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[1]

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).

1 This classification information is provided on an advisory basis and 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. Other hazard classes and categories may apply – see http://esis.jrc.ec.europa.eu/index.php?PGM=cla. These classifications are legally binding within the European Union.

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.