appointment should be arranged with the medical practitioner and workers should receive additional counselling on the potential health effects of PAH on the skin.

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

5. **Monitoring exposure to PAH**

The assessment of work-related exposure to PAH is difficult because workers are exposed to a mixture of compounds. However, the metabolite of pyrene, 1-hydroxypyrene (1-HP) in urine, is most often used as the biomarker for PAH exposure as pyrene is a very thermodynamically stable compound and therefore most abundant in a PAH mixture.

The registered medical practitioner may choose to assess exposure to PAH through urine analysis. Where urine analysis is undertaken, the following values should be considered when assessing exposure to PAH:

Biological level	Source
1 µg 1-HP/L urine	Workcover NSW Biological Occupational Exposure Limit (BOEL) Committee. *Note: this value is under review.*
< 0.3 µg 1-HP/L urine – unexposed non-smokers 0.5 µg 1-HP/L urine – median for non-exposed smokers 1 µg 1-HP/L urine (benchmark value)	American Conference of Governmental Industrial Hygienists (ACGIH)
4 µmol 1-HP/mol creatinine in urine (benchmark guidance value)	Health and Safety Executive (UK)

Note: 1.0 μg of 1-HP/L urine = 1.4 μmol of 1-HP/mol creatinine

Where results of urine testing indicate there may be high workplace exposure to PAH, the registered medical practitioner should consider providing the person conducting the business or undertaking (PCBU) with the following recommendations:

- the worker should be removed from PAH work
- the PCBU should review control measures and carry out recommended remedial action
- the worker must be informed of the results of the health monitoring.

OTHER INFORMATION

Measurement of airborne levels of PAHs fails to take into account the potential pathway of skin absorption, which can contribute significantly to the total internal dose. The levels of 1-hydroxypyrene in the urine can increase during the course of a workday, reaching maximum values three to nine hours after the end of exposure. If the contribution of dermal exposure is important, post-shift 1-hydroxypyrene excretion can be lower than pre-shift levels when the worker has been exposed to PAH on the day prior to sampling. The difference between beginning and end of workweek excretion gives an indication of the average exposure over the workweek.

Note: Other hydroxylated metabolites of PAH have been proposed as markers of PAH exposure, however, currently correlation between metabolite levels and exposure have not been determined.

AT TERMINATION OF WORK IN A PAH PROCESS

6. Final medical examination

A final medical examination will be conducted and will include health advice including the recognition of photosensitivity and skin changes.

SUPPLEMENTARY INFORMATION ON PAH

7. Work activities that may represent a high risk exposure

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

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.

8. Non-work sources

PAHs are released during incomplete combustion of coal, oil and gas, garbage, or other organic substances like tobacco or charbroiled meat. They are found throughout the environment in the air, water and soil.

Exposure occurs by breathing air containing PAHs from cigarette smoke, wood smoke, vehicle exhausts and agricultural burn smoke. Exposure can also occur by eating grilled or charred meats, contaminated cereals, flour, bread, vegetables, fruits or meats and drinking contaminated water.

Psoriasis patients treated with coal-tar are also exposed to PAHs.

POTENTIAL HEALTH EFFECTS FOLLOWING EXPOSURE TO PAH

9. Route of entry into the body

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

10. 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.

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. The skin can be affected by dermal exposure or 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.

11. Acute effects

The systemic effects of naphthalene (moth-repellent) are known from numerous cases of accidental intake, particularly by children. The typical effect after dermal or oral exposure is acute haemolytic anaemia. The lethal oral dose is 5000 to 15 000 mg for adults and 2000 mg taken over two days for a child.

12. Chronic effects

Dermal toxic effects of PAH are enhanced by exposure to ultraviolet light. The skin is prone to erythema, 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 have also been noted.

13. Carcinogenicity

There is sufficient evidence PAH are carcinogenic to experimental animals. Work-related exposure to soot as a cause of scrotal cancer was noted for the first time in 1775 [1]. Later, work-related exposure to tars and paraffin was reported to induce skin cancer. There is evidence workers exposed to high airborne levels of some PAH show excess rates of lung, kidney, bladder, gastrointestinal and skin cancers.

Epidemiologic studies have been conducted of workers exposed at coke ovens during coal coking and coal gasification, at asphalt works, foundries, and aluminium smelters and workers exposed to diesel exhaust. Increased lung tumour rates due to exposure to PAH have been found in coke oven workers, asphalt workers and workers in pot rooms of aluminium reduction plants. The highest risk was found for coke oven workers, with a standardised mortality ratio of 195.

The lung is now the main site of PAH-induced cancer, whereas skin tumours have become rarer because of better personal hygiene.

Some well-known carcinogenic PAH are benzo[a]pyrene, benz[a]anthracene and dibenz[a,h]anthracene.

14. Carcinogen, germ cell mutagen and reproductive toxicant classifications

Benzo[a]pyrene, benz[a]anthracene and dibenz[a,h]anthracene are classified according to the GHS as Carcinogenicity Category 1B (May cause cancer).

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

REFERENCED DOCUMENTS

1. Brown JR, Thornton JL, 'Percivall Pott (1714 -1788) and Chimney Sweepers' Cancer of the Scrotum', *British Journal of Industrial Medicine*, vol 14(1), pp 68-70, 1957.

FURTHER READING

Agency for Toxic Substances and Disease Registry, *Toxicological Profile Polycyclic Aromatic Hydrocarbons*, Agency for Toxic Substances and Disease Registry, United States Department of Health and Human Services, Public Health Service, Atlanta, 1995.

Australian Safety and Compensation Council, *Guidance Note for the Protection of Workers from the Ultraviolet Radiation in Sunlight*, Australian Safety and Compensation Council, 2008.

International Agency for Research on Cancer, *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Volume 92: Some Non-heterocyclic Polycyclic Aromatic Hydrocarbons and Some Related Exposures*, International Agency for Research on Cancer, Lyon, 2010.

International Programme on Chemical Safety, *Environmental Health Criteria 202, Selected Non-Heterocyclic Polyclyclic Aromatic Hydrocarbons*, International Programme on Chemical Safety, World Health Organization, Geneva, 1998.

Lauwerys RR, Hoet P, *Industrial Chemical Exposure Guidelines for Biological Monitoring*, 3rd Ed, Lewis Publishers, Boca Raton, 2001.

HEALTH MONITORING REPORT POLYCYCLIC AROMATIC HYDROCARBONS (PAH)

This health monitoring report is a confidential health record and must not be disclosed to another person except in accordance with the Work Health and Safety Regulations or with the consent of the worker.

There are two sections. Complete both sections and all questions if applicable.

Section 1 is to be forwarded to the PCBU who has engaged your services. A copy of laboratory report(s) must be attached > > > >

Section 2 may contain confidential information which may not be relevant to the health monitoring program being carried out. This section should be retained by the medical practitioner. Information which is required to be given to the PCBU should be summarised in part 7 of section 1.

SECTION 1 – THIS SECTION TO BE RETURNED TO THE PCBU			
1. PERSON CONDUCTING A BUSINESS OR UNDERTAKING			
Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
2. OTHER BUSINESSES OR UNDERTAKINGS ENGAGING THE WORKER			
Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
3. WORKER		(✓) all relevant boxes	
Surname:		Given names:	
Date of birth: DD/MM/YYYY	Sex:	☐ Male	☐ Female
Address:			
Suburb:			Postcode:
Current Job:	Tel(H):		Mob:
Date started employment : DD/MM/YYYY			
4. EMPLOYMENT IN PAH RISK WORK		(✓) all relevant boxes	
1. ☐ New to PAH work			
2. ☐ New worker but not new to PAH work			
3. ☐ Current worker continuing in PAH work			
4. Worked with PAH since DD/MM/YYYY			
5. Satisfactory personal hygiene (for example nail biting, frequency of hand washing)		☐ Yes	☐ No
6. Risk assessment completed		☐ Yes	☐ No

5. WORK ENVIRONMENT ASSESSMENT			(✓) all relevant boxes
Date of assessment: DD/MM/YYYY			
PAH Industry			
☐ Coke Plant	**Controls:**		
☐ Aluminium Plant	Wear gloves	☐ Yes	☐ No
☐ Roofing Industry	Respirator use	☐ Yes	☐ No
☐ Road Surfacing	Local exhaust ventilation	☐ Yes	☐ No
☐ Automotive Industry (diesel)	Overalls / work clothing	☐ Yes	☐ No
☐ Other (specify):	Laundering by employer	☐ Yes	☐ No
	Wash basins & showers (with hot & cold water)	☐ Yes	☐ No
	Smoking or eating in workshop	☐ Yes	☐ No
	Personal hygiene:		
	Clean Shaven	☐ Yes	☐ No
	Shower & change into clean clothes at end of shift	☐ Yes	☐ No

6. BIOLOGICAL MONITORING RESULTS Include at least the previous two test results (if available)		
Date	**Tests performed**	**Recommended Action and/or Comment**
1. DD/MM/YYYY		
2. DD/MM/YYYY		
3. DD/MM/YYYY		
4. DD/MM/YYYY		
5. DD/MM/YYYY		
6. DD/MM/YYYY		
7. DD/MM/YYYY		

7. RECOMMENDATIONS (by Medical Practitioner) (✓) all relevant boxes
1. ☐ Suitable for work with PAH
2. ☐ Counselling required
3. ☐ Review workplace controls
4. ☐ Repeat health assessment in _____ month(s) / _____ week(s)

5. ☐ Removal from work with PAH	On DD/MM/YYYY
6. ☐ Medical examination by Medical Practitioner	On DD/MM/YYYY
7. ☐ Fit to resume PAH risk work	From DD/MM/YYYY
8. ☐ Referred to Medical Specialist (respiratory/dermatology/other):	On DD/MM/YYYY
Specialist's name:	
Additional comments or recommendations arising from health monitoring:	

Medical Practitioner (responsible for supervising health monitoring)		
Name:	Signature	Date: DD/MM/YYYY
Tel:	Fax:	Registration Number:
Medical Practice:		
Address:		
Suburb:		Postcode:

SECTION 2 – THIS SECTION TO BE RETAINED BY THE MEDICAL PRACTITIONER			
1. PERSON CONDUCTING A BUSINESS OR UNDERTAKING			
Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
2. OTHER BUSINESSES OR UNDERTAKINGS ENGAGING THE WORKER			
Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
3. WORKER			(✓) all relevant boxes
Surname:		Given names:	
Date of birth: DD/MM/YYYY	Sex:	☐ Male	☐ Female ☐ Pregnant/Breast Feeding?
Address:			
Suburb:			Postcode:
Current Job:		Tel(H):	Mob:
Date started employment : DD/MM/YYYY			
4. GENERAL HEALTH ASSESSMENT (if applicable)			
Symptoms of:	**Comments**	**Further testing?**	
Skin disorders		☐ Yes	☐ No
Headaches, dizziness		☐ Yes	☐ No
Respiratory tract		☐ Yes	☐ No
Eyes		☐ Yes	☐ No
Photosensitivity		☐ Yes	☐ No
Cough		☐ Yes	☐ No
CNS		☐ Yes	☐ No

Others		☐ Yes ☐ No
Height _____cm Weight _____kg Bp ____/____ mmHg		☐ Yes ☐ No

5. OTHER MEDICAL HISTORY, FAMILY MEDICAL HISTORY, CURRENT MEDICATION, COMMENTS, TESTS OR RECOMMENDATIONS (use separate sheet if necessary)

Medical Practitioner (responsible for supervising health monitoring)

Name:	Signature	Date: DD/MM/YYYY
Tel:	Fax:	Registration Number:
Medical Practice:		
Address:		
Suburb:		Postcode:

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PENTACHLOROPHENOL (PCP)

BASELINE HEALTH MONITORING BEFORE STARTING WORK IN A PCP PROCESS

1. **Collection of demographic data**
2. **Work history**
3. **Medical history**
4. **Physical examination**

A physical examination will be conducted with emphasis on the skin, noting abnormal lesions or effects of irritancy.

5. **Investigation**

The following tests will be used to test the worker's baseline exposure:

- A spot urine test for total PCP will be conducted and the result will be corrected for creatinine. Where there is 1 mg or more of total PCP per gram of creatinine, a repeat spot urine for total PCP should be performed at the same time of the day.
- A dipstick urinalysis for haematuria and proteinuria will also be conducted.

6. **Health advice**

Workers must be informed of the potential health effects associated with exposure to PCP. In particular, workers should be aware of the occurrence and recognition of skin changes and irritancy, and the need to report them as soon as possible, even if they occur between regular health monitoring.

DURING EXPOSURE TO A PCP PROCESS

7. **Monitoring exposure to PCP**

COMPARISON OF RESULTS WITH BASELINE LEVEL

A spot urine for total PCP corrected for creatinine and a dipstick urinalysis for proteinuria and haematuria will be conducted every 180 days and compared with the worker's baseline levels.

Tests should be conducted preshift towards the end of the working week.

ACTION LEVEL

Where testing shows a level of 1 mg total PCP or more per gram of creatinine:

- a repeat spot urine for total PCP should be performed at the same time of the day to confirm results
- a medical examination should be performed, with emphasis on the hepatic and renal systems and skin
- depending on medical examination findings, further tests may be needed including serum biochemistry, urea and electrolytes, and a coagulation profile
- the person conducting a business or undertaking must review control measures and carry out recommended remedial action
- the worker must be informed of the results of the health monitoring.

REMOVAL LEVEL

Removal from PCP work should be considered if there is evidence of adverse health effects due to PCP. A spot urine for total PCP corrected for creatinine should be repeated every 30 days until the level falls below 1 mg per gram of creatinine.

RETURN TO WORK

The worker must not return to PCP work until they have been assessed as medically fit to return to work by the medical practitioner supervising the health monitoring.

AT TERMINATION OF WORK IN A PCP PROCESS

8. Final medical examination

A final medical examination will be conducted with emphasis on the skin, noting abnormal lesions or effects of irritancy.

SUPPLEMENTARY INFORMATION ON PCP

9. 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.

10. Non-work sources

PCP is found in all environmental media as a result of its past widespread use and can be detected in the urine and plasma of most non-work exposed people. Pentachlorophenol has been detected in fruits, vegetables, meats, water and soils. Small amounts of pentachlorophenol of up to 40 μg/L in urine may be found in the general population.

POTENTIAL HEALTH EFFECTS FOLLOWING EXPOSURE TO PCP

11. Route of entry into the body

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

12. Target organ/effect

Skin, mucous membranes – irritation.

Eyes – visual damage and irritation.

Kidneys – impaired renal function.

Liver – hepatocellular damage.

13. Acute effects

Acute toxicity results from the uncoupling of oxidative phosphorylation causing stimulation of cell metabolism and accompanying heat dissipation. Acute poisoning can affect both renal and hepatic function with elevations of alkaline phosphatase, serum creatinine and blood urea nitrogen.

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/m^3. Airborne concentrations as low as 0.3 mg/m^3 may cause some irritation of the upper respiratory tract. Direct skin contact can lead to skin irritation and systemic toxicity. 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, tachycardia, tachypnoea, respiratory distress and hepatic enlargement. In fatal cases, the 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 are possibly more susceptible to poisoning from this substance.

14. Chronic effects

Chronic exposure is associated with inflammation of the upper respiratory tract and bronchitis, blood effects like aplastic anaemia, effects on the kidney and liver, immunological effects, irritation of the eyes, nose and skin, increased prevalence of conjunctivitis, chronic sinusitis, polyneuritis and dermatitis.

Bronchitis has been reported at airborne dust or mist concentrations of 1.0 mg/m^3. 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 two per cent PCP solution was used without adequate control measures.

In the body, PCP acts to uncouple oxidative phosphorylation, resulting in hyperthermia. Medications that cause dehydration, or possess anticholinergic properties, and diuretics, phenothiazines, antihistamines and antidepressants may also increase the susceptibility of exposed people to hyperthermia.

Aspirin, which can also uncouple oxidative phosphorylation when absorbed in large amounts, may enhance the risk of toxicity for PCP-exposed people. Because PCP is highly protein-bound, people taking medications on a long-term basis that have an affinity for plasma proteins may be at increased risk of PCP-induced toxicity. Phenytoin, warfarin, furosemide, ethacrynic acid, naproxen and ibuprofen can compete with PCP for protein binding sites, increasing the level of free PCP circulating in the blood.

15. Carcinogen classification

Pentachlorophenol is classified according to the GHS as Carcinogenicity Category 2 (Suspected of causing cancer).

FURTHER READING

Agency for Toxic Substances and Disease Registry, *Toxicological Profile for Pentachlorophenol*, Agency for Toxic Substances and Disease Registry, United States Department of Health and Human Services, Public Health Service, Atlanta, 2001.

American Conference of Governmental Industrial Hygienists (ACGIH), *Documentation of the Threshold Limit Values and Biological Exposure Indices, Pentachlorophenol*, 7th Ed, Cincinnati, 2011.

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There are two sections. Complete both sections and all questions if applicable.

Section 1 is to be forwarded to the PCBU who has engaged your services. A copy of laboratory report(s) must be attached > > > >

Section 2 may contain confidential information which may not be relevant to the health monitoring program being carried out. This section should be retained by the medical practitioner. Information which is required to be given to the PCBU should be summarised in part 7 of section 1.

SECTION 1 – THIS SECTION TO BE RETURNED TO THE PCBU			
1. PERSON CONDUCTING A BUSINESS OR UNDERTAKING			
Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
2. OTHER BUSINESSES OR UNDERTAKINGS ENGAGING THE WORKER			
Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
3. WORKER		(✓) all relevant boxes	
Surname:		Given names:	
Date of birth: DD/MM/YYYY	Sex:	☐ Male	☐ Female
Address:			
Suburb:			Postcode:
Current Job:	Tel(H):		Mob:
Date started employment : DD/MM/YYYY			
4. EMPLOYMENT IN PENTACHLOROPHENOL (PCP) RISK WORK (✓) all relevant boxes			
1. ☐ New to PCP work			
2. ☐ New worker but not new to PCP work			
3. ☐ Current worker continuing in PCP work			
4. Worked with PCP since DD/MM/YYYY			
5. Satisfactory personal hygiene (for example nail biting, frequency of hand washing)		☐ Yes	☐ No
6. Risk assessment completed		☐ Yes	☐ No

5. WORK ENVIRONMENT ASSESSMENT		(✓) all relevant boxes	
Date of assessment: DD/MM/YYYY			
Pentachlorophenol Industry			
☐ Timber Industry	**Controls:**		
☐ Other (specify):	Wear gloves	☐ Yes	☐ No
	Respirator use	☐ Yes	☐ No
	Local exhaust ventilation	☐ Yes	☐ No
	Overalls / work clothing	☐ Yes	☐ No
	Laundering by employer	☐ Yes	☐ No
	Wash basins & showers (with hot & cold water)	☐ Yes	☐ No
	Smoking or eating in workshop	☐ Yes	☐ No
	Personal hygiene:		
	Clean Shaven	☐ Yes	☐ No
	Shower & change into clean clothes at end of shift	☐ Yes	☐ No

6. BIOLOGICAL MONITORING RESULTS Include at least the previous two test results (if available)		
Date	**Tests performed**	**Recommended Action and/or Comment**
1. DD/MM/YYYY		
2. DD/MM/YYYY		
3. DD/MM/YYYY		
4. DD/MM/YYYY		
5. DD/MM/YYYY		
6. DD/MM/YYYY		
7. DD/MM/YYYY		

7. RECOMMENDATIONS (by Medical Practitioner) (✓) all relevant boxes
1. ☐ Suitable for work with PCP
2. ☐ Counselling required
3. ☐ Review workplace controls
4. ☐ Repeat health assessment in _____ month(s) / _____ week(s)

5. ☐ Removal from work with PCP	On DD/MM/YYYY
6. ☐ Medical examination by Medical Practitioner	On DD/MM/YYYY
7. ☐ Fit to resume PCP risk work	From DD/MM/YYYY
8. ☐ Referred to Medical Specialist (respiratory/dermatology/other): On DD/MM/YYYY Specialist's name:	
Additional comments or recommendations arising from health monitoring:	

Medical Practitioner (responsible for supervising health monitoring)		
Name:	Signature	Date: DD/MM/YYYY
Tel:	Fax:	Registration Number:
Medical Practice:		
Address:		
Suburb:		Postcode:

SECTION 2 – THIS SECTION TO BE RETAINED BY THE MEDICAL PRACTITIONER			
1. PERSON CONDUCTING A BUSINESS OR UNDERTAKING			
Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
2. OTHER BUSINESSES OR UNDERTAKINGS ENGAGING THE WORKER			
Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
3. WORKER			(✓) all relevant boxes
Surname:		Given names:	
Date of birth: DD/MM/YYYY	Sex:	☐Male	☐ Female ☐ Pregnant/Breast Feeding?
Address:			
Suburb:			Postcode:
Current Job:		Tel(H):	Mob:
Date started employment : DD/MM/YYYY			
4. GENERAL HEALTH ASSESSMENT (if applicable)			
Symptoms of:	**Comments**	**Further testing?**	
Skin disorders		☐ Yes	☐ No
Headaches, dizziness		☐ Yes	☐ No
Respiratory tract		☐ Yes	☐ No
Eyes		☐ Yes	☐ No
Mucous membranes		☐ Yes	☐ No
CNS		☐ Yes	☐ No
Others		☐ Yes	☐ No
Height _____cm Weight _____kg Bp ____/____ mmHg		☐ Yes	☐ No

5. OTHER MEDICAL HISTORY, FAMILY MEDICAL HISTORY, CURRENT MEDICATION, COMMENTS, TESTS OR RECOMMENDATIONS (use separate sheet if necessary)		
Medical Practitioner (responsible for supervising health monitoring)		
Name:	Signature	Date: DD/MM/YYYY
Tel:	Fax:	Registration Number:
Medical Practice:		
Address:		
Suburb:		Postcode:

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THALLIUM

BASELINE HEALTH MONITORING BEFORE STARTING WORK IN A THALLIUM PROCESS

1. **Collection of demographic data**
2. **Work history**
3. **Medical history**
4. **Physical examination**

A physical examination will be conducted only if indicated by work and medical history.

5. **Investigation**

A spot urine test for thallium will be used to test the worker's baseline exposure. The result is corrected for creatinine, that is the thallium concentration in micrograms per gram of creatinine. Where there is 50 µg thallium or more per gram creatinine, a repeat spot urine test should be performed at the same time of the day.

DURING EXPOSURE TO A THALLIUM PROCESS

6. **Monitoring exposure to thallium**

COMPARISON OF RESULTS WITH BASELINE LEVEL

A spot urine test for thallium will be conducted every 90 days and compared with the worker's baseline levels.

Where there is 50 µg thallium or more per gram of creatinine:

- a repeat spot urine for thallium should be performed at the same time of the day to confirm results
- a physical examination should be performed with particular attention to the nervous system and noting hair loss
- the person conducting a business or undertaking must review control measures and carry out recommended remedial action
- the worker must be informed of the results of the health monitoring.

Note: A spot urine for thallium should also be conducted if there is an acute exposure, for example following a reagent spill.

OTHER INFORMATION

Following absorption, thallium rapidly appears in the urine, which is the main excretory route. Excretion, however, is slow and levels may remain elevated for several weeks following exposure—half-life is between 15 to 30 days. The concentration of thallium in urine is generally below 0.83 µmol/mol creatinine (7.3 nmol/L or 1.5 µg/L). Work exposure should be below 28 µmol/mol creatinine (245 nmol/L or 50µg/L).

REMOVAL LEVEL

Where results of urine testing indicate a level of thallium in urine above 100 µg per gram of creatinine, the following action should be carried out until the level falls below 50 µg thallium per gram of creatinine:

- a repeat spot urine for thallium should be performed at the same time of the day to confirm results
- a medical examination should be performed and the registered medical practitioner should consider whether the worker should be removed from thallium work
- urine tests should be repeated every 30 days and then at regular intervals until the level of thallium in urine falls below 50 µg thallium per gram of creatinine
- the person conducting a business or undertaking should review control measures and carry out recommended remedial action
- the worker must be informed of the results of health monitoring.

RETURN TO WORK

The worker must not return to thallium work until they have been assessed as medically fit to return to work by the medical practitioner supervising the health monitoring.

AT TERMINATION OF WORK IN A THALLIUM PROCESS

7. Final Medical Examination

A final medical examination will be conducted and will include a spot urine for thallium.

SUPPLEMENTARY INFORMATION ON THALLIUM

8. 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, which may occur in the above processes.

9. Non-work sources

Thallium is present in the environment as a result of natural processes and from man-made sources. It is ubiquitous in nature and occurs especially in sulphide ores of various heavy metals. Losses to the environment mainly occur from mineral smelters, coal-burning power-generating plants, brickworks and cement plants as thallium is a trace element of the raw materials. Thallium volatilises during the burning of coal or raw material for cement production and recondenses on the surface of fly-ash. Thallium enters food because it is easily taken up by plants through the roots. Cigarette smoking is also a source of thallium. People who smoke have twice as much thallium in their bodies as non-smokers.

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).

10. Route of Entry into the Body

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

11. Target organ/effect

CNS – unco-ordination, tremors, encephalopathy, convulsions, coma, paralysis, optic nerve atrophy.

Peripheral nervous system – motor and sensory peripheral neuropathy.

Skin – alopecia.

Gastrointestinal tract – anorexia, gastroenteritis.

12. Acute Effects

Thallium and thallium compounds are extremely toxic. For adults, doses which have proved lethal vary between 6 and 40 mg/kg. Thallium behaves as a potassium analogue and is distributed in the intracellular space of most tissues. Intracellular thallium is less rapidly released than potassium.

Poisoning from industrial exposure has rarely been reported, and those cases that have been reported were not fatal.

The triad of gastroenteritis, polyneuropathy and alopecia is regarded as the classic syndrome of thallium poisoning. 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 10 days by the development of polyneuritis, psychosis, delirium, optic nerve atrophy and blindness, increased heart rate and blood pressure, skin eruptions and hepatic or renal injury. Hair loss occurs within 15 to 20 days.

13. 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 symptomatology (polyneuropathy and partial hair loss). At a higher exposure level, fatigue, anorexia, leg joint pain, optic nerve atrophy with visual disturbances, and ascending neuropathy may occur.

14. 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.

FURTHER READING

Agency for Toxic Substances and Disease Registry, *ToxFAQs for Thallium*, Agency for Toxic Substances and Disease Registry, United States Department of Health and Human Services, Public Health Service, Atlanta, 1995. www.atsdr.cdc.gov/.

Agency for Toxic Substances and Disease Registry, *Toxicological Profile for Thalllium*, Agency for Toxic Substances and Disease Registry, United States Department of Health and Human Services, Public Health Service, Atlanta, 1992.

International Programme on Chemical Safety, *Environmental Health Criteria 182: Thallium*, International Programme on Chemical Safety, World Health Organization, Geneva, 1996

Lauwerys RR, Hoet P, *Industrial Chemical Exposure Guidelines for Biological Monitoring*, 3rd Ed, Lewis Publishers, Boca Raton, 2001.

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There are two sections. Complete both sections and all questions if applicable.

Section 1 is to be forwarded to the PCBU who has engaged your services. A copy of laboratory report(s) must be attached > > > >

Section 2 may contain confidential information which may not be relevant to the health monitoring program being carried out. This section should be retained by the medical practitioner. Information which is required to be given to the PCBU should be summarised in part 7 of section 1.

SECTION 1 – THIS SECTION TO BE RETURNED TO THE PCBU			
1. PERSON CONDUCTING A BUSINESS OR UNDERTAKING			
Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
2. OTHER BUSINESSES OR UNDERTAKINGS ENGAGING THE WORKER			
Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
3. WORKER			(✓) all relevant boxes
Surname:		Given names:	
Date of birth: DD/MM/YYYY	Sex:	☐ Female	☐ Male
Address:			
Suburb:			Postcode:
Current Job:	Tel(H):		Mob:
Date started employment : DD/MM/YYYY			
4. EMPLOYMENT IN THALLIUM RISK WORK			(✓) all relevant boxes
1. ☐ New to thallium work			
2. ☐ New worker but not new to thallium work			
3. ☐ Current worker continuing in thallium work			
4. Worked with thallium since DD/MM/YYYY			

5. Satisfactory personal hygiene (for example nail biting, frequency of hand washing)		☐ Yes	☐ No
6. Risk assessment completed		☐ Yes	☐ No
5. WORK ENVIRONMENT ASSESSMENT		(✓) all relevant boxes	
Date of assessment: DD/MM/YYYY			
Thallium Industry			
☐ Laboratory Work	**Controls:**		
☐ Paint/Pigment Production	Wear gloves	☐ Yes	☐ No
☐ Glass Production	Respirator use	☐ Yes	☐ No
☐ Electronic Production	Local exhaust ventilation	☐ Yes	☐ No
☐ Power Industry	Overalls / work clothing	☐ Yes	☐ No
☐ Other (specify):	Laundering by employer	☐ Yes	☐ No
	Wash basins & showers (with hot & cold water)	☐ Yes	☐ No
	Smoking or eating in workshop	☐ Yes	☐ No
	Personal hygiene:		
	Clean Shaven	☐ Yes	☐ No
	Shower & change into clean clothes at end of shift	☐ Yes	☐ No

6. BIOLOGICAL MONITORING RESULTS Include at least the previous two test results (if available)

Date	Tests performed	Recommended Action and/or Comment
1. DD/MM/YYYY		
2. DD/MM/YYYY		
3. DD/MM/YYYY		
4. DD/MM/YYYY		
5. DD/MM/YYYY		
6. DD/MM/YYYY		
7. DD/MM/YYYY		

7. RECOMMENDATIONS (by Medical Practitioner)	(✓) all relevant boxes
1. ☐ Suitable for work with thallium	
2. ☐ Counselling required	
3. ☐ Review workplace controls	

4. ☐ Repeat health assessment in _____ month(s) / _____ week(s)		
5. ☐ Removal from work with thallium		On DD/MM/YYYY
6. ☐ Medical examination by Medical Practitioner		On DD/MM/YYYY
7. ☐ Fit to resume thallium risk work		From DD/MM/YYYY
8. ☐ Referred to Medical Specialist (respiratory/dermatology/other): On DD/MM/YYYY Specialist's name:		
Additional comments or recommendations arising from health monitoring:		
Medical Practitioner (responsible for supervising health monitoring)		
Name:	Signature	Date: DD/MM/YYYY
Tel:	Fax:	Registration Number:
Medical Practice:		
Address:		
Suburb:		Postcode:

SECTION 2 – THIS SECTION TO BE RETAINED BY THE MEDICAL PRACTITIONER			
1. PERSON CONDUCTING A BUSINESS OR UNDERTAKING			
Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
2. OTHER BUSINESSES OR UNDERTAKINGS ENGAGING THE WORKER			
Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
3. WORKER		(✓) all relevant boxes	
Surname:		Given names:	
Date of birth: DD/MM/YYYY	Sex: ☐Male	☐ Female ☐ Pregnant/Breast Feeding?	
Address:			
Suburb:			Postcode:
Current Job:		Tel(H):	Mob:
Date started employment : DD/MM/YYYY			
4. GENERAL HEALTH ASSESSMENT (if applicable)			
Symptoms of:	**Comments**	**Further testing?**	
Skin disorders		☐ Yes	☐ No
Headaches, dizziness		☐ Yes	☐ No
Respiratory tract		☐ Yes	☐ No
GIT		☐ Yes	☐ No
Eyes		☐ Yes	☐ No
Mucous membranes		☐ Yes	☐ No
CNS		☐ Yes	☐ No

Peripheral nervous system		☐ Yes ☐ No
Others		☐ Yes ☐ No
Height _____cm Weight _____kg Bp ____/____ mmHg		☐ Yes ☐ No

5. OTHER MEDICAL HISTORY, FAMILY MEDICAL HISTORY, CURRENT MEDICATION, COMMENTS, TESTS OR RECOMMENDATIONS (use separate sheet if necessary)

Medical Practitioner (responsible for supervising health monitoring)

Name:	Signature	Date: DD/MM/YYYY
Tel:	Fax:	Registration Number:
Medical Practice:		
Address:		
Suburb:		Postcode:

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VINYL CHLORIDE

BASELINE HEALTH MONITORING BEFORE STARTING WORK IN A VINYL CHLORIDE PROCESS

1. **Collection of demographic data**
2. **Work history**
3. **Medical history**

There are many non-work factors associated with hepatocellular carcinoma, including excessive alcohol consumption and viral hepatitis that the medical practitioner needs to be aware of. The following details about the worker's medical history will be collected by the medical practitioner:

- presence of symptoms
- smoking history
- alcohol consumption
- viral hepatitis – hepatitis B or C
- haemachromatosis
- other liver disease

4. **Physical examination**

A physical examination will be conducted only if work and medical history indicates this is necessary, for example if the symptoms of vinyl chloride exposure are present.

5. **Investigation**

In addition to medical history and physical examination, there are a number of test methods that can be used to assess exposure to vinyl chloride. These are:

- full blood count including mean cell volume and platelets
- liver function tests including aspartate transaminase (AST), alanine transaminase (ALT), gamma glutamyl transpeptidase (GGT), alkaline phosphatase and bilirubin.

The registered medical practitioner may choose to conduct these tests to assess the worker's exposure to vinyl chloride.

The medical practitioner should consider testing for viral markers for hepatitis B and hepatitis C after pre-test counselling.

Note: There is no specific biological marker for angiosarcoma of the liver (ASL) and hepatocellular carcinoma (HCC) due to vinyl chloride monomer exposure.

BACKGROUND INFORMATION ON VINYL CHLORIDE EXPOSURE

With the level of potential exposure in vinyl chloride monomer/polyvinyl chloride (VCM/PVC) plants at such a low level these days, it is unlikely workers will develop ASL—only one case has been diagnosed in Australia in 1978—or HCC due to VCM. However, workers should be made aware of the additional risk of developing HCC when they have hepatitis B virus (HBV) or hepatitis C virus (HCV) or when their alcohol consumption is too high.

If workers are suffering from active hepatitis, the registered medical practitioner should discuss with the treating gastroenterologist possible exemption from working in duties potentially exposing them to VCM.

DURING EXPOSURE TO A VINYL CHLORIDE PROCESS

6. Monitoring exposure to vinyl chloride

Medical examinations should occur every two years, with laboratory tests repeated annually where required.

7. Medical examination

The person conducting a business or undertaking should arrange an appointment with the registered medical practitioner for workers who are excessively exposed to vinyl chloride, are suspected of being excessively exposed to vinyl chloride, or have concerns about vinyl chloride exposure.

AT TERMINATION OF WORK IN A VINYL CHLORIDE PROCESS

8. Final medical examination

A final medical examination will be conducted and may include tests used by the registered medical practitioner to assess exposure including:

- full blood count including mean cell volume and platelets
- liver function tests including AST, ALT, GGT, alkaline phosphatase and bilirubin.

SUPPLEMENTARY INFORMATION ON VINYL CHLORIDE

9. Work activities that may represent a high risk 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.

Special attention should also be given to acute exposures that may occur in the above vinyl chloride processes.

10. Non-work sources

Vinyl chloride is not known naturally although it has been found in landfill, gas and groundwater as a degradation product of chlorinated hydrocarbons deposited as solvent wastes in landfills. The level of residual vinyl chloride in PVC has been regulated since the late 1970's in many countries. Since then, release of vinyl chloride monomer from the thermal degradation of PVC is either not detectable or is at very low levels. Vinyl chloride is also present in cigarette smoke.

POTENTIAL HEALTH EFFECTS FOLLOWING EXPOSURE TO VINYL CHLORIDE

11. Route of entry into the body

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

12. Target organ/effect

Liver – fibrosis, angiosarcoma.

CNS – dizziness, ataxia, visual disturbance, coma, death.

Irritant – eyes, mucous membranes, respiratory tract.

Skin – scleroderma.

Circulatory – Raynaud's syndrome.

Bone – resorption particularly of the fingertips—acro-osteolysis.

13. Acute effects

CNS: The central nervous system (CNS) is the primary target of vinyl chloride acute toxicity. Vinyl chloride is thought to depress the CNS via a solvent effect on lipids and protein components of neural membranes that interrupts signal transmission. **There may be a latent period of hours to days between exposure and symptom onset.** Symptoms include euphoria, dizziness, ataxia, 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.

Respiratory: 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.

Cardiovascular: Vinyl chloride may lower the myocardial threshold to the dysrhythmogenic effects of catecholamines.

Dermal, Ocular: Exposure to escaping compressed gas or liquid can cause frostbite injury with redness, blistering, and scaling of the skin and corneal and conjunctival irritation or burns.

14. Chronic effects

Chronic exposure to lower levels of around 100-1000 ppm has been associated with a spectrum of symptoms collectively termed 'vinyl chloride disease' which includes Raynaud's syndrome, scleroderma and acro-osteolysis—bone resorption of the terminal phalanges of the fingers.

Liver and spleen fibrosis, portal hypertension and cirrhosis can occur. Other effects of chronic exposure include sensory-motor polyneuropathy; pyramidal, extrapyramidal, and cerebellar abnormalities and immunopathologic phenomena like purpura and thrombocytopaenia.

15. Carcinogenicity

Vinyl chloride is genotoxic.

A large number of epidemiological studies [1] and case reports have substantiated the causal association between vinyl chloride and haemangiosarcoma of the liver. It was recognised that the cause of haemangiosarcoma was likely to be inhalation of vinyl chloride at concentrations of probably a few hundred parts per million over long periods.

In June 2007, IARC concluded exposure to vinyl chloride also causes hepatocellular carcinoma (HCC) [2]. From Maltoni's work with rats, the VCM exposure required to induce a HCC is ten times that required to produce an angiosarcoma of the liver (ASL).

The World register of ASL contains 231 cases. Sixteen cases of HCC are published that are probably related to VCM exposure. In all cases, the first exposure was before 1974.

There are many non-work factors associated with HCC including excessive alcohol consumption and viral hepatitis. Mastrangelo's work in 2004 [3] suggested exposure to vinyl chloride and excessive alcohol consumption had a synergistic effect on the development of HCC.

16. Carcinogen classification

Vinyl chloride is classified according to the GHS as Carcinogenicity Category 1A (May cause cancer).

REFERENCED DOCUMENTS

1. International Agency for Research on Cancer, *Overall Evaluations of Carcinogenicity: An Updating of IARC Monographs Volumes 1-42*, Supplement No. 7, International Agency for Research on Cancer, Lyon, 1987.
2. International Agency for Research on Cancer, *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Volume 97: Vinyl Chloride, pp 311-443*, International Agency for Research on Cancer, Lyon, 2008.
3. Mastrangelo G, Fedeli U, Fadda E, Valentini F, Agnesi R, Magarotto G, Marchi T, Buda A, Pinzani M, Martines D, 'Increased Risk of Hepatocellular Carcinoma and Liver Cirrhosis in Vinyl Chloride Workers: Synergistic Effect of Occupational Exposure with Alcohol Intake', *Environmental Health Perspectives* (2004) vol 112(11), pp 1188-92, 2004.

FURTHER READING

Agency for Toxic Substances and Disease Registry, *Case Studies in Environmental Medicine 2: Vinyl Chloride Toxicity,* Agency for Toxic Substances and Disease Registry, United States Department of Health and Human Services, Public Health Service, Atlanta, 1990.

Agency for Toxic Substances and Disease Registry, *Medical Management Guidelines for Vinyl Chloride*, Agency for Toxic Substances and Disease Registry, United States Department of Health and Human Services, Public Health Service, Atlanta, 2008. http://www.atsdr.cdc.gov/MMG/index.asp

Agency for Toxic Substances and Disease Registry, *Toxicological Profile for Vinyl Chloride*, Agency for Toxic Substances and Disease Registry, United States Department of Health and Human Services, Public Health Service, Atlanta, 2006. http://www.atsdr.cdc.gov/toxprofiles/index.asp

European Centre for Ecotoxicology and Toxicology of Chemicals, *Technical Report 31: The Mutagenicity and Carcinogenicity of Vinyl Chloride—A Historical Review and Assessment*, European Centre for Ecotoxicology and Toxicology of Chemicals, Brussels, 1988.

International Program for Chemical Safety, *Environmental Health Criteria 215 Vinyl Chloride*, WHO Geneva, 1999. www.inchem.org

Lauwerys RR, Hoet P, *Industrial Chemical Exposure Guidelines for Biological Monitoring*, 3rd Ed, Lewis Publishers, Boca Raton, 2001.

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Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
2. OTHER BUSINESSES OR UNDERTAKINGS ENGAGING THE WORKER			
Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
3. WORKER		(✓) all relevant boxes	
Surname:		Given names:	
Date of birth: DD/MM/YYYY	Sex:	☐ Male	☐ Female
Address:			
Suburb:			Postcode:
Current Job:	Tel(H):	Mob:	
Date started employment : DD/MM/YYYY			
4. EMPLOYMENT IN VINYL CHLORIDE RISK WORK		(✓) all relevant boxes	
1. ☐ New to vinyl chloride work			
2. ☐ New worker but not new to vinyl chloride work			
3. ☐ Current worker continuing in vinyl chloride work			
4. Worked with vinyl chloride since DD/MM/YYYY			
5. Satisfactory personal hygiene (for example nail biting, frequency of hand washing)		☐ Yes	☐ No
6. Risk assessment completed		☐ Yes	☐ No

5. WORK ENVIRONMENT ASSESSMENT	(✓) all relevant boxes		
Date of assessment: DD/MM/YYYY			
Vinyl Chloride Industry			
☐ Polyvinyl Chloride Production	**Controls:**		
☐ Other (specify):	Wear gloves	☐ Yes	☐ No
	Respirator use	☐ Yes	☐ No
	Local exhaust ventilation	☐ Yes	☐ No
	Overalls / work clothing	☐ Yes	☐ No
	Laundering by employer	☐ Yes	☐ No
	Wash basins & showers (with hot & cold water)	☐ Yes	☐ No
	Smoking or eating in workshop	☐ Yes	☐ No
	Personal hygiene:		
	Clean Shaven	☐ Yes	☐ No
	Shower & change into clean clothes at end of shift	☐ Yes	☐ No

6. BIOLOGICAL MONITORING RESULTS Include at least the previous two test results (if available)		
Date	**Tests performed**	**Recommended Action and/or Comment**
1. DD/MM/YYYY		
2. DD/MM/YYYY		
3. DD/MM/YYYY		
4. DD/MM/YYYY		
5. DD/MM/YYYY		
6. DD/MM/YYYY		
7. DD/MM/YYYY		

7. RECOMMENDATIONS (by Medical Practitioner) (✓) all relevant boxes		
1. ☐ Suitable for work with vinyl chloride		
2. ☐ Counselling required		
3. ☐ Review workplace controls		
4. ☐ Repeat health assessment in _____ month(s) / _____ week(s)		
5. ☐ Removal from work with vinyl chloride	On DD/MM/YYYY	
6. ☐ Medical examination by Medical Practitioner	On DD/MM/YYYY	
7. ☐ Fit to resume vinyl chloride risk work	From DD/MM/YYYY	
8. ☐ Referred to Medical Specialist (respiratory/dermatology/other): On DD/MM/YYYY Specialist's name:		
Additional comments or recommendations arising from health monitoring:		

Medical Practitioner (responsible for supervising health monitoring)			
Name:	Signature		Date: DD/MM/YYYY
Tel:	Fax:	Registration Number:	
Medical Practice:			
Address:			
Suburb:			Postcode:

SECTION 2 – THIS SECTION TO BE RETAINED BY THE MEDICAL PRACTITIONER			
1. PERSON CONDUCTING A BUSINESS OR UNDERTAKING			
Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
2. OTHER BUSINESSES OR UNDERTAKINGS ENGAGING THE WORKER			
Company / Organisation name:			
Site address:			
Suburb:			Postcode:
Site Tel:	Site Fax:	Contact Name:	
3. WORKER			(✓) all relevant boxes
Surname:		Given names:	
Date of birth: DD/MM/YYYY	Sex:	☐Male	☐ Female ☐ Pregnant/Breast Feeding?
Address:			
Suburb:			Postcode:
Current Job:		Tel(H):	Mob:
Date started employment : DD/MM/YYYY			
4. GENERAL HEALTH ASSESSMENT (if applicable)			
Symptoms of:	**Comments**	**Further testing?**	
Skin disorders		☐ Yes	☐ No
Headaches, dizziness		☐ Yes	☐ No
Respiratory tract		☐ Yes	☐ No
Eyes		☐ Yes	☐ No
Mucous Membranes		☐ Yes	☐ No
CNS		☐ Yes	☐ No
Others		☐ Yes	☐ No
Height _____cm Weight _____kg Bp ____/____ mmHg		☐ Yes	☐ No

5. OTHER MEDICAL HISTORY, FAMILY MEDICAL HISTORY, CURRENT MEDICATION, COMMENTS, TESTS OR RECOMMENDATIONS (use separate sheet if necessary)

Medical Practitioner (responsible for supervising health monitoring)			
Name:	Signature		Date: DD/MM/YYYY
Tel:	Fax:	Registration Number:	
Medical Practice:			
Address:			
Suburb:		Postcode:	

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THIS GUIDE IS INTENDED FOR USE BY MEDICAL PRACTITIONERS CARRYING OUT OR SUPERVISING A HEALTH MONITORING PROGRAM FOR WORKERS WHO MAY BE EXPOSED TO HAZARDOUS CHEMICALS AND ASBESTOS.