GUIDELINES FOR HEALTH SURVEILLANCE

[NOHSC:7039(1995)]

DECEMBER 1995

FOREWORD

The National Occupational Health and Safety Commission is a tripartite body established by the Commonwealth Government to develop, facilitate and implement a national occupational health and safety strategy.

This strategy includes standards development, the development of hazards-specific and industry-based preventive strategies, research, training, information collection and dissemination and the development of common approaches to occupational health and safety legislation.

The National Commission comprises representatives of peak employee and employer bodies—the Australian Council of Trade Unions and the Australian Chamber of Commerce and Industry—as well as the Commonwealth, State and Territory governments.

Consistent with the National Commission's philosophy of consultation, tripartite standing committees have been established to deal with issues relating to standards development, research and the mining industry. Expert groups and reference groups may be established to provide advice to the standing committees on those issues with which the National Commission is concerned.

PREFACE

The following is a list of the *Guidelines for Health Surveillance* [NOHSC:7039(1995)] which are published as part of a series by the National Commission:

- acrylonitrile;
- inorganic arsenic;
- asbestos;
- benzene;
- cadmium;
- inorganic chromium;
- inorganic lead;
- creosote;
- isocyanates;
- inorganic mercury;
- 4,4'-methylene bis (2-chloroaniline) [MOCA];
- organophosphate pesticides;
- pentachlorophenol;
- polycyclic aromatic hydrocarbons (PAH);
- crystalline silica;
- thallium; and
- vinyl chloride.

These publications are likely to be updated or the list amended from time to time as more information becomes available or other workplace substances are recognised as needing health surveillance.

INTRODUCTION TO THE GUIDELINES FOR HEALTH SURVEILLANCE

These guidelines are intended for use by the appointed medical practitioner when planning and implementing a program of health surveillance.

The purpose of health surveillance is to ensure that control measures are effective and to provide an opportunity to reinforce specific preventive measures and safe work practices.

The National Commission's *National Model Regulations for the Control of Workplace Hazardous Substances* [NOHSC:1005(1994)] provide for health surveillance. Health surveillance is part of an integrated range of measures directed at controlling hazardous substances, to ensure the health and safety of people at work.

The health surveillance guidelines set out in a practical manner the minimum requirements for health surveillance. Each guideline should be read in conjunction with this Introduction.

The appointed medical practitioner responsible for health surveillance should be familiar with the health surveillance sections in the National Commission's *National Model Regulations for the Control of Workplace Hazardous Substances* [NOHSC:1005(1994)] and *National Code of Practice for the Control of Workplace Hazardous Substances* [NOHSC:2007(1994)]. ***See*** Appendix 1.

SUBSTANCES LISTED IN SCHEDULE 3 OF THE NATIONAL MODEL REGULATIONS FOR THE CONTROL OF WORKPLACE HAZARDOUS SUBSTANCES

The National Commission recognised that it would be useful to determine a list of substances for which health surveillance is required, where there is a significant health risk to workers from exposure to that substance. These substances are set out in Schedule 3 of the *National Model Regulations for the Control of Workplace Hazardous Substances* [NOHSC:1005(1994)], together with the minimum requirements for health surveillance. ***See*** Appendix 2.

New substances will be added to Schedule 3 from time to time. The requirements for health surveillance may be updated to reflect advancing knowledge.

SIGNIFICANT RISK TO HEALTH

A workplace assessment, required by the *National Model Regulations for the Control of Workplace Hazardous Substances* [NOHSC:1005(1994)], should determine if the workplace exposure represents a significant risk to health for Schedule 3 substances. The employer is responsible for conducting these workplace assessments which include assessments where there has been a change to work practices or exposure.

The employer should permit the appointed medical practitioner to have access to the workplace assessment conducted for the *National Model Regulations for the Control of Workplace Hazardous Substances* [NOHSC:1005(1994)]. This should provide useful information on all workplace exposure factors.

Guidance on workplace assessment for hazardous substances is set out in the National Commission's *Guidance Note for the Assessment of Health Risks Arising from the Use of Hazardous Substances in the Workplace* [NOHSC:3017(1994)].

The appointed medical practitioner should consult with the employer and jurisdictions from time to time to ensure appropriate health surveillance.

CONSIDER IF HEALTH SURVEILLANCE IS NEEDED FOR OTHER HAZARDOUS SUBSTANCES

As it is not possible for the National Commission to review all workplace hazardous substances, an employer must consider if any of the other substances present in the workplace present a significant risk to health and, if so, would there be benefits in establishing a health surveillance program.

This would be appropriate where specific health effects are known to occur and there are valid biological monitoring techniques or health screening methods available. Appointed medical practitioners may assist the employer in deciding if health surveillance is needed and setting up appropriate health surveillance programs.

The criteria used by the Expert Working Group on Health Surveillance, at Appendix 3, may assist those deciding if health surveillance is needed for a hazardous substance.

FOLLOW UP

These guidelines set out how to meet the minimum requirements for health surveillance. They are not intended to take the place of normal medical investigations. Where an abnormality is observed, the appointed medical practitioner must take the course of management most appropriate in the circumstances.

WHERE THERE ARE INDICATORS OF EXCESS EXPOSURE

Where results of the health surveillance indicate that workplace control measures may have failed, this must be communicated to the employee and to the employer as soon as practicable so that investigations and, where appropriate, remedial action can be taken.

Employees who have been removed from work with hazardous substances should continue to be provided with information concerning the results of workplace assessment and their health status. Employers should review their training programs.

CONFIDENTIALITY

Those providing health surveillance must maintain the confidentiality of the medical records of all employees past and present.

CONSENT

Consent for health surveillance should be sought from an employee before health surveillance commences. Consent should be separate from consent given for any other medical treatment.

Employees should participate in the health surveillance program unless there is some compelling reason to the contrary. In this case, the matter should be discussed with the appointed medical practitioner responsible for health surveillance.

The written consent of the employee has to be obtained before the results can be released to a third party not covered by professional confidentiality.

Similarly, any blood or tissue samples, X-rays, questionnaires or other materials taken for health surveillance should not be used for any purpose other than health surveillance (health surveillance includes longer-term epidemiological studies) without the express consent of the employee.

QUALITY ASSURANCE

Those providing health surveillance need to ensure the quality of the program through acceptable quality assurance practices, such as those set out in Standards Australia's AS 3900 *Quality Management and Quality Assurance Standards*.

Where there are any specific tests required, the analytical laboratory providing the test service should be accredited for the procedure with the National Accreditation Testing Authority.

SAMPLE COLLECTION

In those cases where the collection of biological samples is required, the instructions of the analytical laboratory providing the test service with regards to the collection, storage and transport of the samples must be strictly adhered to.

FURTHER INFORMATION

Each substance-specific health surveillance guideline contains information on the health effects of the substance. Material Safety Data Sheets for particular substances should be consulted.

APPENDIX 1

EXTRACT FROM THE NATIONAL MODEL REGULATIONS FOR THE CONTROL OF WORKPLACE HAZARDOUS SUBSTANCES [NOHSC:1005(1994)]

HEALTH SURVEILLANCE

14(1) The employer shall provide health surveillance for an employee who has been identified in the assessment process as being exposed to a hazardous substance where:

(a) there is a significant risk to the health of the employee from one of the hazardous substances listed at Schedule 3;

(b) the exposure of the employee to a hazardous substance is such that:

(i) an identifiable disease or health effect may be related to the exposure,

(ii) there is reasonable likelihood that the disease or health effect may occur under the particular conditions of work, and

(iii) there are valid techniques for detecting indications of the disease or health effect;

OR

(c) where there is a valid biological monitoring procedure available and a reasonable likelihood that accepted values might be exceeded.

(2) Health surveillance shall be performed under the supervision of a registered medical practitioner adequately trained in the requisite testing or medical examinations for the hazardous substances in question and, where appropriate, as specified by the relevant public authority.

(3) The selection of a registered medical practitioner to supervise health surveillance is to be undertaken by the employer following consultation with the relevant employees.

(4) Health surveillance shall be at the expense of the employer.

(5) Where the employee is undergoing health surveillance in accordance with sub-regulation 14(1), the registered medical practitioner shall ensure, as soon as practicable, that:

(a) the employee is notified of the results of health surveillance, together with any necessary explanation of these results;

(b) the employer is notified of the outcome of health surveillance and is advised on the need for remedial action; and

(c) the relevant public authority is notified of any prescribed adverse health effect that has been detected which is consistent with exposure to the hazardous substance in question.

(6) Where the employer has been advised by the registered medical practitioner under sub-regulation 14(5)(b) on the need for remedial action, the employer shall, as soon as practicable, revise the assessment(s) of the employee's exposure to hazardous substances and implement the control measures required under sub-regulations 12(3) and (4).

(7) The registered medical practitioner shall ensure that medical records obtained as a result of health surveillance are retained as confidential records for the purposes of these national model regulations.

(8) When a registered medical practitioner ceases his/her practice, all medical records in his/her possession obtained as a result of health surveillance shall be offered to the relevant public authority.

(9) Where a registered medical practitioner examines or treats a patient for other purposes, these records and the records obtained as a result of health surveillance shall be kept clearly identifiable.

(10) The informed written consent of the employee shall be obtained before the medical records obtained as a result of health surveillance, which identify that person, are provided to a third party not covered by professional confidentiality.

(11) The employer shall ensure that health surveillance results obtained are retained as confidential records for the purposes of these national model regulations.

Note: The following codes of practice also relate to the control of workplace hazardous substances:

(a) *National Code of Practice for the Control of Workplace Hazardous Substances* [NOHSC:2007(1994)][1];

(b) *National Code of Practice for the Preparation of Material Safety Data Sheets* [NOHSC:2011(1994)][2]; and

(c) *National Code of Practice for the Labelling of Workplace Substances* [NOHSC:2012(1994)][3].

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1. National Occupational Health and Safety Commission, *National Code of Practice for the Control of Workplace Hazardous Substances* [NOHSC:2007(1994)], Australian Government Publishing Service, Canberra, 1994.

2. National Occupational Health and Safety Commission, *National Code of Practice for the Preparation of Material Safety Data Sheets* [NOHSC:2011(1994)], Australian Government Publishing Service, Canberra, 1994.

3. National Occupational Health and Safety Commission, *National Code of Practice for the Labelling of Workplace Substances* [NOHSC:2012(1994)], Australian Government Publishing Service, Canberra, 1994.

EXTRACT FROM THE NATIONAL CODE OF PRACTICE FOR THE CONTROL OF WORKPLACE HAZARDOUS SUBSTANCES [NOHSC:2007(1994)]

14. HEALTH SURVEILLANCE

PURPOSE OF HEALTH SURVEILLANCE

14.1 Health surveillance, which includes biological monitoring, can assist in minimising the risk to health from hazardous substances for which there are known and acceptable health surveillance procedures by:

(a) confirming that the absorbed dose is below the accepted level;

(b) indicating biological effects requiring cessation or reduction of exposure; or

(c) collecting data to evaluate the effects of exposure.

14.2 Health surveillance should not be used as an alternative to maintenance of control measures. Further information on health surveillance is at Appendix 3.

THOSE EMPLOYEES REQUIRING HEALTH SURVEILLANCE

14.3 Health surveillance is required for employees who have been identified in the workplace assessment as having:

(a) a significant risk to health from one of the hazardous substances listed at Schedule 3 to the national model regulations;

(b) exposure to a hazardous substance for which:

(i) an identifiable disease or health effect may be related to the exposure,

(ii) there is reasonable likelihood that the disease or health effect may occur under the particular conditions of work, and

(iii) there are valid techniques for detecting indications of the disease or the effect; or

(c) where there is a valid biological monitoring procedure available and a reasonable likelihood that values might be exceeded.

14.4 Employees should participate in the health surveillance program unless there is some compelling reason to the contrary, in which case the matter should be discussed with the registered medical practitioner responsible for the health surveillance program.

RESPONSIBILITY FOR HEALTH SURVEILLANCE

14.5 The employer is responsible for providing health surveillance which has been established as necessary as a result of the assessment process. A registered medical practitioner shall be responsible for the supervision of health surveillance, either by directly carrying out the health surveillance program or by supervising a program carried out by a suitably qualified person such as an occupational health nurse. Coordination of the selection of a registered medical

practitioner to supervise health surveillance is the responsibility of the employer, in order to ensure that consistent methods are used for the health surveillance of employees exposed to the same hazardous substance. However, the selection of the registered medical practitioner shall be done in consultation with the employees concerned in order to give these employees a reasonable choice in the selection of the medical practitioner. In normal circumstances, the registered medical practitioner should be appropriately qualified in occupational medicine.

EMPLOYER RESPONSIBILITIES

14.6 The employer shall:

(a) pay any reasonable expenses due to health surveillance, for example, medical fees, pathology tests, travelling expenses and time off work; and

(b) ensure that health surveillance results obtained are retained as a confidential record for the purposes of the national model regulations.

14.7 The employer should also:

(a) inform employees of the purpose and procedures for health surveillance;

(b) make acceptable arrangements for employees to participate in the health surveillance program;

(c) provide the registered medical practitioner with access to a list of the hazardous substances for which employees are required to have health surveillance and a copy of the MSDS and exposure standards information for those hazardous substances; and

(d) permit the registered medical practitioner to have access to any relevant assessment reports.

14.8 Where the employer receives notice from the registered medical practitioner of an adverse health surveillance result considered by the practitioner to be related to exposure to a hazardous substance in the workplace, action should be taken, as soon as practicable, to reassess the workplace and to provide appropriate controls to minimise any further risks to health or safety.

14.9 Where a registered medical practitioner has certified that an employee is unfit for further exposure to a hazardous substance in the workplace or should only work under conditions specified by the medical practitioner, the employer should follow these recommendations. This may involve relocating the employee to suitable alternative work or changes to the work to prevent exposure. This should be done only after consultation with the employee, employee representatives and the registered medical practitioner.

A SUITABLY TRAINED REGISTERED MEDICAL PRACTITIONER

14.10 Where there are no registered medical practitioners authorised by the relevant public authority for a specified test or examination, the registered medical practitioner shall be adequately trained to undertake the health surveillance. The registered medical practitioner should also have an understanding of the employees' work activities and be aware of his or her own duties under the national model regulations.

THE APPOINTED REGISTERED MEDICAL PRACTITIONER'S RESPONSIBILITIES

14.11 The responsibilities of the appointed registered medical practitioner are to:

(a) assist with the planning and implementation of health surveillance;

(b) maintain medical records and ensure their confidentiality;

(c) advise each employee of the results of his or her health surveillance, provide any necessary explanation and arrange treatment, preventive measures or rehabilitation, if necessary;

(d) decide if a clinical finding or examination result is abnormal, if a trend is significant and whether this indicates an unacceptable level of exposure to a hazardous substance;

(e) notify the employer of the outcome of health surveillance and of any trends which indicate inadequate control and the need for remedial action (the information provided to the employer shall allow the registered medical practitioner to maintain medical confidentiality);

(f) notify the relevant public authority of any adverse effect prescribed by that authority; and

(g) ensure that health surveillance results are maintained as confidential medical records, and in doing so:

(i) clearly identify them from records obtained for other purposes such as records of examinations not connected with health surveillance, and

(ii) provide the relevant public authority with all health surveillance records in their possession on cessation of their medical practice.

LENGTH OF TIME HEALTH SURVEILLANCE RESULTS SHALL BE KEPT

14.12 The results of health surveillance shall be kept by the employer for at least 30 years from the date of the last entry made in the records.

SCHEDULE 3 — NATIONAL MODEL REGULATIONS FOR THE CONTROL OF WORKPLACE HAZARDOUS SUBSTANCES

HAZARDOUS SUBSTANCES FOR WHICH HEALTH SURVEILLANCE IS REQUIRED

NOTE: The second column of Schedule 3 provides guidance as a shorthand summary of the health surveillance guidelines, which should be referred to if health surveillance is being considered.

HAZARDOUS SUBSTANCE	TYPE OF HEALTH SURVEILLANCE
acrylonitrile	• Demography, occupational and medical history and health advice. • Physical Examination if indicated. • Records of personal exposure.
inorganic arsenic	• Demography, occupational and medical history and health advice. • Physical examination with emphasis on the peripheral nervous system and skin. • Urinary total arsenic. • Records of personal exposure.
asbestos	• Demography, occupational and medical history and health advice. • Physical examination if indicated. • Records of personal exposure.
benzene	• Demography, occupational and medical history and health advice. • Baseline blood sample for haematological profile. • Records of personal exposure.
cadmium	• Demography, occupational and medical history. • Health advice, including counselling on additional cadmium burden from smoking. • Physical examination with emphasis on the respiratory system. • Completion of a standardised respiratory questionnaire. • Standardised respiratory function tests such as FEV_1, FVC and FEV_1/FVC. • Urinary cadmium and β_2-microglobulin. • Records of personal exposure.

HAZARDOUS SUBSTANCE	TYPE OF HEALTH SURVEILLANCE
inorganic chromium	• Demography, occupational and medical history and health advice. • Physical examination with emphasis on the respiratory system and skin. • Weekly skin inspection of hands and forearms by a 'responsible person'.
creosote	• Demography, occupational and medical history. • Health advice, including recognition of photosensitivity and skin changes. • Physical examination with emphasis on the neurological system and skin, noting any abnormal lesions, and evidence of skin sensitisation. • Records of personal exposure, including photosensitivity.
isocyanates	• Demography, occupational and medical history and health advice. • Completion of a standardised respiratory questionnaire. • Physical examination of the respiratory system and skin. • Standardised respiratory function tests such as FEV_1 FVC and FEV_1/FVC.
inorganic mercury	• Demography, occupational and medical history and health advice. • Physical examination with emphasis on neurological, renal and gastrointestinal systems and skin. • Urinary inorganic mercury.
4,4'- methylene bis 2-chloroaniline (MOCA)	• Demography, occupational and medical history and health advice. • Urinary total MOCA. • Dipstick analysis of urine for haematuria. • Urine cytology.

HAZARDOUS SUBSTANCE	TYPE OF HEALTH SURVEILLANCE
organophosphate pesticides	• Demography, occupational and medical history and health advice. • Physical examination. • Baseline estimation of red cell and plasma cholinesterase activity levels by the Ellman method. Estimation of red cell and plasma cholinesterase activity towards the end of the working day.
pentachlorophenol (PCP)	• Demography, occupational and medical history and health advice. • Physical examination with emphasis on skin, noting any abnormal lesions or effects of irritancy. • Urinary total pentachlorophenol. • Dipstick urinalysis for haematuria and proteinuria. • Records of personal exposure.
polycyclic aromatic hydrocarbons (PAH)	• Demography, occupational and medical history. • Health advice, including recognition of photosensitivity and skin changes. • Physical examination if indicated. • Records of personal exposure, including photosensitivity.
crystalline silica	• Demography, occupational and medical history and health advice. • Completion of a standardised respiratory questionnaire. • Standardised respiratory function tests such as FEV_1, FVC and FEV_1/FVC. • Chest X-ray, full size PA view. • Records of personal exposure.
thallium	• Demography, occupational and medical history and health advice. • Physical examination if indicated. • Urinary thallium.
vinyl chloride	• Demography, occupational and medical history and health advice. • Physical examination if indicated. • Records of personal exposure.

APPENDIX 3

HEALTH SURVEILLANCE EXPERT WORKING GROUP: CRITERIA FOR DETERMINING IF A HAZARDOUS SUBSTANCE SHOULD BE SCHEDULED AS REQUIRING HEALTH SURVEILLANCE

INTRODUCTION

These criteria were developed by the Health Surveillance Expert Working Group to consistently and systematically select hazardous substances that require scheduling for health surveillance. The criteria may be useful for employers in identifying hazardous substances for health surveillance in accordance with sub-regulation 14 (1)(b) of the National Commission's *National Model Regulations for the Control of Workplace Hazardous Substances* [NOHSC:1005(1994)][1]. Criterion one is included for completeness, but relates to a national level screening process and would not apply to individual workplaces.

The criteria are a series of questions which are intended to investigate a wide variety of issues relating to health surveillance. They are structured in a flow chart format (***see*** Attachment 1) and are designed to be answered by either 'yes' or 'no'. If on the weight of evidence the substance reaches Section 9 of the flow chart, then the use of the substance is likely to warrant a health surveillance program.

Beneath each criterion is a series of questions which identify specific areas to be considered when answering the criterion. The questions address areas which will help explore the criteria. Not all the questions are required to be answered. A 'yes' answer to all the questions is not necessary for an overall 'yes' to the criterion.

There are eight questions in the flow chart:

- Is the substance used or expected to be used in Australia?
- Is the substance hazardous to health?
- Is there evidence that the substance is injuring the health of workers or is there reason to suspect that this could be so, under the anticipated conditions of use?
- Is atmospheric monitoring, without health surveillance, sufficient to evaluate exposure to the substance?
- Are there health surveillance techniques available for this substance?
- Would health surveillance be beneficial for those at risk?
- Are the methods of health surveillance likely to be acceptable to those at risk?
- Are the methods of health surveillance practical and ethically acceptable?

The remainder of this paper will discuss the criteria in greater detail.

PHASE I

FLOW CHART QUESTIONS

Is the substance used or expected to be used in Australia?

This is a straightforward initiating criterion. If the product is not being used in Australia or is not expected to be used in the future, then there is no reason to include it in the health surveillance schedule.

A check should be made to ascertain if the substance is listed on the *Australian Inventory of Chemical Substances*[2]. This will indicate if the chemical is, or can be, used in Australia without being assessed under the National Industrial Chemicals Notification and Assessment Scheme. If the substance is not on this list and the answer to the question is unknown, then the substance should be considered for possible health surveillance scheduling until there is a determination that the substance will not be used in Australia.

Is the substance hazardous to health?

The National Commission's *Approved Criteria for Classifying Hazardous Substances* [NOHSC:1008(1994)][3] is the basis for determining if a substance is hazardous. The national model regulations define a hazardous substance as 'a substance which has the potential through being used at work to harm the health or safety of persons in the workplace'[3]. There are certain substances which are outside this definition when their use is not related to a work activity. Examples of these substances include foodstuffs, therapeutic agents, cosmetics, tobacco or products made of tobacco, and toiletries and toilet products[3].

The approved criteria specifies the criteria by which a substance is classified as being hazardous[3]. The approved criteria also gives details on how to apply the criteria[3]. The classification of a substance and the information on how the classification is based should be documented. A literature search will usually be required to determine if a substance is hazardous.

The *List of Designated Hazardous Substances* [NOHSC:10005(1994)][4] also provides a quick reference which provides classification details for a large number of substances.

Is there evidence that the substance is injuring the health of workers or is there reason to suspect that this could be so, under anticipated conditions of use?

Once a substance has been found to be hazardous, there needs to be an assessment as to whether it represents a risk under the anticipated conditions of use. A range of information would be used to establish this.

First, consideration needs to be given to the population at risk. This will involve identifying the total number of people exposed and also any susceptible populations. The likely range of exposures must be determined, that is, frequency and duration of exposure. All this information can be used in deciding the magnitude of the risk presented and also whether the correct group is being targeted. Table 7A in the document *Selection Procedures for Priority Existing Chemicals*[5] will aid in this process if there is a significant risk to health.

Another consideration is the variability and effectiveness of control of occupational exposure to the substance. A description of the processes to manufacture and use the substance should be obtained*.

Consideration should be given to the potential routes of exposure to the substance and how these can be assessed. This includes methods for determining exposures from inhalation or dermal absorption. Also, it requires consideration of the substance, its toxicology, metabolism and the substance's physical characteristics.

Is atmospheric monitoring, without health surveillance, sufficient to evaluate exposure to the substance?

For this question, one must determine if an atmospheric monitoring system exists which can adequately evaluate the existing workplace control regime. Consideration also needs to be given as to whether the monitoring of the substance in the workplace can be related to the health effects anticipated.

A 'yes' answer to this question does not mean that health surveillance for the substance in question should not be considered. The existence of atmospheric monitoring for a substance may indicate that this type of monitoring is adequate to evaluate the control of the substance in the workplace. Health surveillance should be considered in relation to atmospheric monitoring, and the best method to evaluate control of the workplace hazard should be identified.

* This generic approach is paralleled by the more precise risk assessment required in individual workplaces.

Are there health surveillance techniques available for this substance?

The next step in the procedure is to decide whether health surveillance techniques exist. Health surveillance is defined by the National Commission as 'the monitoring of individuals for the purpose of identifying changes in health status due to occupational exposure to a hazardous substance.' It includes biological monitoring[1].

The major focus of health surveillance is the monitoring of individuals. However, health surveillance also includes the systematic collection, analysis and evaluation of health data in order to identify individual disease cases or population patterns and trends which suggest adverse health effects and the need for future investigation or remedial action.

Health surveillance includes more than just biological monitoring. It is a process which may include the taking of an occupational history, a physical examination and laboratory tests which include testing for biological monitoring, as well as biological effect monitoring.

Methods to determine if health surveillance techniques exist for a substance include determining if surveillance is required by States, Territories or other countries. If this is the case, the techniques used should be defined. Another method for identifying whether health surveillance techniques exist for a substance is determining if a Biological Exposure Indices exists[6]. This can indicate that a test with an action level has been developed. A review of the relevant scientific literature should be undertaken in order to establish the supporting text for the Biological Exposure Indices. All techniques which are available should be documented. The toxicokinetics of the substance should be documented, if known.

In any of the health surveillance techniques identified, the test's validity and reliability should be explored. A technique is valid for health surveillance if it possesses a high sensitivity and specificity. This will allow the highest portion of people adversely affected by exposure to be detected. Not all tests possess a high sensitivity and specificity. Unfortunately, no single set of criteria for a health surveillance test's performance can be recommended. Determining acceptable performance is greatly dependent on the prevalence of disease in the screened population.

An ideal health surveillance test should produce consistent results upon repeated testing. The test's reproducibility depends on a number of factors—the imprecision of the test itself and the intra-individual biological variation in the biochemical or physiological parameter being measured. To determine if a test is reliable, one must determine how much test imprecision is tolerable.

The final step in determining if a test is valid is to decide if the results have meaning. Therefore, a test must have a standard reference range which is interpretable and easy to make comparisons with. Without a reference range, the test results will have limited intrinsic meaning.

Another question to answer when reviewing a health surveillance technique is Can a test detect early adverse effects? Early detection allows for implementation of preventive measures at a stage where the adverse effect can be averted. A health surveillance test which does not have the ability to detect early adverse effects is still useful if it allows for preventive measures to be enacted which will prevent further adverse effects.

Would health surveillance be beneficial for those at risk?

When analysing the benefits of health surveillance techniques, two types of benefits should be accounted for—individual and group benefits. For the individual, the most important benefit is whether there is an exposure level identified by the test which can be used to motivate the improvment of controls in order to reduce risk or prevent further exposure and progression of the adverse effect. Identification of this exposure level fulfils the most important purpose of a health surveillance program, which is the prevention of a workplace hazard.

Another benefit which results from health surveillance is the ability to assess whether control methods employed in the workplace are effective for reducing an individual's exposure.

Finally, if through health surveillance the individual can be educated to reduce their risk, the individual will ultimately benefit.

Groups, as well as individuals, can benefit from health surveillance. Through health surveillance of a group, control measures can be evaluated, risk assessment can be performed for foreseen hazards, and the future likelihood of risks can be evaluated by epidemiological techniques. Therefore, when assessing the advantages of a health surveillance technique, both the individual

and group benefits should be reviewed.

Are the methods of health surveillance likely to be acceptable to those at risk?

In order for a health surveillance technique to be successful, it needs to be acceptable to the individuals at risk. This means, acceptance in terms of personal convenience, attendant risk, side effects and perceived personal value. A surveillance test which is inconvenient, represents a risk to an individual, or is not perceived by the individual as an advantage to partake in, will alienate workers.

Are the methods of health surveillance practical and ethically acceptable?

The final question to be asked in determining if health surveillance is appropriate for a hazardous substance is whether the techniques used are administratively practical and ethically acceptable? Can the tests be accomplished in a feasible manner by all who would be involved in the monitoring? Can the results of the tests be evaluated in an effective manner? Are the test results interpretable?

PHASE II

Consider substance for health surveillance program.

If the substance satisfies all of the questions (and therefore should be included on the health surveillance schedule), then Phase II of assessment of the substance is appropriate. This will focus on monitoring and prevention of occupational disease through a health surveillance program, and development of specific guidelines. An extensive literature search should be completed in order to develop and evaluate such a program.

Health surveillance is unlikely to be appropriate for this substance.

If some of the answers to questions are equivocal or negative, then a decision for scheduling for health surveillance needs to be made on the weight of the evidence.

When scheduling for health surveillance is clearly not appropriate, the need for health surveillance should be monitored. As new information arises for a substance, the criteria should be periodically reapplied to determine if scheduling for health surveillance is warranted.

CONCLUSIONS

This flow chart is to assist in determining whether a substance is appropriate for health surveillance. Once this process has been completed and the substance has been determined to be appropriate for a health surveillance program, then further investigation will be required. This investigation will need to focus on the details of developing a health surveillance program and the components of the program.

ATTACHMENT 1

PHASE I — FLOW CHART FOR DETERMINATION IF HEALTH SURVEILLANCE IS REQUIRED FOR A SUBSTANCE

1. IS THE SUBSTANCE USED OR EXPECTED TO BE USED IN AUSTRALIA?

- Check against the *Australian Inventory of Chemical Substances.*

Yes **No**

2. IS THE SUBSTANCE HAZARDOUS TO HEALTH?

- Determine whether the substance is on the National Commission's *List of Designated Hazardous Substances* [NOHSC:10005(1994)].
- If not, then determine if the substance meets the definition of a hazardous substance. Review literature relevant to the substance in order to determine if the substance meets the National Commission's *Approved Criteria for Classifying Hazardous Substances* [NOHSC:1008(1994)]. Define how the substance meets the *Approved Criteria for Classifying Hazardous Substances* [NOHSC:1008(1994)].

Yes **No**

3. IS THERE EVIDENCE THAT THE SUBSTANCE IS INJURING THE HEALTH OF WORKERS OR IS THERE REASON TO SUSPECT THAT THIS COULD BE SO, UNDER THE ANTICIPATED CONDITIONS OF USE?

- Consider the total population at risk.
- Does a susceptible population exist?
- Determine the likely range of exposures.
- Assess the ability to control occupational exposure.
- Consider the hazards from all routes of exposure.

Yes **No**

4. IS ATMOSPHERIC MONITORING, WITHOUT HEALTH SURVEILLANCE, SUFFICIENT TO EVALUATE EXPOSURE TO THE SUBSTANCE?

- Is current monitoring sufficient to evaluate the control regime?
- Can the ability to monitor the substance in the workplace be related to the health effects anticipated?

No **Yes**

5. ARE THERE HEALTH SURVEILLANCE TECHNIQUES AVAILABLE FOR THIS SUBSTANCE?

- Consider the definition of health surveillance.
- Determine if health surveillance is required by States, Territories or other countries.
- Consider Biological Exposure Indices.
- Consider the toxicokinetics of the substance.
- Are these techniques valid and reliable?
- Define the sensitivity, specificity and positive predictive value.

Yes **No**

6. WOULD HEALTH SURVEILLANCE BE BENEFICIAL TO THOSE AT RISK?

Individual Benefits:

- Is there an exposure level identifiable which will reduce risk, reverse disease or prevent progression?
- Control methods can be assessed to determine if they are effective for reducing an individual's exposure.
- Education on prevention can be delivered effectively.

Group Benefits:

- Adequacy of control measures can be evaluated.
- Adequacy of risk assessment for foreseen hazards can be reviewed.
- Likelihood of risks which may need to be assessed can be analysed by epidemiological techniques.

Yes **No**

7. ARE THE METHODS OF HEALTH SURVEILLANCE LIKELY TO BE ACCEPTABLE TO THOSE AT RISK?

- The test must be tolerated and accepted by the persons at risk.
- Would the persons at risk cooperate with the health surveillance?
- Consider other factors, such as cultural and religious.
- Consider any side effects which may result from health surveillance.

Yes **No**

8. ARE THE METHODS OF HEALTH SURVEILLANCE PRACTICAL AND ETHICALLY ACCEPTABLE?

- The tests to be conducted are feasible.
- Evaluation of effectiveness is feasible.
- The tests must be interpretable.

Yes **No**

9. CONSIDER SUBSTANCE FOR HEALTH SURVEILLANCE PROGRAM—PHASE II

- Define criteria for the assessment (measurements).
- Define guidelines and procedures (that is, how to conduct health surveillance).

10. HEALTH SURVEILLANCE IS UNLIKELY TO BE APPROPRIATE FOR THIS SUBSTANCE.

REFERENCES

1. National Occupational Health and Safety Commission, *National Model Regulations for the Control of Workplace Hazardous Substances* [NOHSC:1005(1994)], Australian Government Publishing Service, Canberra, 1994.

2. Commonwealth Environment Protection Agency, *Australian Inventory of Chemical Substances*, Australian Government Publishing Service, Canberra, 1992.

3. National Occupational Health and Safety Commission, *Approved Criteria for Classifying Hazardous Substances* [NOHSC:1008(1994)], Australian Government Publishing Service, Canberra, 1994.

4. National Occupational Health and Safety Commission, *List of Designated Hazardous Substances* [NOHSC:10005(1994)], Australian Government Publishing Service, Canberra, 1994.

5. National Industrial Chemicals Notification and Assessment Scheme, *Selection Procedures for Priority Existing Chemicals*, Worksafe Australia, Sydney, October 1991.

6. American Conference of Governmental Industrial Hygienists, *Documentation of the Threshold Limit Values and Biological Exposure Indices,* 6th Ed, American Conference of Governmental Industrial Hygienists, Cincinnati, Ohio, 1991.

RESPIRATORY QUESTIONNAIRES

MEDICAL RESEARCH COUNCIL (MRC) QUESTIONNAIRE ON RESPIRATORY SYMPTOMS — 1986

And

INTERNATIONAL UNION AGAINST TUBERCULOSIS (IUAT) BRONCHIAL SYMPTOMS QUESTIONNAIRE — 1986

PREFACE

The questionnaires contained in this publication are two examples of internationally accepted standardised respiratory questionnaires. These questionnaires are referred to in the National Occupational Health and Safety Commission's *Guidelines for Health Surveillance* [NOHSC:7039(1995)] for certain hazardous substances where assessment of the respiratory system has been recommended.

In the *Guidelines for Health Surveillance* [NOHSC:7039(1995)], standardised respiratory questionnaires are recommended for use as an aid to data collection in a more inclusive, uniform and comparable way. They are suggested, together with the occupational and medical history and physical examination, in order to assist the appointed medical practitioner in the health surveillance of people at work potentially exposed to a particular hazard.

It is intended that the appointed medical practitioner responsible for conducting health surveillance be free to select a questionnaire or alternatively those questions most suited to the hazardous substance under consideration.

OTHER SUITABLE RESPIRATORY QUESTIONNAIRES

'Respiratory Symptoms Study in the Aluminium Industry', further details on this respiratory questionnaire are available in Kongerud J, Vale J R and Aalen O O, 'Questionnaire Reliability and Validity for Aluminium Potroom Workers', *Scandinavian Journal of Environmental Health*, vol. 15, pp. 364-70, 1989.

Questionnaire on

Respiratory Symptoms (1986)*

Instructions to Interviewers

The diagnosis of chronic bronchitis and other respiratory disorders during life is at present largely based on symptoms, together with other features of the clinical history, X-rays and/or lung function tests. It is well known, however, that the symptoms to which an individual admits may be influenced to some extent by the exact phrasing of the questions and by the person who asks them. To overcome some of these difficulties, this questionnaire provides a set of standard questions for inquiring about the presence or absence of common respiratory symptoms. The aim in completing it is to elicit the facts and to avoid bias due to different techniques of questioning. Provision is made for the inclusion of some basic ventilatory capacity measurements, but additional tests may be incorporated as appropriate to each investigation.

Training

Before embarking on a survey, the questionnaire and instructions should be studied and any difficulties discussed. Interviewers should apply the questionnaire to 10 or more subjects (such as hospital patients) who have at least some chest symptoms (since no difficulty arises with subjects who answer all questions with a confident 'no'). These interviews should be either witnessed by an experienced colleague or, better tape-recorded so that any mistakes or doubtful points can be corrected and clarified at leisure afterwards. Tape-recordings of a series of interviews based on the questionnaire are available and should be listened to if possible. These tapes are designed to illustrate difficulties arising in the interpretation of answers to the standard questionnaire during field surveys. A series of interviews is also provided which a potential interviewer can use to compare his own ratings of the responses given with those of the group of British workers responsible for the production of the tapes.

General instructions

Before starting to ask questions an interviewer should instruct subjects to answer simply 'yes' or 'no' to the questions. The actual printed wording should be used for each question. In most cases this should lead to a simple 'yes' or 'no' answer, which should be accepted and recorded. Occasionally the subject will express doubt about the meaning of the question or the appropriate reply. When this happens further probing will be needed. Repetition of the question is usually sufficient. Some guidance for dealing with the commoner difficulties is given below. When, after a brief explanation, doubt remains about whether the answer is 'yes' or 'no', the answer should be recorded as 'no'.

Recording the replies to the questions

The questionnaire has been set out to facilitate transfer of the data to punched cards. Most of the questions are of the 'yes'/'no' type and replies to these questions may be coded directly in the boxes provided. Instructions for coding responses need to be defined by the survey planner before the survey begins. (Suggested coding: yes = 1, no = 2, not applicable = 8). Where the answer to a question is a number, for example, the number of cigarettes smoked (Q. 17b), the number may be recorded directly in the boxes provided. Where the question is of a more 'open' type, for example, occupation, or brand of cigarettes smoked, the reply may be recorded in full and the coding performed later. In some studies, however, a coding schedule for these factors may be drawn up before the study begins (for example, civil state: 1 = single, 2 = married, 3 = widowed, 4 = divorced, 5 = other) and replies may be recorded directly in the boxes provided.

Comments on individual items

Ethnic group: This should be defined in a way that is appropriate for the study, as reporting of respiratory symptoms depends to some extent on cultural and ethnic background.

Occupation and industry: Details of occupation that need to be recorded may vary with each survey before interviewing begins.

Cough and phlegm:

Question 1 Count a cough with first smoke or on first going out of doors. Exclude clearing the throat or a single cough.

Question 4 Count phlegm with first smoke or on first going out of doors. Exclude phlegm from the nose, count phlegm swallowed.

In those parts of the world where respiratory symptoms are most common at some other time of the year, the appropriate word should be substituted for 'winter'. Where there is no seasonal variation in respiratory symptoms the word 'winter' should be omitted. When night shift workers are interviewed, the words 'on getting up' should be used instead of 'first thing in the morning' in questions 1 and 4.

With regard to coughing during the day, in question 2, an 'occasional' cough may be considered normal and the answer should then be recorded as 'no'. It is impossible to define the limits of 'occasional' accurately, but to provide a rough guide it is suggested that single coughs of a frequency of less than six per day are 'occasional'. On the other hand, in question 5, 'occasional' phlegm production from the chest is considered abnormal if it occurs twice or more per day. The interviewer may use any suitable word that accords with local usage provided that it distinguishes phlegm from the chest or throat from pure nasal discharge. Some subjects admit to bringing up phlegm without admitting to coughing. This should be accepted without changing the replies to the questions about cough. A claim that phlegm is coughed from the chest but swallowed counts as a positive reply.

In questions 1, 2, 4 and 5 the word 'usually' should be emphasised. If one of the first two questions about cough (1-2) or one of those on phlegm (4-5) is answered clearly 'yes', questions 3 and 6 should be asked as confirmatory questions, and they should be asked at the point at which they are printed in the questionnaire (as in Example 1, questions 4 and 5).

Example 1

Q4 Interviewer:	Do you usually bring up any phlegm from your chest first thing in the morning in the winter?
Subject:	Yes

Q5 Interviewer: Do you **usually** bring up any phlegm from your chest during the day, or at night, in the winter?

Subject: Yes, but only a little bit.

Q6 Interviewer: Do you bring up phlegm like this on most days for as much as three months each year?

Subject: No, not as often as that.

The interviewer should record these answers as follows:
Question 4: Yes, Question 5: Yes, Question 6: No.

If, however, a doubtful answer to question 1 or 2 or to question 4 or 5 is obtained (for example, 'yes, sometimes') question 3 or 6 should be asked immediately as a probing question. If the answer to the probing question is 'no' the answer to the basic question should be recorded as if it had been 'no'. If a subsequent question in the same set receives a definite 'yes' the probing question should be repeated (see Example 2),

Example 2

Q1 Interviewer: Do you **usually** cough first thing in the morning in the winter?

Subject; Yes, sometimes.

Q3 Interviewer: Do you cough like this on most days for as much as three months each year?

Subject: Oh, no, not most days.

Q2 Interviewer: Do you **usually** cough during the day, or at night, in the winter?

Subject: Well from time to time.

Interviewer: Do you cough as much as six times a day?

Subject: Yes, more than that I'd say.

Q3 Interviewer: Do you cough like this on most days for as much as three months each year?

Subject: Well, not every day.

Interviewer: More often than not?

Subject: Yes, I'd say so.

The interviewer should record these answers as follows:
Question 1: No, Question 2: Yes, Question 3: Yes.

In question 7a the word 'increased' should be used only for subjects who have already admitted to some habitual cough and phlegm.

Breathlessness: In order to increase uniformity between surveys carried out at different seasons, it is suggested that the question on breathlessness should refer to the time of the year when breathlessness is at its worst. 'Hurrying' implies walking quickly. If the subject is disabled from walking by any condition other than heart or lung disease this should be recorded.

Wheezing: If this question is not understood, vocal demonstration of wheezing by the interviewer is often helpful. No distinction is made between those who only wheeze during the day and those who only wheeze at night. The word 'asthma' should not be used.

Chest illnesses: Asking about 'usual activities' is designed to avoid biases which are known to arise from sickness benefit considerations if subjects are asked about illnesses interfering with their work.

Smoking: Questions on smoking are essential in any study on respiratory symptoms, yet the reliability of answer has diminished over time. People are more likely to deny that they smoke than in the past and also to underestimate the amount smoked. With the change in cigarette types it is important also to know the tar and nicotine yields of the product. Since subjects are unreliable in reporting such detail, investigators should attempt to collect an empty cigarette pack from the smoker in order to identify the brand positively and thence to obtain tar/nicotine yields from published lists. Although a

question on inhaling is retained, this too is not reliably answered. The actual uptake of smoke components is determined by the individual's smoking pattern as well as by the amount and type of product smoked, and investigators are encouraged to use an objective method of assessment, eg there is a simple test for nicotine metabolites in urine samples (Ellard et al., *Thorax 1985*, vol. 40, pp. 351-357), and other tests based on blood or saliva samples are available.

Those who smoke cigarettes must also be asked about other forms of smoking. 'Small' cigars are those which are the same size as cigarettes: all cigars larger than cigarettes should be classified as 'other'. Amounts of tobacco (for pipe smoking) or hand-rolled cigarettes should be recorded in units appropriate for each study: the form is laid out for grams (1 ounce = 28g). Specific inquiry is made about smoking habits at weekends because some people smoke more or less at these times than during the week, and if necessary allowances should be made for this when assessing the weekly consumption.

An ex-smoker is defined as anyone who has smoked as much as one cigarette per day (or one large cigar per week or an ounce (= 28g) of tobacco per month) for as long as a year and who at the time of the interview had not smoked for 6 months or more.

Ventilatory capacity: The exact procedure to be adopted varies with the type of instrument used, and training sessions are required before embarking on a survey. Spirometric readings may include the forced expiratory volume in one second (FEV_1) and the forced vital capacity (FVC) from a number of successive blows. The recommended procedure is to obtain and report five technically satisfactory blows from each subject, then using the three highest FEV's and the three highest FVC's (not necessarily from the same blows) for the calculation of mean values, though other criteria may be adopted providing they are specified and adhered to within any given series of studies. The temperature of the instrument or in its surroundings is required to correct the values to BTPS: barometric pressure will normally only be required if measurements are made at a great altitude. Conventions for measuring and recording height and weight should be established care-fully: for example, height may be recorded without shoes, to the nearest cm below, and weight with light clothing to the nearest 1/10th kg below.

Peak expiratory flow rates (PEFR) are usually measured on a separate instrument, and they do not require temperature correction. Again the exact procedure will depend on the instrument selected for the survey, but two practice blows should be made, followed by three technically satisfactory ones.

Further information on the use of the questionnaire

This information sheet provides basic guidance to research workers concerned with the planning and conduct of surveys. The items available are as follows:

Questionnaire on respiratory symptoms (1986).

Instructions to Interviewers.

Inquiries about supplies of these items, or about training tapes and other background material related to earlier versions of the questionnaire should be sent to: Publications Group, Medical Research Council, 21 Park Crescent, London WIN 4AL.

CONFIDENTIAL

QUESTIONNAIRE ON

Respiratory Symptoms (1986)

Approved by Medical Research Council's Committee on Environmental and Occupational Health

Before this questionnaire is used the instruction sheet must be read

---

Surname

First name(s)

Address

Serial number ☐☐☐☐☐

Sex (M=1 F=2) ☐

Date of birth Day ☐☐ Month ☐☐ Year ☐☐

Name at birth if different from above

Own doctor
Name **Address**

Other identifying data

Civil state ☐

Occupation ☐☐

Industry ☐☐

Ethnic group ☐

Interviewer ☐☐

Date of Interview Day ☐☐ Month ☐☐ Year ☐☐

Use the actual wording of each question. Put 1= Yes, 2= No, or other codes as indicated in boxes. When in doubt record as no.

Preamble

I am going to ask some questions, mainly about your chest. I should like you to answer **Yes** or **No** whenever possible.

Cough

1 Do you usually cough first thing in the morning in winter? ☐

2 Do you usually cough during the day—or at night—in the winter? ☐

If Yes to 1 or 2

3 Do, you cough like this on most days for as much as three months each year? ☐

Phlegm

4 Do you **usually** bring up phlegm from your chest first thing in the morning in the winter? ☐

5 Do you **usually** bring up any phlegm from your chest during the day—or at night—in winter? ☐

If Yes to 4 or 5

6 Do you bring up phlegm like this on most days for as much as three months each year? ☐

Periods of cough and phlegm

7a In the past three years have you had a period of (increased) cough and phlegm lasting for three weeks or more? ☐

If Yes

7b Have you had more than one such period? ☐

Breathlessness

If subject is disabled from walking by any condition other than heart or lung disease, omit question 8 and enter 1 here ☐

8a Are you troubled by shortness of breath when hurrying on level ground or walking up a slight hill? ☐

If Yes

8b Do you get short of breath walking with other people of your own age on level ground? ☐

If Yes

8c Do you have to stop for breath when walking at your own pace on level ground? ☐

Wheezing

9 Have you had attacks of wheezing or whistling in your chest at any time in the last 12 months? ☐

10a Have you ever had attacks of shortness of breath with wheezing? ☐

If Yes

10b Is/was your breathing absolutely normal between attacks? ☐

11 Have you at any time in the last 12 months been woken at night by an attack of shortness of breath? ☐

Chest illnesses

12a During the past three years have you had any chest illness which has kept you from your usual activities for as much as a week? ☐

If Yes

12b Did you bring up more phlegm than usual in any of these illnesses? ☐

If Yes

12c Have you had more than one illness like this in the past three years? ☐

Past illnesses

Have you ever had, or been told that you have had:

13a An injury or operation affecting your chest ☐

13b Heart trouble ☐

13c Bronchitis ☐

13d Pneumonia ☐

13e Pleurisy ☐

13f Pulmonary tuberculosis ☐

13g Bronchial asthma ☐

13h Other chest trouble ☐

13i Hay fever ☐

Tobacco smoking **1 = Yes, 2 = No**

14 Do you smoke? ☐

If No

14a Have you ever smoked as much as one cigarette a day (or one cigar a week or an ounce of tobacco a month) for as long as a year? ☐

If No to both parts of question 14, omit remaining questions on smoking

15a Do (did) you inhale smoke? ☐

If Yes

15b Would you say you inhaled the smoke slightly = 1, moderately = 2, or deeply = 3? ☐

16 How old were you when you started smoking regularly? ☐☐

17a Do (did) you smoke manufactured cigarettes? ☐

If yes

17b How many do (did) you usually smoke per day on weekdays? ☐☐

17c How many per day at weekends? ☐☐

17d Do (did) you usually smoke plain (=1) or filter tip (=2) cigarettes? ☐

17e What brands do (did) you usually smoke? ☐☐☐

18a Do (did) you smoke hand-rolled cigarettes? ☐

If Yes

18b How much tobacco do (did) you usually smoke per week in this way? ☐☐☐

18c Do (did) you put filters in these cigarettes? ☐

19a Do(did) you smoke a pipe? ☐

If Yes

19b How much pipe tobacco do (did) you usually smoke per week? ☐☐☐

20a Do (did) you smoke small cigars? ☐

If Yes

20b How many of these do (did) you usually smoke per day? ☐☐

21a Do (did) you smoke other cigars? ☐

If Yes

21b How many of these do (did) you usually smoke per week? ☐☐

For present smokers

22a Have you been cutting down smoking over the past year? ☐

For ex-smokers Month Year

22b When did you give up smoking altogether? ☐☐ ☐☐

Additional observations

Ventilatory capacity

Standing height (m) ☐.☐☐

Weight (kg) ☐☐☐.☐

Ambient temperature (°C) ☐☐

Barometric pressure (mm Hg) ☐☐☐

Time of day (24 h) ☐☐ ☐☐

Observer ☐☐

Spirometer

Instrument number ☐☐☐☐

Enter readings as made, for subsequent correction to BTPS.

If additional readings are made, enter below number 5 and delete the ones they replace.

		FEV_1 (litres)	FVC (litres)
Reading	1	☐.☐☐	☐.☐☐
	2	☐.☐☐	☐.☐☐
	3	☐.☐☐	☐.☐☐
	4	☐.☐☐	☐.☐☐
	5	☐.☐☐	☐.☐☐

Peak expiratory flow

Instrument number ☐☐☐☐

If additional readings are made, enter below number 5 and delete the ones they replace

	PEFR (litres/min)
Reading 1	☐☐☐
2	☐☐☐
3	☐☐☐
4	☐☐☐
5	☐☐☐

Additional observations

Bronchial Symptoms Questionnaire*

PREPARED FOR THE RESPIRATORY DISEASE COMMITTEE OF THE
INTERNATIONAL UNION AGAINST TUBERCULOSIS

1986

ENQUIRES TO:

IUAT/UICT,
3 RUE GEORGES VILLE,
PARIS,
16,
FRANCE.

DEPARTMENT OF COMMUNITY MEDICINE,
UNITED MEDICAL AND DENTAL SCHOOLS,
ST THOMAS'S HOSPITAL,
LONDON, SE1 7EH,
ENGLAND.

IUAT BRONCHIAL SYMPTOMS QUESTIONNAIRE—1986

NOTES FOR USERS

The IUAT bronchial symptoms questionnaire has been developed for the Respiratory Disease Committee of the IUAT for use in epidemiological studies of asthma in adults. It is a self-administered questionnaire and is based on a program of research undertaken on behalf of the IUAT.

The questionnaire is composed of questions that relate to symptoms of airway disease. Most of these have been shown to be associated with bronchial hyperresponsiveness to inhaled histamine, though this is not true of all questions on cough which are included for the sake of completeness.

In addition to questions on symptoms, there are questions on attacks of asthma, on whether subjects are taking medication for asthma, on smoking habits, age and sex. In any particular survey it may be that there are other questions which need to be added. These should be included at the end of this questionnaire.

Coding of the Questionnaire

The questionnaire has been provided in a format that can be easily entered on a computer. Each answer has a column number beside it indicating into which column of data the response should be coded. Columns 1-10 have been left blank and should be used for the subject identification number. Subjects are asked to tick ('check') the appropriate box and instruction must be given to the data entry clerks on how to code these ticks when entering the data onto the computer. Where the answer to the question is either 'yes' or 'no' a positive answer should be coded '2' and a negative answer '1'. Questions 9, 15 and 17 should be coded '1', '2' or '3' according to whether the first, second or third box has been ticked. Where there is particular interest in smoking habits, it is possible to code column 27 according to the tar level of the cigarette normally smoked, but if researchers wish to do this, they should probably ask for an empty packet of the subject's cigarettes to be included with the questionnaire. Otherwise columns 27-30 should be coded '2' if ticked and '1' otherwise.

Interpretation of the Questionnaire

The questionnaire is currently issued as a collection of relevant questions without prejudice concerning their interpretation. The first question has been shown to be the best single predictor of bronchial hyperresponsiveness. This is so even for the German translation where there is considerable difficulty in translating the term 'wheezing'. As most of the questions are independently related to bronchial hyperresponsiveness it should be possible to devise a predictor of hyperresponsiveness that would improve on this. We are currently searching for the best overall predictor, but as the usefulness of any such predictor will depend on its overall ability to predict hyperreactivity in different settings, we are not yet in a position to give guidance on this.

Continuing Evaluation

The 1986 questionnaire is the first to be generally released. It is in translation in English, French, German, Spanish and Finnish. The repeatability of the questionnaires is known except for questions 4, 6 and 7 which were not in the pilot questionnaire. Again with the exception of questions 4, 6 and 7 the relationship of each question to bronchial reactivity has been measured in these languages. However, our knowledge of the questionnaire is incomplete and we are anxious to hear views from those who use the questionnaire and to collect data from different areas where this includes information either on repeatability or on the questionnaire in conjunction with data on non-specific bronchial hyperresponsiveness. Researchers using the questionnaire for research purposes who are willing to archive this material at the end of their research should write to:

Miss Susan Chinn
Department of Community Medicine
United Medical and Dental Schools
St Thomas's Hospital
London, SE1 7EH.

It is expected that the questionnaire will be revised in time. All comments will, in the meantime, be gratefully received either through members of the Respiratory Disease Committee or through the Director of the IUAT or directly to:

Dr Peter Burney
Department of Community Medicine
United Medical and Dental Schools
St Thomas's Hospital
London SE1 7EH.

IUAT BRONCHIAL SYMPTOMS QUESTIONNAIRE—1986

TO ANSWER THE QUESTIONS, PLEASE TICK THE APPROPRIATE BOX; IF YOU ARE UNSURE OF THE ANSWER PLEASE TICK "NO".

Wheeze and Tightness in the Chest

1. Have you had wheezing or whistling in your chest, at any time in the last 12 months? Yes ☐ No ☐ 11

2. Have you woken up with a feeling of tightness in your chest first thing in the morning, at any time in the last 12 months? Yes ☐ No ☐ 12

Shortness of Breath

3. Have you, at any time in the last 12 months, had an attack of shortness of breath that came on during the day when you were not doing anything strenuous? Yes ☐ No ☐ 13

4. Have you had an attack of shortness of breath that came on after you stopped exercising, at any time in the last 12 months? Yes ☐ No ☐ 14

5. Have you, at any time in the last 12 months, been woken at night by an attack of shortness of breath? Yes ☐ No ☐ 15

Cough and Phlegm from the Chest

6. Have you, at any time in the last 12 months, been woken at night by an attack of coughing? Yes ☐ No ☐ 16

7. Do you usually cough first thing in the morning? Yes ☐ No ☐ 17

8. Do you usually bring up phlegm from your chest first thing in the morning? Yes ☐ No ☐ 18

Breathing

9. Which of the following statements best describes your breathing? tick one box only 19

(a) I never or rarely get trouble with my breathing. ☐

(b) I get regular trouble with my breathing, but it always gets completely better. ☐

(c) My breathing is never quite right. ☐

Animals, dust, feathers

10. When you are in a dusty part of the house or with animals (for instance, dogs, cats or horses) or near feathers (including pillows, quilts and eiderdowns) do you ever:

	Yes	No	
(a) Get a feeling of tightness in your chest?	☐	☐	20
(b) Start to feel short of breath?	☐	☐	21

Illness

	Yes	No	
11. Have you ever had an attack of asthma?	☐	☐	22
12. Have you had an attack of asthma at any time in the last 12 months?	☐	☐	23
13. Are you currently taking any medicines, pills or inhalers for asthma?	☐	☐	24

Smoking

	Yes	No	
14. Have you ever smoked for as long as a year?	☐	☐	25

(This means at least one or more cigarettes a day (or one or more cigars a week or one or more ounces of pipe tobacco a month) for as long as a year).

15. Do you now smoke:

tick one box only

Not at all	☐	
Occasionally	☐	
Daily	☐	26

IF 'NOT AT ALL' GO TO QUESTION 17, OTHERWISE:

16. What do (did) you usually smoke?

tick any number of boxes

Manufactured cigarettes ._____. (please give full brand name)	☐	27
Hand rolled cigarettes	☐	28
Pipe	☐	29
Cigar	☐	30

17. If you have given up smoking altogether, how long is it since you last gave up smoking?

tick one box only

☐ Less than a month ago

☐ More than a month ago

31

More about yourself

18. When were you born?

☐☐ day ☐☐ month ☐☐ year

32-37

19. Are you a male or female?

MALE ☐ FEMALE ☐

38

20. What is today's date?

☐☐ day ☐☐ month ☐☐ year

39-44

21. How old are you?

☐☐ YEARS

45-46

1. GUIDELINES* FOR HEALTH SURVEILLANCE FOR ACRYLONITRILE

BASELINE HEALTH SURVEILLANCE AT TIME OF EMPLOYMENT IN AN ACRYLONITRILE PROCESS

1. Collection of Demographic Data

- Name and unique company identification number.
- Date of birth.
- Sex.
- Address.
- Date of starting company service.
- Descriptive job title. To include the Australian Bureau of Statistics' *Australian Standard Classification of Occupations* (ASCO)[1] and *Australian Standard Industrial Classification* (ASIC)[2].
- Places of previous employment.

2. Occupational History

- Past work history, including previous exposure to acrylonitrile.
- Potential current exposure.
- Whether suitable personal protective equipment is used for that specific acrylonitrile process.

3. Medical History

- Presence of symptoms.
- Smoking history.

4. Physical Examination

Conducted only if indicated by occupational and medical history.

5. Health Advice

The appointed medical practitioner should inform the employee of the potential health effects associated with exposure to acrylonitrile.

*These guidelines set out in a practical manner the minimum requirements for health surveillance and should be read in conjunction with the Introduction to the *Guidelines for Health Surveillance* [NOHSC:7039(1995)].

DURING EXPOSURE TO AN ACRYLONITRILE PROCESS

6. Personal Protective Equipment

The availability, type, fit, maintenance and frequency of use of personal protective equipment should be monitored regularly.

7. Data for Inclusion in Health Records

The following data should be included with the health records of the individual employee:

- Any formal assessments carried out in compliance with the National Occupational Health and Safety Commission's *National Model Regulations for the Control of Workplace Hazardous Substances* [NOHSC:1005(1994)][3].
- Descriptive job titles, with relevant start and finish dates. Those jobs within areas where acrylonitrile is used should be clearly identified.
- Results of atmospheric and personal monitoring, and investigation of results that exceed the national exposure standard.

8. Medical Examination

If employees are excessively exposed or suspected of being so, or have concerns which may be related to acrylonitrile exposure, they should be seen by the appointed medical practitioner.

The employer should be informed when abnormal findings are detected so that control measures can be checked. The employee should be informed of the results of the health surveillance.

AT TERMINATION OF EMPLOYMENT IN AN ACRYLONITRILE PROCESS

9. Data that should be Collected

- Date of termination.
- Reason for termination:
  - ill-health (if 'yes', give details),
  - other reasons, and
  - date and cause of death if in service.

2. SUPPLEMENTARY INFORMATION ON ACRYLONITRILE

WORK ACTIVITIES THAT MAY REPRESENT A HIGH RISK EXPOSURE

The major uses of acrylonitrile are in the manufacture of polymers, resins, plastics and nitrile rubbers.

Examples of work activities involving acrylonitrile which require special attention when assessing exposure include acrylic fibre production — especially in the procedure where solvent is removed from newly-formed fibres.

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

POTENTIAL HEALTH EFFECTS FOLLOWING EXPOSURE TO ACRYLONITRILE

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.

Acute Effects

Acrylonitrile is irritating to the eyes, nose, throat and skin. Acrylonitrile is a cellular asphyxiant with actions similar to cyanide, causing symptoms such as weakness, headache, nausea, shortness of breath, dizziness, collapse, convulsions and death.

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.

Carcinogen Classification

Acrylonitrile is listed in the National Commission's *List of Designated Hazardous Substances* [NOHSC:10005(1994)][4] and is classified as Carcinogen Category 2. According to the National Commission's *Approved Criteria for Classifying Hazardous Substances* [NOHSC:1008(1994)][5], a substance is assigned Carcinogen Category 2 if there is sufficient evidence, on the basis of appropriate long-term animal studies or other relevant information, to provide a strong presumption that human exposure to that substance may result in the development of cancer.

3. REFERENCED DOCUMENTS

1. Australian Bureau of Statistics, Australian Standard Classification of Occupations: ASCO Coding System, Australian Bureau of Statistics, Canberra, 1993.

2. Australian Bureau of Statistics, *Australian Standard Industrial Classficiation*, Australian Bureau of Statistics, Canberra, 1985.

3. National Occupational Health and Safety Commission, *National Model Regulations for the Control of Workplace Hazardous Substances* [NOHSC:1005(1994)], Australian Government Publishing Service, Canberra, 1994.

4. National Occupational Health and Safety Commission, *List of Designated Hazardous Substances* [NOHSC:10005(1994)], Australian Government Publishing Service, Canberra, 1994.

5. National Occupational Health and Safety Commission, *Approved Criteria for Classifying Hazardous Substances* [NOHSC:1008(1994)], Australian Government Publishing Service, Canberra, 1994.

4. FURTHER READING

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

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

Lauwerys RR and Hoet P, *Industrial Chemical Exposure Guidelines for Biological Monitoring,* 2nd Ed, Lewis Publishers, Boca Raton, 1993.

1. GUIDELINES* FOR HEALTH SURVEILLANCE FOR INORGANIC ARSENIC

BASELINE HEALTH SURVEILLANCE AT TIME OF EMPLOYMENT IN AN INORGANIC ARSENIC PROCESS

1. Collection of Demographic Data

- Name and unique company identification number.
- Date of birth.
- Sex.
- Address.
- Date of starting company service.
- Descriptive job title. To include the Australian Bureau of Statistics' *Australian Standard Classification of Occupations* (ASCO)[1] and *Australian Standard Industrial Classification* (ASIC)[2].
- Places of previous employment.

2. Occupational History

- Past work history, including previous exposure to inorganic arsenic.
- Potential current exposure.
- Whether suitable personal protective equipment is used for that specific arsenic process.

3. Medical History

- Presence of symptoms.
- Smoking history.

4. Physical Examination

With emphasis on the peripheral nervous system and skin.

5. Investigation

- Employees should be advised to abstain from seafood for three days prior to urine collection.
- Spot creatinine corrected urine for total arsenic to be conducted. Where there is 100 µg total arsenic or more per gram creatinine, a repeat spot creatinine corrected urine for total arsenic should be performed at the same time of the day.

*These guidelines set out in a practical manner the minimum requirements for health surveillance and should be read in conjunction with the Introduction to the *Guidelines for Health Surveillance* [NOHSC:7039(1995)].

6. Health Advice

The appointed medical practitioner should inform the employee of the potential health effects associated with exposure to inorganic arsenic.

DURING EXPOSURE TO AN INORGANIC ARSENIC PROCESS

7. Personal Protective Equipment

The availability, type, fit, maintenance and frequency of use of personal protective equipment should be monitored regularly.

8. Urinary Total Arsenic

Employees should be advised to abstain from seafood for three days prior to urine collection.

Baseline Level

Spot creatinine corrected urine for total arsenic to be conducted every 90 days. Where there is 150 µg total arsenic or more per gram creatinine, a repeat spot creatinine corrected urine for total arsenic should be performed at the same time of the day.

Action Level

On confirmation of a level of 150 µg total arsenic or more per gram creatinine, a medical examination with particular emphasis on the peripheral nervous system and skin should be performed.

The employer should be informed when abnormal findings are detected so that control measures can be checked. The employee should be informed of the results of the health surveillance.

Removal Level

Removal from arsenic work should be considered if the level is above 200 µg per gram of creatinine. Spot creatinine corrected urine for total arsenic should be repeated within 10 days and then at regular intervals until the level falls below 100 µg total arsenic per gram creatinine.

Return to Work

The person must be medically fit to return to arsenic work.

9. Data for Inclusion in Health Records

The following data should be included with the health records of the individual employee:

- Any formal assessments carried out in compliance with the National Occupational Health and Safety Commission's *National Model Regulations for the Control of Workplace Hazardous Substances* [NOHSC:1005(1995)][3].

- Descriptive job titles, with relevant start and finish dates. Those jobs within the area where arsenic is used should be clearly identified.
- Results of atmospheric and personal monitoring, and investigation of results that exceed the national exposure standard.

AT TERMINATION OF EMPLOYMENT IN AN INORGANIC ARSENIC PROCESS

10. Data that should be Collected

- Date of termination.
- Reason for termination:
  - ill-health (if 'yes', give details),
  - other reasons, and
  - date and cause of death if in service.

11. Continuing Medical Surveillance

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

2. SUPPLEMENTARY INFORMATION ON INORGANIC ARSENIC

WORK ACTIVITIES THAT MAY REPRESENT A HIGH RISK EXPOSURE

Arsenic exists in three common valence states—the metalloid (As^0), arsenite (trivalent state, As^{3+}) and arsenate (pentavalent state, As^{5+}).

Arsenic forms the basis of a large number of compounds which, when heated to decomposition or on contact with acids or acid fumes, emit highly toxic fumes of arsenic. It is a common contaminant in most mineral ores.

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, cattle dips (arsenic trioxide), fungicides (copper aceto-arsenite or Paris green), sheep dips (sodium arsenite), and as a wood preservative such as copper chrome arsenic (arsenic pentoxide);
- production of pigments (arsenic trisulphide and trioxide), ceramic enamels and anti-fouling paints (arsenic trioxide); and
- hide preservation in the leather industry (arsenic trioxide).

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

POTENTIAL HEALTH EFFECTS FOLLOWING USE OF INORGANIC ARSENIC

The relative toxicity of an arsenical 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, for example, 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 arsenical.

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 arsenical 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.

Acute Effects

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.

Chronic Effects

Arsenic trioxide dust may cause dermatitis of the face and eyelids, and conjunctivitis. In the presence of sweat, skin abrasions, chafing or wounds, arsenic readily promotes ulceration of the skin. Dermatitis, with scaling and excessive pigmentation of the skin, has occurred among workers manufacturing insecticides, in vineyard workers and unprotected smelter workers. Exposure to arsenic trichloride has also produced blistering of the skin. Irritation of the nose, throat and lungs, impaired respiration and perforation of the nasal septum have also been reported in smelter workers.

Chronic arsenic poisoning due to exposure to compounds such as calcium arsenate and copper acetoarsenate is characterised by weakness, loss of appetite, gastro-intestinal disturbances, numbness and tingling of the extremities (peripheral neuritis). Chronic exposure to arsenic compounds such as arsenic trioxide may lead to liver damage and skin disorders such as keratoses and pigmentation.

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

Carcinogen Classification

Arsenic (white) and arsenic trioxide are listed in the National Commission's *List of Designated Hazardous Substances* [NOHSC:10005(1994)][4] and both are classified as Carcinogen Category 1. According to the National Commission's *Approved Criteria for Classifying Hazardous Substances* [NOHSC:1008(1994)][5], a substance is assigned Carcinogen Category 1 if there is sufficient evidence to establish a causal association between human exposure and the development of cancer on the basis of epidemiological data.

The International Agency for Research on Cancer and the Exposure Standards Expert Working Group have carcinogen ratings for other forms of arsenic and arsenic compounds.

The Exposure Standards Expert Working Group[6] classification for arsenic and soluble compounds, and arsenic trioxide production, is Carcinogen Category 1, established human carcinogen (1995). Established human carcinogens are those substances known to be carcinogenic to humans. There is sufficient evidence to establish a causal association between human exposure to these substances and the development of cancer.

The International Agency for Research on Cancer[7] classification for arsenic and arsenic compounds is Group 1, carcinogenic to humans (1987). According to the International Agency for Research on Cancer, this category is used only when there is sufficient evidence of carcinogenicity in humans.

3. REFERENCED DOCUMENTS

1. Australian Bureau of Statistics, *Australian Standard Classification of Occupations: ASCO Coding System*, Australian Bureau of Statistics, Canberra, 1993.

2. Australian Bureau of Statistics, *Australian Standard Industrial Classification,* Australian Bureau of Statistics, Canberra, 1985.

3. National Occupational Health and Safety Commission, *National Model Regulations for the Control of Workplace Hazardous Substances* [NOHSC:1005(1994)], Australian Government Publishing Service, Canberra, 1994.

4. National Occupational Health and Safety Commission, *List of Designated Hazardous Substances* [NOHSC:10005(1994)], Australian Government Publishing Service, Canberra, 1994.

5. National Occupational Health and Safety Commission, *Approved Criteria for Classifying Hazardous Substances* [NOHSC:1008(1994)], Australian Government Publishing Service, Canberra, 1994.

6. National Occupational Health and Safety Commission, *Exposure Standards for Atmospheric Contaminants in the Occupational Environment* [NOHSC:1003(1995)], Australian Government Publishing Service, Canberra, 1995.

7. 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.

4. FURTHER READING

Agency for Toxic Substances and Disease Registry, *Case Studies in Environmental Medicine 5: Arsenic Toxicity,* Agency for Toxic Substances and Disease Registry, United States Department of Health and Human Services, Public Health Service, Atlanta, 1990.

Health and Safety Executive (United Kingdom), *Arsenic: Health and Safety Precautions,* Guidance Note EH 8, Health and Safety Executive, London, 1990.

Health and Safety Executive (United Kingdom), *Arsine: Health and Safety Precautions*, Guidance Note EH 11, Health and Safety Executive, London, 1990.

International Programme on Chemical Safety, *Environmental Health Criteria 18: Arsenic,* International Programme on Chemical Safety, World Health Organization, Geneva, 1981.

Lauwerys RR and Hoet P, *Industrial Chemical Exposure Guidelines for Biological Monitoring,* 2nd Ed, Lewis Publishers, Boca Raton, 1993.

Worksafe Australia, *Arsenic and its Compounds,* Australian Government Publishing Service, Canberra, 1989.

National Occupational Health and Safety Commission, *National Code of Practice* [NOHSC:2003(1989)] *and Guidance Note for the Safe Handling of Timber Preservatives and Treated Timber* [NOHSC:3007(1989)], Australian Government Publishing Service, Canberra, 1989.

1. GUIDELINES* FOR HEALTH SURVEILLANCE FOR ASBESTOS

These guidelines are consistent with the recommendations in the National Occupational Health and Safety Commission's *Guide to the Control of Asbestos Hazards in Buildings and Structures* [NOHSC:3002(1988)][1].

BASELINE HEALTH SURVEILLANCE AT TIME OF EMPLOYMENT IN AN ASBESTOS PROCESS

1. Collection of Demographic Data

- Name and unique company identification number.
- Date of birth.
- Sex.
- Address.
- Date of starting company service.
- Descriptive job title. To include the Australian Bureau of Statistics' *Australian Standard Classification of Occupations* (ASCO)[2] and *Australian Standard Industrial Classification* (ASIC)[3].
- Places of previous employment.

2. Occupational History

- Past work history, including previous exposure to asbestos.
- Potential current exposure.
- Whether suitable personal protective equipment is used for that specific asbestos process.

3. Medical History

- Presence of symptoms.
- Smoking history.
- Administration of a standardised respiratory questionnaire. Two examples are the International Union Against Tuberculosis' *Bronchial Symptoms Questionnaire 1986*[4] **or** the Medical Research Council's *Questionnaire on Respiratory Symptoms* 1986[5].

*These guidelines set out in a practical manner the minimum requirements for health surveillance and should be read in conjunction with the Introduction to the *Guidelines for Health Surveillance* [NOHSC:7039(1995)].

POINTS 4 AND 5 BELOW SHOULD ONLY BE CONDUCTED IF INDICATED BY THE OCCUPATIONAL AND MEDICAL HISTORY.

4. Physical Examination

With emphasis on the respiratory system.

5. Investigation

Chest X-ray and standardised respiratory function tests*. The tests are FEV_1, FVC and FEV_1/FVC. The norms for predictive values should be stated.

6. Health Advice

The appointed medical practitioner should inform the employee of the
potential health effects associated with exposure to asbestos.

DURING EXPOSURE TO AN ASBESTOS PROCESS

7. Personal Protective Equipment

The availability, type, fit, maintenance and frequency of use of personal protective equipment should be monitored regularly.

8. Data for Inclusion in Health Records

The following data should be included with the health records of the individual employee:

- Any formal assessments carried out in compliance with the relevant regulations, for example, regulations following the National Commission's *National Model Regulations for the Control of Workplace Hazardous Substances* [NOHSC:1005(1994)][6].
- Descriptive job titles, with relevant start and finish dates. Those jobs within the area where asbestos exposure occurs should be clearly identified.
- Results of atmospheric and personal monitoring, and investigation of results that exceed the national exposure standard.

9. Medical Examination

A medical examination every two years. The examination is a medical interview which includes:

- occupational history;
- medical history; but
- does not include respiratory function tests, chest X-ray and physical examination unless indications present.

The interview also provides an opportunity for assurance as to the understanding of specific preventive measures against asbestos exposure and for reassurance on any concerns about exposure.

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

AT TERMINATION OF EMPLOYMENT IN AN ASBESTOS PROCESS

10. Data that should be Collected

- Date of termination.
- Reason for termination:
  - ill-health (if 'yes', give details),
  - other reasons, and
  - date and cause of death if in service.

11. Final Medical Examination

A medical examination as described in point 9 above.

2. SUPPLEMENTARY INFORMATION ON ASBESTOS

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 products in the automotive industry — manufacture and installation of asbestos-containing automotive products, for example, brakelinings and gaskets;
- asbestos removal and demolition work in buildings, power stations, boilers and ships; and
- maintenance workers, such as electricians, and computer cabling installers and airconditioning installers working in ceiling spaces of buildings where sprayed asbestos has not been removed, sealed or encapsulated.

In some industries, such as mining and construction, amphiboles, such as tremolite and/or anthophyllite, are present as geological contaminants.

POTENTIAL HEALTH EFFECTS FOLLOWING EXPOSURE TO ASBESTOS

Route of Entry into the Body

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

Respiratory Effects

Small fibrous particles may become airborne and inhaled. Fibres below 3 μm in diameter are referred to as 'respirable', meaning that they may enter the deepest parts of the lung. Most larger fibres are deposited in the nose and major airways and are cleared by normal physiological processes. However, smaller fibres are generally deposited in the minor airways and alveoli.

Inhalation of high concentrations of asbestos may result in asbestosis, a progressive scarring of lung tissue. Further development of scar tissue (fibrosis) may occur after the cessation of exposure. Pleural effects of asbestos include plaques (with and without calcification), diffuse pleural thickening and effusions.

Carcinogenicity

The two main forms of cancer associated with the inhalation of asbestos are lung cancer and mesothelioma.

All forms of asbestos have been found to cause lung cancer, both in a variety of experimental animals and in exposed humans. Cigarette smoking increases the risk of lung cancer in people heavily exposed to asbestos. The risk of cancer is also greater with increased exposure (the product of airborne concentration and time) to asbestos.

Mesothelioma is cancer of the lining of the chest cavity (the pleura) or, less commonly, the lining of the abdominal cavity (the peritoneum). Crocidolite and amosite have the most potent documented effects in producing this tumour, which is highly malignant. There is a long latency period which ranges from 10 to 50 years between exposure and the development of mesothelioma. The majority of mesothelioma cases appear to be related mainly to crocidolite. The tumours have also been observed in occupational groups exposed to amosite. An association with chrysotile exposure is still being debated.

Carcinogen Classification

Asbestos (blue, brown and white) is listed in the National Commission's *List of Designated Hazardous Substances* [NOHSC:10005(1994)][7] and is classified as Carcinogen Category 1. According to the National Commission's *Approved Criteria for Classifying Hazardous Substances* [NOHSC:1008(1994)][8], a substance is assigned Carcinogen Category 1 if there is sufficient evidence to establish a causal association between human exposure and the development of cancer on the basis of epidemiological data.

3. REFERENCED DOCUMENTS

1. National Occupational Health and Safety Commission, 'Guide to the Control of Asbestos Hazards in Buildings and Structures' [NOHSC:3002(1988)], in *Asbestos: Code of Practice and Guidance Notes*, Australian Government Publishing Service, Canberra, 1988.

2. Australian Bureau of Statistics, *Australian Standard Classification of Occupations: ASCO Coding System,* Australian Bureau of Statistics, Canberra, 1993.

3. Australian Bureau of Statistics, *Australian Standard Industrial Classification*. Australian Bureau of Statistics, Canberra, 1985.

4. Respiratory Disease Committee of the International Union Against Tuberculosis, *IUAT Bronchial Symptoms Questionnaire,* International Union Against Tuberculosis, 1986☎.

5. Medical Research Council Committee on Research into Chronic Bronchitis, *MRC Questionnaire on Respiratory Symptoms*, Medical Research Council, 1986☎.

6. National Occupational Health and Safety Commission, *National Model Regulations for the Control of Workplace Hazardous Substances* [NOHSC:1005(1994)], Australian Government Publishing Service, Canberra, 1994.

7. National Occupational Health and Safety Commission, *List of Designated Hazardous Substances* [NOHSC:10005(1994)], Australian Government Publishing Service, Canberra, 1994.

8. National Occupational Health and Safety Commission, *Approved Criteria for Classifying Hazardous Substances* [NOHSC:1008 (1994)], Australian Government Publishing Service, Canberra, 1994.

☎Reproductions of these respiratory questionnaires can be purchased from Publications Sales at Worksafe Australia. Telephone (02) 577 9555.

4. FURTHER READING

Antman K, *Asbestos Related Malignancy,* Grune and Stratton, Orlando, Florida, 1987.

International Programme on Chemical Safety, *Environmental Health Criteria 53: Asbestos and Other Natural Mineral Fibres,* International Programme on Chemical Safety, World Health Organization, Geneva, 1986.

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.

1. GUIDELINES* FOR HEALTH SURVEILLANCE FOR BENZENE

BASELINE HEALTH SURVEILLANCE AT TIME OF EMPLOYMENT IN A BENZENE PROCESS

1. Collection of Demographic Data

- Name and unique company identification number.
- Date of birth.
- Sex.
- Address.
- Date of starting company service.
- Descriptive job title. To include the Australian Bureau of Statistics' *Australian Standard Classification of Occupations* (ASCO)[1] and *Australian Standard Industrial Classification* (ASIC)[2].
- Places of previous employment.

2. Occupational History

- Past work history, including previous exposure to benzene.
- Potential current exposure.
- Whether suitable personal protective equipment is used for that specific benzene process.

3. Medical History

- Presence of symptoms.
- Smoking history.

4. Investigation

Blood sample for haematological profile.

5. Health Advice

The appointed medical practitioner should inform the employee of the potential health effects associated with exposure to benzene.

* These guidelines set out in a practical manner the minimum requirements for health surveillance and should be read in conjunction with the Introduction to the *Guidelines for Health Surveillance* [NOHSC:7039(1995)].

DURING EXPOSURE TO A BENZENE PROCESS

6. Personal Protective Equipment

The availability, type, fit, maintenance and frequency of use of personal protective equipment should be monitored regularly.

7. Data for Inclusion in Health Records

The following data should be included with the health records of the individual employee:

- Any formal assessments carried out in compliance with the National Occupational Health and Safety Commission's *National Model Regulations for the Control of Workplace Hazardous Substances* [NOHSC:1005(1994)][3].
- Descriptive job titles, with relevant start and finish dates. Those jobs where exposure has been assessed as significant should be clearly identified.
- Results of atmospheric and personal monitoring, and investigation of results that exceed the national exposure standard.

8. Medical Examination

If employees are excessively exposed or suspected of being so, or have concerns which may be related to benzene exposure, they should be seen by the appointed medical practitioner.

The employer should be informed when abnormal findings are detected so that control measures can be checked. The employee should be informed of the results of the health surveillance.

AT TERMINATION OF EMPLOYMENT IN A BENZENE PROCESS

9. Data that should be Collected

- Date of termination.
- Reason for termination:
  - ill-health (if 'yes', give details),
  - other reasons, and
  - date and cause of death if in service.

10. Continuing Medical Surveillance

People identified with haematological abnormalities should be advised to seek continuing medical surveillance.

2. SUPPLEMENTARY INFORMATION ON BENZENE

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

WORK ACTIVITIES THAT MAY REPRESENT A HIGH RISK EXPOSURE

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

- refining operations, for example, maintenance of equipment used for handling benzene-containing refinery streams and sampling benzene-containing refinery streams in open containers;
- chemical manufacturing;
- handling of petrol, that is, storage and transport, for example, filling rail tankers and/or top-filling road tankers with gasoline; and
- plastics and rubber manufacturing.

POTENTIAL HEALTH EFFECTS FOLLOWING EXPOSURE TO BENZENE

Route of Entry into the Body

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

Acute Effects

The acute effects from exposure to high levels of benzene, that is 500-1,000 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.

Chronic Effects

Chronic exposure to levels of 100-500 ppm have resulted in depression of bone marrow haemopoiesis. This has led to anaemia, leucopoenia, thrombocytopenia or pancytopenia

Carcinogenicity

Acute myeloid leukaemia has been demonstrated to occur more frequently in workers occupationally exposed to benzene. Several reports suggest that exposure to benzene may be related to non-Hodgkins lymphoma and multiple myeloma.

Carcinogen Classification

Benzene is listed in the National Commission's *List of Designated Hazardous Substances* [NOHSC:10005(1995)][4] and is classified as Carcinogen Category 1. According to the National Commission's *Approved Criteria for Classifying Hazardous Substances* [NOHSC:1008(1994)][5], a substance is assigned Carcinogen Category 1 if there is sufficient evidence to establish a causal association between human exposure and the development of cancer on the basis of epidemiological data.

3. REFERENCED DOCUMENTS

1. Australian Bureau of Statistics, *Australian Standard Classification of Occupations: ASCO Coding System*, Australian Bureau of Statistics, Canberra, 1993.

2. Australian Bureau of Statistics, *Australian Standard Industrial Classification*, Australian Bureau of Statistics, Canberra, 1985.

3. National Occupational Health and Safety Commission, *National Model Regulations for the Control of Workplace Hazardous Substances* [NOHSC:1005(1994)], Australian Government Publishing Service, Canberra, 1994.

4. National Occupational Health and Safety Commission, *List of Designated Hazardous Substances* [NOHSC:10005(1994)], Australian Government Publishing Service, Canberra, 1994.

5. National Occupational Health and Safety Commission, *Approved Criteria for Classifying Hazardous Substances* [NOHSC:1008(1994)], Australian Government Publishing Service, Canberra, 1994.

4. FURTHER READING

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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.

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.

Paustenbach DJ, Bass RD and Price P, 'Benzene Toxicity and Risk Assessment, 1972-1992: Implications for Future Regulation', *Environmental Health Perspectives*, vol 101 (supplement 6), pp 177-200, 1993.

1. GUIDELINES* FOR HEALTH SURVEILLANCE FOR CADMIUM

BASELINE HEALTH SURVEILLANCE AT TIME OF EMPLOYMENT IN A CADMIUM PROCESS

1. Collection of Demographic Data

- Name and unique company identification number.
- Date of birth.
- Sex.
- Address.
- Date of starting company service.
- Descriptive job title. To include the Australian Bureau of Statistics' *Australian Standard Classification of Occupations* (ASCO)[1] and *Australian Standard Industrial Classification* (ASIC)[2].
- Places of previous employment.

2. Occupational History

- Past work history, including previous exposure to cadmium.
- Potential current exposure.
- Whether suitable personal protective equipment is used for that specific cadmium process.

3. Medical History

- Presence of symptoms.
- Smoking history with counselling on the additional cadmium burden from smoking.
- Administration of a standardised respiratory questionnaire. Two examples are the International Union Against Tuberculosis' *Bronchial Symptoms Questionnaire 1986*[3] **or** the Medical Research Council's *Questionnaire on Respiratory Symptoms 1986*[4].

4. Physical Examination

With emphasis on the respiratory system.

*These guidelines set out in a practical manner the minimum requirements for health surveillance and should be read in conjunction with the Introduction to the *Guidelines for Health Surveillance* [NOHSC:7039(1995)].

5. Investigation

- Standardised respiratory function tests* to be performed. The tests are FEV_1, FVC and FEV_1/FVC. The norms for predictive values should be stated.

- Spot creatinine corrected urine for cadmium to be conducted. Where there is 5 µg or more cadmium per gram creatinine, a repeat spot creatinine corrected urine for cadmium should be performed at the same time of the day.

6. Health Advice

The appointed medical practitioner should inform the employee of the potential health effects associated with exposure to cadmium.

DURING EXPOSURE TO A CADMIUM PROCESS

7. Personal Protective Equipment

The availability, type, fit, maintenance and frequency of use of personal protective equipment should be monitored regularly.

8. Urinary Cadmium

Baseline Level

Spot creatinine corrected urine for cadmium to be conducted annually. Where there is 5 µg or more cadmium per gram creatinine, a repeat spot creatinine corrected urine for cadmium should be performed at the same time of the day.

Action Level

On confirmation of a level of 5 µg cadmium or more per gram of creatinine, then a medical examination to include urinary ß$_2$-microglobulin should be performed.

The employer should be informed when abnormal findings are detected so that control measures can be checked. The employee should be informed of the results of the health surveillance.

Removal Level

Removal from cadmium work should be considered if the level is above 10 µg per gram of creatinine or ß$_2$-microglobulin exceeds 1,500 µg per gram creatinine. Spot creatinine corrected urine for cadmium should be repeated every 180 days until the level falls below 5 µg cadmium per gram creatinine and ß$_2$-microglobulin is less than 300 µg per gram creatinine.

Return to Work

The person must be medically fit to return to cadmium work.

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

9. Medical Examination

A medical examination every two years as described in points 3, 4 and 5 above, that is:

- medical history and reinforcement by counselling of the additional cadmium burden from tobacco smoking;
- physical examination; and
- respiratory function tests.

10. Data for Inclusion in Health Records

The following data should be included with the health records of the individual employee:

- Any formal assessments carried out in compliance with the National Occupational Health and Safety Commission's *National Model Regulations for the Control of Workplace Hazardous Substances* [NOHSC:1005(1994)][5].
- Descriptive job titles, with relevant start and finish dates. Those jobs within the area where cadmium is used should be clearly identified.
- Results of atmospheric and personal monitoring, and investigation of results that exceed the national exposure standard.

AT TERMINATION OF EMPLOYMENT IN A CADMIUM PROCESS

11. Data that should be Collected

- Date of termination.
- Reason for termination:
  - ill-health (if 'yes', give details),
  - other reasons, and
  - date and cause of death if in service.

12. Continuing Medical Surveillance

People with a history of raised $ß_2$-microglobulin should be advised to seek continuing medical surveillance.

2. SUPPLEMENTARY INFORMATION ON CADMIUM

WORK ACTIVITIES THAT MAY REPRESENT A HIGH RISK EXPOSURE

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

- processes such as 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;
- 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;
- cadmium batteries;
- manufacture and handling of paints and plastics containing cadmium pigments and the recycling of these plastics; and
- textile production.

Special attention should also be given to any acute exposures, including high temperature processes where cadmium fumes are evolved, that may occur in the above processes.

POTENTIAL HEALTH EFFECTS FOLLOWING EXPOSURE TO CADMIUM

Route of Entry into the Body

The primary route of cadmium entry into the body is through inhalation. Respiratory absorption of cadmium fumes ranges from 25-50 per cent. Only small cadmium particles are absorbed by the alveoli, and these small particles are typically found in fumes and cigarette smoke. The cadmium content of a cigarette is 2 µg and approximately 50 per cent is absorbed during active cigarette smoking.

There is some risk of ingestion if personal hygiene is inadequate. Gastrointestinal absorption ranges from 3-7 per cent, and absorption may be increased in the presence of calcium and iron deficiency. Percutaneous absorption is not significant. Cadmium does not pass the placenta.

Acute Effects

High inhalation exposure to cadmium oxide fume can cause respiratory irritation, pneumonitis and metal fume fever. Such exposure may be fatal. High ingestion exposure of soluble cadmium salts causes acute gastroenteritis.

Chronic Effects

Long-term occupational 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 such as ß$_2$-microglobulin. 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.

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[6].

There are reports[6] 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 such as zinc, copper and lead[6].

Results of studies[6] conducted in the United Kingdom, United States and Sweden on the incidence of lung cancer in cadmium-exposed workers provide consistent evidence that long-term occupational 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[6].

Carcinogen Classification

Cadmium chloride is listed in the National Commission's *List of Designated Hazardous Substances* [NOHSC:10005(1994)][7]and is classified as Carcinogen Category 2. According to the National Commission's *Approved Criteria for Classifying Hazardous Substances* [NOHSC:1008(1994)][8], a substance is assigned Carcinogen Category 2 if there is sufficient evidence, on the basis of appropriate long-term animal studies or other relevant information, to provide a strong presumption that human exposure to that substance may result in the development of cancer.

The International Agency for Research on Cancer and the Exposure Standards Expert Working Group have carcinogen ratings for other forms of cadmium and cadmium compounds.

The Exposure Standards Expert Working Group[9] classification for cadmium dusts and salts, cadmium oxide (fume) and cadmium oxide production is Carcinogen Category 2, probable human carcinogen. Probable human carcinogens are those substances for which there is sufficient evidence to provide a strong presumption that human exposure might result in the development of cancer. This evidence is generally based on appropriate long-term animal studies, limited epidemiological evidence or other relevant information.

The International Agency for Research on Cancer[6] 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.

3. REFERENCED DOCUMENTS

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2. Australian Bureau of Statistics, *Australian Standard Industrial Classification,* Australian Bureau of Statistics, Canberra, 1985.

3. Respiratory Disease Committee of the International Union Against Tuberculosis, *IUAT Bronchial Symptoms Questionnaire*, International Union Against Tuberculosis, 1986☎.

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☎Repoductions of these respiratory questionnaires can be purchased from Publications Sales at Worksafe Australia. Telephone (02) 577 9555.

4. FURTHER READING

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1. GUIDELINES* FOR HEALTH SURVEILLANCE FOR INORGANIC CHROMIUM

BASELINE HEALTH SURVEILLANCE AT TIME OF EMPLOYMENT IN AN INORGANIC CHROMIUM PROCESS

1. Collection of Demographic Data

- Name and unique company identification number.
- Date of birth.
- Sex.
- Address.
- Date of starting company service.
- Descriptive job title. To include the Australian Bureau of Statistics' *Australian Standard Classification of Occupations* (ASCO)[1] and *Australian Standard Industrial Classification* (ASIC)[2].
- Places of previous employment.

2. Occupational History

- Past work history, including previous exposure to inorganic chromium.
- Potential current exposure.
- Whether suitable personal protective equipment is used for that specific chromium process.

3. Medical History

- Presence of symptoms.
- Smoking history.

4. Physical Examination

With emphasis on the respiratory system and skin.

5. Health Advice

The appointed medical practitioner should inform the employee of the potential health effects associated with exposure to inorganic chromium.

* These guidelines set out in a practical manner the minimum requirements for health surveillance and should be read in conjunction with the Introduction to the *Guidelines for Health Surveillance* [NOHSC:7039(1995)].

DURING EXPOSURE TO AN INORGANIC CHROMIUM PROCESS

6. Personal Protective Equipment

The availability, type, fit, maintenance and frequency of use of personal protective equipment should be monitored regularly.

7. Workplace Skin Care Program

Participation in a workplace skin care program. Skin inspection of hands and forearms by a 'responsible person' should be conducted weekly, with referral to the appointed medical practitioner when required. The 'responsible person' would need adequate training as part of the inorganic chromium workplace skin care program.

8. Respiratory Symptoms

Respiratory symptoms should be reported to the appointed medical practitioner.

9. Data for Inclusion in Health Records

The following data should be included with the health records of the individual employee:

- Any formal assessments carried out in compliance with the National Occupational Health and Safety Commission's *National Model Regulations for the Control of Workplace Hazardous Substances* [NOHSC:1005(1994)][3].
- Descriptive job titles, with relevant start and finish dates. Those jobs where exposure has been assessed as significant should be clearly identified.
- Results of atmospheric and personal monitoring, and investigation of results that exceed the national exposure standard.

AT TERMINATION OF EMPLOYMENT IN AN INORGANIC CHROMIUM PROCESS

10. Data that should be Collected

- Date of termination.
- Reason for termination:
  - ill-health (if 'yes', give details),
  - other reasons, and
  - date and cause of death if in service.

2. SUPPLEMENTARY INFORMATION ON INORGANIC CHROMIUM

Chromium exists in a series of oxidation states from $^{-}2$ valence to $^{+}6$. The most important stable states are elemental metal (Cr^{0}), trivalent (Cr^{3+}) and hexavalent (Cr^{6+}).

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 and hard-facing of stainless steel;
- manual metal arc welding of high chromium steels;
- hard-plating;
- refractory production;
- leather tanning;
- timber preservation (copper chrome arsenic);
- chromate use in the textile industry; and
- chrome pigment use.

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.

Route of Entry into the Body

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

Acute and Chronic Effects

Hexavalent Chromium

Hexavalent chromium compounds on contact with skin, generally as liquids, mists or dusts, may act as both irritants and sensitisers. These also cause corrosive skin and mucous ulcerations, including chrome ulcers and perforation of the nasal septum.

At concentrations below those resulting in irritation, skin sensitivity is the most common effect following exposure to chromium compounds. Allergic dermatitis is well known in printers, cement workers, metal workers, painters, textile workers

and leather tanners[4]. Chromate sensitivity, once induced, may prove difficult to deal with in multiple settings and is very persistent once developed.

Inhaled chromium is a respiratory tract irritant, resulting in airway irritation and airway obstruction. Also, exposure by inhalation can cause allergic asthmatic reactions. A single inhalation exposure to highly water-soluble compounds can result in irritation and inflammation of the respiratory tract.

Studies of welders and chromium platers have shown that 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 that 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.

Carcinogenicity

There is considerable epidemiological evidence that 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[5-9]. The International Agency for Research on Cancer's[7] 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.

While metallic chromium and trivalent compounds have an International Agency for Research on Cancer classification of Group 3, there are some mutagenicity tests and an epidemiological study of chrome platers pointing to some carcinogenic potential of soluble chromates, 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[10].

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

Carcinogen Classification

Various hexavalent chromium compounds are listed in the National Commission's *List of Designated Hazardous Substances* [NOHSC:10005(1994)][11]. Chromium-, calcium-, and strontium- chromate are classified as Carcinogen Category 2. Zinc chromate is also listed and is classified as Carcinogen Category 1. According to the National Commission's *Approved Criteria for Classifying Hazardous Substances* [NOHSC:1008(1994)][12], a substance is assigned Carcinogen Category 1 if there is sufficient evidence to establish a causal relationship between human exposure and the development of cancer on the basis of epidemiological data, and Carcinogen Category 2 if there is sufficient evidence, on the basis of appropriate long-term animal studies or other relevant information, to provide a strong presumption that human exposure to that substance may result in the development of cancer.

3. REFERENCED DOCUMENTS

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2. Australian Bureau of Statistics, *Australian Standard Industrial Classification*, Australian Bureau of Statistics, Canberra, 1985.

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1. GUIDELINES* FOR HEALTH SURVEILLANCE FOR CREOSOTE

BASELINE HEALTH SURVEILLANCE AT TIME OF EMPLOYMENT IN A CREOSOTE PROCESS

1. Collection of Demographic Data

- Name and unique company identification number.
- Date of birth.
- Sex.
- Address.
- Date of starting company service.
- Descriptive job title. To include the Australian Bureau of Statistics' *Australian Standard Classification of Occupations* (ASCO)[1] and *Australian Standard Industrial Classification* (ASIC)[2].
- Places of previous employment.

2. Occupational History

- Past work history, including previous exposure to creosote.
- Potential current exposure.
- Whether suitable personal protective equipment is used for that specific creosote process.

3. Medical History

- Presence of symptoms.
- Smoking history.

4. Physical Examination

- With emphasis on the neurological system.
- A thorough examination of all skin areas, 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.

* These guidelines set out in a practical manner the minimum requirements for health surveillance and should be read in conjunction with the Introduction to the *Guidelines for Health Surveillance* [NOHSC:7039(1995)].

5. Health Advice

The appointed medical practitioner should inform the employee of the potential health effects associated with exposure to creosote. In particular, employees should be aware of the occurrence and recognition of photosensitivity and skin changes, and the need to report them as soon as possible to the appointed medical practitioner, even if they occur between regular surveillance.

DURING EXPOSURE TO A CREOSOTE PROCESS

6. Personal Protective Equipment

The availability, type, fit, maintenance and frequency of use of personal protective equipment should be monitored regularly.

7. Photosensitivity

Employees should be reminded to notify the appointed medical practitioner if photosensitivity occurs between regular surveillance. Employees reporting photosensitivity should receive additional counselling on the potential health effects of creosote on the skin.

8. Physical Examination

A physical examination as described in point 4 above, to be conducted annually. Evidence of skin sensitisation should also be noted.

The employer should be informed when abnormal findings are detected so that control measures can be checked. The employee should be informed of the results of the health surveillance.

9. Data for Inclusion in Health Records

The following data should be included with the health records of the individual employee:

- Any formal assessments carried out in compliance with the National Occupational Health and Safety Commission's *National Model Regulations for the Control of Workplace Hazardous Substances* [NOHSC:1005(1994)][3].
- Descriptive job titles, with relevant start and finish dates. Those jobs where exposure has been assessed as significant should be clearly identified.
- Record and review of any photosensitivity which a worker has had, indicating specific processes involved.

AT TERMINATION OF EMPLOYMENT IN A CREOSOTE PROCESS

10. Data that should be Collected

- Date of termination.
- Reason for termination:
  - ill-health (if 'yes', give details),
  - other reasons, and
  - date and cause of death if in service.

11. Final Physical Examination

A physical examination as described in point 4 above.

12. Continuing Medical Surveillance

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

2. SUPPLEMENTARY INFORMATION ON CREOSOTE

WORK ACTIVITIES THAT MAY REPRESENT A HIGH RISK EXPOSURE

In Australia, creosote is produced by distilling coal tar derived as a by-product of metallurgical coke ovens. Technically, creosote is a low-boiling distillate of coal tar. The chemical constituents of creosote are numerous. It is estimated that a complex mixture of 1,000 compounds may be present, mostly in trace amounts, many being of the aromatic series.

The major use of creosote is as a timber preservative against fungi, termites and marine borers. Timber preservation is an example of a work activity which requires special attention when assessing exposure.

POTENTIAL HEALTH EFFECTS FOLLOWING EXPOSURE TO CREOSOTE

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.

Photosensivity

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 sex of the person exposed[4].

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[4].

Acute Effects

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 and/or vascular collapse.

- Neurological:
    - headaches, fainting, vertigo and mental disturbances.

Chronic Exposure

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

Carcinogenicity

Repeated contact over long periods of time may be responsible for the development of cutaneous neoplasms. Skin cancer has been produced in experimental animals. There are no validated published epidemiological studies showing whether workers exposed to creosote have suffered from an increased risk of skin cancer, although there are number of case reports[5].

3. REFERENCED DOCUMENTS

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1. GUIDELINES* FOR HEALTH SURVEILLANCE FOR ISOCYANATES

BASELINE HEALTH SURVEILLANCE AT TIME OF EMPLOYMENT IN AN ISOCYANATE PROCESS

Current evidence suggests that a history of atopy or asthma does not preclude working with isocyanates. However, exposure to isocyanates likely to cause respiratory irritation may aggravate pre-existing asthma.

1. Collection of Demographic Data

- Name and unique company identification number.
- Date of birth.
- Sex.
- Address.
- Date of starting company service.
- Descriptive job title. To include the Australian Bureau of Statistics' *Australian Standard Classification of Occupations* (ASCO)[1] and *Australian Standard Industrial Classification* (ASIC)[2].
- Places of previous employment.

2. Occupational History

- Past work history, including previous exposure to isocyanates.
- Potential current exposure.
- Whether suitable personal protective equipment is used for that specific isocyanate process.

3. Medical History

- Presence of symptoms.
- Smoking history.
- Administration of a standardised respiratory questionnaire. Two examples are the International Union Against Tuberculosis' *Bronchial Symptoms Questionnaire 1986*[3] **or** the Medical Research Council's *Questionnaire on Respiratory Symptoms 1986*[4].

4. Physical Examination

With emphasis on the respiratory system and skin.

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*These guidelines set out in a practical manner the minimum requirements for health surveillance and should be read in conjunction with the Introduction to the *Guidelines for Health Surveillance* [NOHSC:7039(1995)].

5. Investigation

Standardised respiratory function tests* to be performed. The tests are FEV_1, FVC and FEV_1/FVC. The norms for predicted values should be stated.

6. Health Advice

The appointed medical practitioner should inform the employee of the potential health effects associated with exposure to isocyanates.

DURING EXPOSURE TO AN ISOCYANATE PROCESS

7. Personal Protective Equipment

The availability, type, fit, maintenance and frequency of use of personal protective equipment should be monitored regularly.

8. Medical Examination

A medical examination as described in points 3, 4 and 5 above at two weeks, six weeks and then at six monthly intervals during continued exposure, that is:

- medical history;
- physical examination for occupational dermatitis; and
- standardised respiratory function tests to be performed 8-16 hours after exposure.

Where practicable, pre- and post-shift standardised respiratory function tests should be performed. If only one test is performed, it should be done post-shift.

The employer should be informed when any abnormal findings are detected so that control measures can be checked. The employee should be informed of the results of the health surveillance.

AT TERMINATION OF EMPLOYMENT IN AN ISOCYANATE PROCESS

9. Data that should be Collected

- Date of termination.
- Reason for termination:
  - ill-health (if 'yes', give details),
  - other reasons, and
  - date and cause of death if in service.

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

10. Health Advice

Those workers sensitised to isocyanates should be strongly advised against further exposure.

2. SUPPLEMENTARY INFORMATION ON ISOCYANATES

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; and
- processes where heat decomposition of polyurethane products occurs, such as welding, heat removal of electrical insulating varnishes and hot wire cutting of foam.

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

POTENTIAL HEALTH EFFECTS FOLLOWING EXPOSURE TO ISOCYANATES

Route of Entry into the Body

The route of isocyanate entry into the body is through inhalation and percutaneous absorption.

Acute and Chronic Effects

Health effects may follow acute or chronic exposure to isocyanates. Isocyanates can cause respiratory sensitisation and lead to occupational asthma. Isocyanate splashes in the eyes can cause severe chemical conjunctivitis. Isocyanates are also mild skin irritants and can cause dermatitis. Sensitisation of the skin may occur, but this is not common. 4,4'Di-isocyanate dicyclohexyl methane is an exception, being a potent skin sensitiser.

In sufficiently high concentrations in the air, isocyanates have a primary irritant effect on the respiratory tract.

Sensitised workers may exhibit asthmatic symptoms when subsequently exposed to atmospheric concentrations well below the exposure standard. Exposure of sensitised workers may initiate reduction in respiratory capacity immediately on exposure, some hours later or both. There is evidence that for sensitised workers, recurrent exposures may result in impairment of ventilatory function and poor recovery.

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

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☎ Reproductions of these respiratory questionnaires can be purchased from Publications Sales at
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1. GUIDELINES* FOR HEALTH SURVEILLANCE FOR INORGANIC MERCURY

BASELINE HEALTH SURVEILLANCE AT TIME OF EMPLOYMENT IN AN INORGANIC MERCURY PROCESS

1. Collection of Demographic Data

- Name and unique company identification number.
- Date of birth.
- Sex.
- Address.
- Date of starting company service.
- Descriptive job title. To include the Australian Bureau of Statistics' *Australian Standard Classification of Occupations* (ASCO)[1] and *Australian Standard Industrial Classification* (ASIC)[2].
- Places of previous employment.

2. Occupational History

- Past work history, including previous exposure to inorganic mercury.
- Potential current exposure.
- Whether suitable personal protective equipment is used for that specific mercury process.

3. Medical History

- Presence of symptoms.
- Smoking history.

4. Physical Examination

With emphasis on neurological, gastrointestinal and renal systems and skin.

5. Investigation

Spot creatinine corrected urine for inorganic mercury to be conducted. Where there is 50 µg inorganic mercury or more per gram creatinine, a repeat spot creatinine corrected urine for inorganic mercury should be performed at the same time of the day.

*These guidelines set out in a practical manner the minimum requirements for health surveillance and should be read in conjunction with the Introduction to the *Guidelines for Health Surveillance* [NOHSC:7039(1995)].

6. Health Advice

The appointed medical practitioner should inform the employee of the potential health effects associated with exposure to inorganic mercury.

DURING EXPOSURE TO AN INORGANIC MERCURY PROCESS

7. Personal Protective Equipment

The availability, type, fit, maintenance and frequency of use of personal protective equipment should be monitored regularly.

8. Urinary Inorganic Mercury

Baseline Level

Spot creatinine corrected urine for inorganic mercury to be conducted every 90 days. Where there is 50 µg inorganic mercury or more per gram of creatinine, a repeat spot creatinine corrected urine for inorganic mercury should be performed at the same time of the day.

Action Level

On confirmation of a level of 50 µg inorganic mercury or more per gram of creatinine, a medical examination with emphasis on the neurological, gastrointestinal, renal and dermatological systems should be performed.

The employer should be informed when abnormal findings are detected so that control measures can be checked. The employee should be informed of the results of the health surveillance.

Removal Level

Removal from mercury work should be considered if clinical signs of mercury poisoning are present or if the level of inorganic mercury in urine is greater than 100 µg per gram of creatinine.

The person should be investigated every 30 days until the level falls below 50 µg inorganic mercury per gram of creatinine on two successive occasions. The test may be performed more frequently in individual circumstances.

The 90 day protocol may then be resumed.

Return to Work

The person must be medically fit to return to mercury work.

AT TERMINATION OF EMPLOYMENT IN AN INORGANIC MERCURY PROCESS

9. Data that should be Collected:

- Date of termination.
- Reason for termination:
  - ill-health (if 'yes', give details),
  - other reasons, and
  - date and cause of death if in service.

10. Final Physical Examination

A physical examination to determine persons with neurological or renal dysfunction due to mercury.

2. SUPPLEMENTARY INFORMATION ON INORGANIC MERCURY

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; and
- the use of mercury-containing fungicides.

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

POTENTIAL HEALTH EFFECTS FOLLOWING EXPOSURE TO INORGANIC MERCURY

Route of Entry into the Body

Inorganic mercury's absorption and potential toxicity depends on its chemical and physical form.

Generally, liquid elemental mercury (Hg^0) is poorly absorbed through the intestinal tract (less than 1 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.

Mercurous (Hg^1) and mercuric (Hg^2) salts are little absorbed (< 10 per cent) following ingestion. Percutaneous absorption of ionic mercury salts can cause toxicity. Generally, mercuric salts are more soluble and more toxic than mercurous salts.

Acute Effects

In acute poisoning, the respiratory system is affected by inhaled mercury vapour, and the gastrointestinal system 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 pnuemonitis. The uptake of mercury vapour into the central nervous system produces tremor and increased excitability.

Acute mercurial poisoning is usually the outcome of accidental or deliberate ingestion. The acute lethal dose of most mercury salts is 1-4 grams. The gastrointestinal tract and later the kidney are affected by ingestion of mercury salts.

Chronic Effects

The primary organ system affected by chronic exposure to elemental mercury is the nervous system, and the kidney is the primary organ affected by chronic exposure to mercury salts.

In chronic poisoning resulting from exposure to elemental mercury or the dust of inorganic mercurial compounds, early symptoms may include nausea, frequent headaches, tiredness and chronic diarrhoea. The characteristic features are stomatitis, muscular tremors and psychotic disturbances. Effects on the mouth may vary from a mere metallic taste to excessive salivation, bleeding of the gums, ulceration and loosening of the teeth. Muscular tremors appear early, often starting in the fingers and spreading to the tongue, lips, eyes and lower limbs. These become apparent when the individual goes to perform some defined action, such as writing, which may become so disordered by the tremor that it is illegible. The psychic 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, such as hallucination, or intellectual deterioration.

Kidney dysfunction sometimes develops, especially in workers exposed to elemental mercury. Its development 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.

The soluble inorganic mercury salts, for example, 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.

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1. GUIDELINES* ✞ FOR HEALTH SURVEILLANCE FOR 4,4' - METHYLENE BIS (2 - CHLOROANILINE) [MOCA]

BASELINE HEALTH SURVEILLANCE AT TIME OF EMPLOYMENT IN A MOCA PROCESS

1. Collection of Demographic Data

- Name and unique company identification number.
- Date of birth.
- Sex.
- Address.
- Date of starting company service.
- Descriptive job titles. To include the Australian Bureau of Statistics' *Australian Standard Classification of Occupations* (ASCO)[1] and *Australian Standard Industrial Classification* (ASIC)[2].
- Places of previous employment.

2. Occupational History

- Past work history, including previous exposure to MOCA and other chemicals that may cause bladder cancer.
- Potential current exposure.
- Whether suitable personal protective equipment is used for that specific MOCA process.

3. Medical History

- Smoking history.
- Presence of symptoms, including history of bladder neoplasms.

4. Investigation

- Dipstick urinalysis for haematuria.
- Urine cytology.

5. Health Advice

The appointed medical practitioner should inform the employee of the
potential health effects associated with exposure to MOCA.

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*These guidelines set out in a practical manner the minimum requirements for health surveillance and should be read in conjunction with the Introduction to the *Guidelines for Health Surveillance* [NOHSC:7039(1995)].

✞These guidelines may be revised when MOCA adduct testing becomes an established detection method.

DURING EXPOSURE TO A MOCA PROCESS

6. Personal Protective Equipment

The availability, type, fit, maintenance and frequency of use of personal protective equipment should be monitored regularly.

7. Urinary Analysis and Urine Cytology

Urinary total MOCA estimation and dipstick urinalysis for haematuria at least twice a year with sampling to be at a time of peak use/exposure.

Urine cytology at yearly intervals.

The employer should be informed when abnormal findings are detected so that control measures can be checked. The employee should be informed of the results of the health surveillance.

AT TERMINATION OF EMPLOYMENT IN A MOCA PROCESS

8. Data that should be Collected

- Date of termination.
- Reason for termination:
  - ill-health (if 'yes', give details),
  - other reasons, and
  - date and cause of death if in service.

9. Final Medical Examination

Urine cytology and dipstick urinalysis should be performed at termination of employment with a medical review of all the records.

10. Continuing Medical Surveillance

The appointed medical practitioner should inform both the employer and employee of the need for continuing urine cytology and dipstick urinalysis. Where practicable, the employer should provide this service and remind employees of its availability.

2. SUPPLEMENTARY INFORMATION ON MOCA

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 such as polyurethane gears, gaskets, belts, rollers, sport boots and roller skate wheels.

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; and
- manual moulding of semi-set polyurethane products.

POTENTIAL HEALTH EFFECTS FOLLOWING EXPOSURE TO MOCA

Route of Entry into the Body

The primary route of MOCA entry into the body is through percutaneous absorption.

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[3,4,5]. More recently, 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[6]. This is the only report of humans with a history of MOCA exposure developing bladder cancer. Based on the animal data and recent human data, MOCA should be regarded as a potential human carcinogen.

Carcinogen Classification

MOCA is listed in the National Occupational Health and Safety Commission's *List of Designated Hazardous Substances* [NOHSC:10005(1994)][7] and is classified as Carcinogen Category 2. According to the National Commission's *Approved Criteria for Classifying Hazardous Substances* [NOHSC:1008(1994)][8], a substance is assigned Carcinogen Category 2 if there is sufficient evidence, on the basis of appropriate long-term animal studies or other relevant information, to provide a strong presumption that human exposure to that substance may result in the development of cancer.

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INTRODUCTION TO THE GUIDELINES FOR HEALTH SURVEILLANCE FOR ORGANOPHOSPHATE PESTICIDES REVISED EDITION 1998

Organophosphate pesticides are listed in Schedule 3 (Substances for which health surveillance is required) of the *National Model Regulations for the Control of Workplace Hazardous Substances* [NOHSC:1005 (1994)][1]. Regulation 14(1)(a) of the Model Regulations requires that health surveillance be carried out when it has been identified from the workplace assessment that there exists a significant risk to health from exposure to organophosphate pesticides. These Guidelines for Health Surveillance for Organophosphate Pesticides set out in a practical manner the minimum requirements for health surveillance for organophosphate pesticides and should be read in conjunction with the Introduction to *Guidelines for Health Surveillance* [NOHSC:7039 (1995)][2].

The Guidelines are divided into two sections.

Section 1 **Page 2**

includes:

- minimum requirements for health surveillance for exposed workers
- specific examinations types and biological monitoring, and
- criteria for removal and return to organophosphate pesticide work.

Section 2 **Page 5**

includes:

- chemistry
- mechanism of action and toxicity in humans
- rationale that supports the recommended biological monitoring technique
- assessment process guidance
- pattern of exposure characterisation.

Note: appointed medical practitioner(s) should be familiar with Table 1 'Definitions of Patterns of Use' before implementing a health surveillance programme for organophosphate pesticides.

Appendix 1 **Page 16**

includes:

- details for the lay person or nurse
- first aid measures in the event of organophosphate pesticide poisoning
- advice on how to maintain essential body functions until medical assistance arrives or advice on how to prevent further contamination of the affected person
- details for the physician for the treatment of acute poisoning cases, including specific antidotes and symptomatic therapy.

Appendix 2 **Page 20**

lists:

- organophosphate actives currently registered for use in Australia.

SECTION 1. GUIDELINES* FOR HEALTH SURVEILLANCE FOR ORGANOPHOSPHATE PESTICIDES

BASELINE HEALTH SURVEILLANCE

BEFORE EXPOSURE TO ORGANOPHOSPHATE PESTICIDES

1.1 Collection of Demographic Data

- Name and unique company identification number.
- Date of birth.
- Sex.
- Address.
- Date of starting company service.
- Descriptive job title. To include the Australian Bureau of Statistics' Australian Standard Classification of Occupations (ASCO)[3] and Australian Standard Industrial Classification (ASIC)[4].
- Places of previous employment.

1.2 Occupational History

- Past work history, including previous exposure to organophosphate and carbamate pesticides.
- Potential current exposure.
- Whether suitable personal protective equipment is used for that specific organophosphate pesticide process.

1.3 Medical History

- Presence of symptoms.
- Smoking history.

1.4 Physical Examination

- Conducted only if indicated by occupational and medical history.

1.5 Investigation

- 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 10 per cent to be regarded as reliable.

*These guidelines set out in a practical manner the minimum requirements for health surveillance and should be read in conjunction with the Introduction to the *Guidelines for Health Surveillance* [NOHSC:7039(1995)].

- If the worker has had previous exposure, then it is desirable that a period of four weeks of no exposure should occur before the base-line level is established.

1.6 Health Advice

- The appointed medical practitioner should advise the employee of the potential health effects associated with exposure to organophosphate pesticides.

DURING EXPOSURE TO ORGANOPHOSPHATE PESTICIDES

1.7 Personal Protective Equipment

- The availability, type, fit, maintenance and frequency of use of personal protective equipment should be monitored regularly.

1.8 Medical Examination

- Workers should be examined by the appointed medical practitioner in the circumstances where:

  - Excessive exposure to organophosphate pesticides is suspected;
  - Workers are concerned; or
  - Symptoms suggestive of organophosphate poisoning are present.

- Additionally, periodic testing of a sample of workers during the period of use of organophosphate pesticides is desirable.

- The medical examination repeats points 1.2, 1.3, 1,4 and 1.5 above, that is:

  - Occupational History.
  - Medical History.
  - Physical Examination. Evidence of dermatitis on the hands and forearms may indicate advice is required on work practices.
  - Estimation of red cell and plasma cholinesterase activity levels by the Ellman method. It is preferable that the estimation be done in the latter half of the working day when organophosphate pesticides are used. If a 20% depression of cholinesterase activity is seen the worker should be re-tested.

1.9 Removal from Organophosphate Pesticide Exposure

- If there is a fall in cholinesterase activity by 40% or more the worker should be removed from further exposure to the organophosphate pesticides until such time as the level returns to baseline levels.
- The person can be moved to another area or can use other classes of pesticides (except pyrethroids eg. Permethrin; and carbamates).

1.10 Information to be Conveyed to the Employer and Employee

- The employer should be informed when abnormal findings are detected so that control measures can be checked. The employee should be informed of the results of the health surveillance.

AT TERMINATION OF EXPOSURE TO ORGANOPHOSPHATE PESTICIDES

1.11 Data That Should be Collected

- Date of termination.
- Reason for termination of employment:
  - Ill-health (if yes give details),
  - Other reasons
  - Date and cause of death if in service.

SECTION 2. SUPPLEMENTARY INFORMATION ON ORGANOPHOSPHATE PESTICIDES

2.1 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.

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

- pest control operators who use OP everyday in their work;
- manufacture and packaging;
- transport, storage and distribution;
- handling used containers, for example, scrap recovery;
- agricultural and horticultural activities, such as mixing, loading and application where direct handling of the chemical occurs (***see*** Table 1 – 'Definitions of Patterns of Use');
- veterinary activities such as cattle and sheep dipping occurs (***see*** Table 1 – 'Definitions of Patterns of Use');
- seasonal field workers exposed to pesticide residues occurs (***see*** Table 1 – 'Definitions of Patterns of Use'); and
- laboratory workers undertaking research on organophosphate pesticides.

2.2 WORKPLACE ASSESSMENT

Because of the seasonal nature of the use of organophosphate pesticides in the rural sector, special attention needs to be paid to the assessment process. A written assessment is evidence of the employer's intention to have a safe and healthy workplace. The assessment may be a written plan of action for the circumstances which apply to that particular employer. For example, a list of the likely operations for which organophosphate pesticides may be used by the employer or employees could be developed with some estimate of length of time of use for a particular operation. Once this assessment is completed then the need for health surveillance in relation to each operation, noting the 'Definitions of Patterns of Use' (***see*** Table 1), should be considered. Any proposed health surveillance should be recorded.

In any event the annual proposed types of use should be set out, and all use should be recorded.

Special attention should also be given to acute exposures, including spills, that may occur in any of the above processes. In the case of organophosphate poisoning, see Appendix 1 for first aid instructions and specific medical treatment.

2.3 POTENTIAL HEALTH EFFECTS FOLLOWING EXPOSURE TO ORGANOPHOSPHATE PESTICIDES

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. All 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 pesticides, ACHE is phosphorylated and is no longer able to break down acetylcholine into choline and acetic acid. The resulting accumulation 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 compounds[5].

2.4 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 any further metabolic modification after absorption into the body[5]. Indirect inhibitors need to be transformed in the body to be effective[5]. All thiono OP pesticides, that is, those containg 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[5]. The insecticide dichlorvos is an example of a direct inhibitor while malathion and parathion are indirect inhibitors[5].

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[6].

Organophosphorous compounds also inhibit tissue carboxyesterases. Although this does not result in any direct toxicity it may increase the toxicity of other pesticides such as most pyrethroids that are detoxified by carboxyesterases.

Route of Entry into the Body

For most organophosphate pesticides, dermal exposure and subsequent absorption through intact skin represents the most important route of entry in case of occupational exposure[5].

The oral route of entry is important in accidental ingestion and deliberate ingestion. Occupational accidental ingestion may occur as a result of poor work practices and lack of personal hygiene[5].

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[5].

Organophosphate pesticides are also absorbed through mucous membranes and eyes.
[1]

Acute Toxicity[1]

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).

[1] Adapted from Ref 7.

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 1 to 6 days. In the case of massive exposure death usually occurs within 24 hours.

Occupational poisoning generally occurs from skin contamination. 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. Also, if the concentrate of any of the more toxic OP is splashed into the eye, absorption may be very rapid. If swallowed, OP are rapidly absorbed from the stomach.

Symptoms of poisoning usually do not occur until enzyme activity has been reduced to between 60 to 25% 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 1% per day.

Chronic Toxicity[1]

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 apperars 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[7].

Many organophosphate pesticides cause primary irritant dermatitis; only a few are known to cause allergic contact dermatitis (eg. parathion and malathion).

TABLE 1 DEFINITION OF PATTERN USE AND ACTION REQUIRED

DEFINITION OF PATTERN OF USE	ACTION REQUIRED
Baseline	If a person uses organophosphate pesticides they should have a baseline measurement done – two are desirable, at a time when there has been at least 4 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 person has symptoms which could be related to organophosphate pesticides during or after use, or there has been an 'exposure incident' leading to symptoms, or the person using the organophosphate pesticide is concerned.
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. A test during a period of use would be useful in order to check work practices. This provides valuable feedback on the effectiveness of controls, PPE etc. Also meets the responsibility of the employer. No further test is needed unless the person has symptoms which could be related to organophosphate pesticides during or after use, or there has been an 'exposure incident' leading to symptoms, or there is concern that 'overexposure' may have occurred.
Seasonal Use If use of organophosphate pesticides is say 4 days a week, and extends over a long season then, this is *seasonal use.*	Use should be recorded. If heavy use, all workers so exposed, should be tested, 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. All workers should be advised of their results (% depression of cholinesterase from their baseline values) and the need to maintain or improve work practices emphasised. The timing of further tests, for individuals or the whole work group, should be based on the nature of the work and the previous test results. Any 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, or there has been an 'exposure incident' leading to symptoms, or there is concern that '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, temulousness, 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.

Source: Ref 8.

2.5 BIOMARKER OF EFFECT

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[5]. Increased values may be found in polycythaemia and in thalassaemia or other congenital blood dyscrasias[5]. Low values of erythrocyte cholinesterase not related to OP exposure have been observed in subjects affected with leukaemias or other neoplasms[5].

Serum Cholinesterase

Serum cholinesterase is synthesised in the liver. Serum cholinesterase shows normal values 10 -15% greater in males than in females[5]. 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)[5,6]. In females lower values are also measured during pregnancy and menstruation[5]. Serum cholinesterase activity can be increased in genetic variants and sometimes when the patient has obesity , hypertension, psoriasis, thyrotoxicosis or asthma[6].

Measurement of blood cholinesterase activity is an accepted method for biological effect monitoring of worker exposure to organophosphate pesticides[6]. This approach measures the common effect of this class of pesticide on certain enzyme activities. This is in contrast to the approach with organochlorine pesticides where the actual level of the insecticide is measured in blood. The number of organophosphorous pesticides in current use is very large (see Appendix 2) and it is unlikely that it would be viable to directly analyse more than a few of the most widely used pesticides.

Baseline Levels of Serum and Erythrocyte Cholinesterase Activiry[2]

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% 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 that the training technician has not used this class of pesticides during instruction.

[2] Reproduced with kind permisson from Dr Wooller[6]

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 occupational 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 occupational exposure if attention is only focused on whether or not the cholinesterase result lies within the reference range. It is recommended that 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 that about 3% of individuals will have this deficiency to some degree and hence will have lower than average cholinesterase activities. It does not appear that such individuals are more at risk than those that do not have this deficiency. Thus they can be permitted to commence using anticholinesterase pesticides. This deficiency should be confirmed by measuring either the dibucaine of fluoride numbers which bear a relationship to the serum cholinesterase genotype.

It is inappropriate to remove a person from further occupational exposure if the cholinesterase activity reflects a genetic deficiency rather than current occupational exposure. However, if a baseline cholinesterase activity has not been established in such individuals then such workers may be removed from further exposure and retested a number of times before it becomes apparent that the worker is probably genetically deficient rather than occupationally exposed. At this time the dicubaine of fluoride numbers should be determined to confmn 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.

2.6 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 in order 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 to a laboratory equipped for cholinesterase determinations without delay. 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 5 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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APPENDIX 1

ORGANOPHOSPHOROUS CHOLINESTERASE-INHIBITING PESTICIDE POISONING[2]

FIRST AID

Where poisoning occurs, the person on the spot can often do a great deal to minimise the effect of the pesticide. The following general principles of treatment should be followed.

1. **Summon expert help:**

   EITHER Send one person to summon the nearest doctor or ambulance

   OR Take the victim to the nearest hospital or doctor. If the casualty is unconscious he/she must be transported in a coma position.

2. Remove any contaminated clothing and wash skin thoroughly.

3. Where the eyes have been splashed, open the eyes and flush with copious quantities of running water for at least 15 minutes.

4. Establish exactly what has happened, so that the doctor or hospital will have a clear idea of which poisons have been involved. Save any containers/labels/material safety data sheets (MSDS). Save any vomit for analysis.

5. It may be appropriate to induce vomiting if a poison has been swallowed. The instructions on the pesticide label must be strictly followed in this regard. NEVER induce vomiting in a semi-conscious or unconscious person.

6. If the victim is unconscious clear the mouth of false teeth, vomitus, etc, then tilt the head back and bring the jaw forward to ensure a clear airway.

7. Check the breathing. If the victim is unconscious but breathing, turn victim onto side.

8. If the victim is unconscious and not breathing, roll the victim onto their back and commence expired air resuscitation. Give 5 full ventilations in ten seconds, then check pulse. If pulse present continue expired air resuscitation at the rate of 15 per minute. If pulse absent, begin external cardiac massage. If one operator, emergency heart-lung resuscitation should consist of 2 ventilations and 15 compressions repeated 4 times per minute. If two operators, emergency heart-lung resuscitation should consist of 1 ventilation and 5 compressions repeated 12 times per minute.

9. If convulsions or fits occur, ensure that the airway is kept clear. A gag, if available, may be put between the teeth to protect the tongue. Whilst the above first aid measures are being carried out, the victim may be transported to medical aid.

[2] Reproduced with kind permission from Dr Wooller[(6)]

GENERAL MEDICAL TREATMENT

Initial non-specific treatment will be directed towards

- preventing further absorption by decontamination of the skin and by gastric lavage
- resuscitation with assisted respiration, administration of oxygen, attention to electrolyte balance
- control of convulsions by the use of diazepam, etc
- symptomatic treatment

The appointed medical practitioner, through local knowledge, may be able to anticipate the type of pesticides in use. However, it is important that accurate details of the particular pesticide involved be obtained as specific treatment depends on this. Where the nature of pesticide exposure is known

- administration of specific antidotes (eg pralidoxime, atropine)
- transfer to base hospital or intensive care unit where necessary .

Checklist of Symptoms and Signs for organophosphorous cholinesterase-inhibiting insecticide poisoning

Dizziness
Headache
Salivation
Nausea and Vomiting
Wheezing
Dysarthria
Ataxia
Muscle fasciculation with/without cyanosis
Respiratory failure
Convulsions
Coma
Loss of pupillary reflex

Poisoning by organophosphorous pesticides occurs usually by skin contact in occupationally exposed persons, by inhalation or by ingestion (usually suicide attempt). The rapidity of onset symptoms depends on the route and magnitude of exposure, previous recent exposure and the insecticide involved.

Antidotes

If the patient is anoxic, this must be treated prior to the administration of atropine.

Atropine antagonises the muscarinic effects of the accumulating acetylcholine and so controls many of the symptoms.

Dosage (adult) is 1 to 5 mg given intravenously, intramuscularly or subcutaneously, repeated every 10 to 30 minutes until atropisation is achieved, that is:

- dilated pupils
- flushing
- drymouth
- bronchodilation
- increased pulse rate (80 to 140 per minute)

Moisture at bases of lungs, nausea, miosis and bradycardia indicate inadequate atropinisation.

Fever, muscle fibrillations and delirium are signs of atropine toxicity.

Maintain atropinisation up to 24 hours then review. Note that more than 100 mg atropine may occasionally be required. Note also that there may be adipose tissue storage of insecticide with later release causing a recurrence of symptoms. Thus caution must be exercised when withdrawing atropine.

Pralidoxine reverses the inhibition of cholinesterases by many, but not all, organophosphorous pesticides if given as soon as possible (within 36 hours) after exposure has occurred. It also relieves the nicotinic effects of accumulated acetylcholine.

Dosage (adult) is 1 g given slowly intravenously or intramuscularly. May be repeated in 1 to 2 hours then 12 hourly as needed.

If ingestion has occurred empty stomach using IPECAC, if alert, or by intubation if not. Save sample of emesis or initial gastric washings for chemical analysis. Instil 50 g activated charcoal.

If convulsions occur, diazepam 5 to 10 mg (adult) may be used.

The following medications are contraindicated: morphine, aminophylline, frusemide, ethacrynic acid, reserpine.

Summary of Management

1. Maintain oxygenation (by mechanical means, if necessary).
2. If dermal exposure, decontaminate the skin.
3. If ingested, induce vomiting.
4. Give atropine sulphate. Obtain venous blood sample for plasma and erythrocyte cholinesterase estimation.
5. Give pralidoxime.
6. Give diazepam if convulsions occur.
7. Repeat antidotes as necessary.
8. Repeat cholinesterase estimations as necessary to monitor recovery .

As a guide plasma cholinesterase may regenerate 25% in 7 to 10 days while erythrocyte cholinesterase activity will increase much more slowly at a rate of ~1% day (as new erythrocytes are released from the bone marrow).

Occupational exposure should not occur until the erythrocyte cholinesterase level returns to at least 80% of the pre-exposure level, or if this information is not available, then to within the reference range. In severe poisoning this may take several months.

APPENDIX 2

LIST OF ORGANOPHOSPHATE PESTICIDES CURRENTLY REGISTERED BY THE NATIONAL REGISTRATION AUTHORITY[3] FOR USE IN AUSTRALIA

NAME OF ACTIVE INGREDIENT	MAIN USE
Acephate	Insecticide
Azamethiphos	Insecticide
Azinphos-ethyl	Insecticide
Azinphos-methyl	Insecticide
Bensulide	Herbicide
Bromophos	Insecticide
Cadusafos	Nematicide Insecticide
Chlorfenvinphos	Insecticide
Chlormephos	Insecticide
Chlorpyrifos	Insecticide
Chlorthiophos	Insecticide
Coumaphos	Insecticide
Crotoxyphos	Insecticide
Demeton-O	Insecticide
Demeton-S	Insecticide
Diazinon	Insecticide
Dichlorvos	Insecticide
Dicrotophos	Insecticide
Dimefox	Insecticide
Dimethoate	Insecticide
Disulfoton	Insecticide
Dithiopyr	Herbicide
Edifenphos	Fungicide
EPN (ESA name)	Insecticide
Ethion	Acaricide Insecticide
Ethoprophus	Insecticide applied to soil
Famphur	Insecticide
Fenamiphos	Nematocide
Fenchlorphos	Insecticide
Fenitrothion	Insecticide
Fensulfothion	Insecticide
Fenthion	Insecticide, Larvicide
Fonofos	Insecticide
Glyphosate	Herbicide
Isofenphos	Insecticide
Leptophos	Insecticide

[3] The National Registration Authority 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, such as licensing of pest control operators and aerial sprayers. All agricultural and veterinary chemical products containing registered active ingredients are required to bear approved labels stating what the active ingredient is and its percent concentration in that product.

NAME OF ACTIVE INGREDIENT	MAIN USE
Malathion	Insecticide
Mephosfolan	Insecticide
Methamidophos	Insecticide
Mevinphos	Insecticide
Monocrotophos	Insecticide
Naled	Insecticide
Omethoate	Insecticide
Parathion	Insecticide
Parathion-methyl	Insecticide
Phorate	Insecticide
Phosalone	Insecticide
Phosfamidon	Insecticide
Phosfolan	Insecticide
Phosmet	Insecticide Acaricide
Phoxim	Insecticide
Pirimiphos-ethyl	Insecticide
Profenofos	Insecticide Acaricide
Propetamphos	Insecticide Acaricide
Prothiofos	Insecticide
Prothoate	Acaricide, Insecticide
Quinalphos	Insecticide
Schradan	Insecticide
SSS- Tributyl phosphorotrithioate	Plant growth regulator
Sulfotep	Insecticide
Sulprofos	Insecticide
Temephos	Insecticide, Larvicide
TEPP	Acaricide
Terbufos	Insecticide applied to soil
Tetrachlorvinphos	Insecticide
Thiometon	Insecticide
Thionazin	Nematocide
Tolclofos-methyl	Fungicide
Triazophos	Insecticide
Trichlorfon	Insecticide
Trichloronat	Insecticide applied to soil
Trichlorphon	Insecticide
Vamidothion	Insecticide

1. GUIDELINES* FOR HEALTH SURVEILLANCE FOR PENTACHLOROPHENOL (PCP)

BASELINE HEALTH SURVEILLANCE AT TIME OF EMPLOYMENT IN A PCP PROCESS

1. Collection of Demographic Data

- Name and unique company identification number.
- Date of birth.
- Sex.
- Address.
- Date of starting company service.
- Descriptive job title. To include the Australian Bureau of Statistics' *Australian Standard Classification of Occupations* (ASCO)[1] and *Australian Standard Industrial Classification* (ASIC)[2].
- Places of previous employment.

2. Occupational History

- Past work history, including previous exposure to PCP.
- Potential current exposure.
- Whether suitable personal protective equipment is used for that specific PCP process.

3. Medical History

- Presence of symptoms.
- Smoking history.

4. Physical Examination

A thorough examination of all skin areas, noting any abnormal lesions.

5. Investigation

- Spot creatinine corrected urine for total PCP to be conducted. Where there is 1 mg or more of total PCP per gram creatinine, a repeat spot creatinine corrected urine for total PCP should be performed at the same time of the day.
- Dipstick urinalysis for haematuria and proteinuria.

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* These guidelines set out in a practical manner the minimum requirements for health surveillance and should be read in conjunction with the Introduction to the *Guidelines for Health Surveillance* [NOHSC:7039(1995)].

6. Health Advice

The appointed medical practitioner should inform the employee of the potential health effects associated with exposure to PCP. In particular, employees should be aware of the occurrence and recognition of skin changes and irritancy, and the need to report them as soon as possible to the appointed medical practitioner, even if they occur between regular surveillance.

DURING EXPOSURE TO A PCP PROCESS

7. Personal Protective Equipment

The availability, type, fit, maintenance and frequency of use of personal protective equipment should be monitored regularly.

8. Urinary Total PCP

Baseline Level

Spot creatinine corrected urine for total PCP and dipstick urinalysis for proteinuria and haematuria to be conducted every 180 days, towards the end of a working week. Where there is 1 mg total PCP or more per gram creatinine, a repeat spot creatinine corrected urine for total PCP should be performed at the same time of the day.

Action Level

On confirmation of a level of 1 mg total PCP or more per gram of creatinine, a medical examination with emphasis on the hepatic and renal systems and skin should be performed. Depending on medical examination findings, further tests may be needed which may include serum biochemistry, urea and electrolytes, and a coagulation profile.

The employer should be informed when abnormal findings are detected so that control measures can be checked. The employee should be informed of the results of the health surveillance.

Removal Level

Removal from PCP work should be considered if there is evidence of adverse health effects due to PCP. Spot creatinine corrected urine for total PCP should be repeated every 30 days until the level falls below 1 mg per gram of creatinine.

Return to Work

The person must be medically fit to return to PCP work.

9. Data for Inclusion in Health Records

The following data should be included with the health records of the individual employee:

- Any formal assessments carried out in compliance with the National Occupational Health and Safety Commission's *National Model Regulations for the Control of Workplace Hazardous Substances* [NOHSC1005(1994)][3].
- Descriptive job titles, with relevant start and finish dates. Those jobs where exposure has been assessed as significant should be clearly identified.
- Results of atmospheric and personal monitoring and investigation of results that exceed the national exposure standard.

AT TERMINATION OF EMPLOYMENT IN A PCP PROCESS

10. Data that should be Collected

- Date of termination.
- Reason for termination:
  - ill-health (if 'yes', give details),
  - other reasons, and
  - date and cause of death if in service.

2. SUPPLEMENTARY INFORMATION ON PCP

WORK ACTIVITIES THAT MAY REPRESENT A HIGH RISK EXPOSURE

PCP is used as a preservative against timber-destroying fungi, sapstain moulds and some timber-boring insects and termites. Chloro-dibenzodioxins and dibenzfurans are known contaminants of PCP.

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

- application of PCP to timber;
- workers who handle or breathe air near wood that has been preserved with PCP; and
- emptying bags of granular or powder formulations of PCP and sodium PCP.

POTENTIAL HEALTH EFFECTS FOLLOWING EXPOSURE TO PCP

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.

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 centering on the circulatory system. Physiological injury is mainly muscular with heart failure.

Exposure to PCP may cause irritation of the eyes and respiratory tract. Concentrations as low as 0.3 mg/m^3 may cause irritation. Skin contact may lead to skin irritation. A 10 per cent solution may cause irritation after a single brief exposure, whereas prolonged or repeated contact with a 1 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 either a large exposure or repeated smaller exposures include weakness, loss of appetite, nausea, vomiting, shortness of breath, chest pain, excessive sweating, headaches and dizziness. 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. Persons with significantly impaired liver or kidney function are possibly more susceptible to poisoning from this substance.

Chronic Effects

Chronic exposure is associated with an increased prevalence of conjunctivitis, chronic sinusitis, bronchitis, polyneuritis and dermatitis.

Bronchitis has been reported at dust or mist concentrations of 1.0 mg/m^3. Repeated exposure to PCP may cause an acne-like rash and liver and/or kidney damage. Deaths have occurred among workers involved in crop and herbicidal spray operations where a 1.5-2 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 persons to hyperthermia.

Aspirin, which can also uncouple oxidative phosphorylation when absorbed in large amounts, may enhance the risk of toxicity for PCP-exposed persons. Because PCP is highly protein-bound, persons taking medications on a long-term basis that have a 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.

Carcinogenicity

The International Agency for Research on Cancer[4] recently reviewed the carcinogenicity of PCP in humans and animals.

In animal studies, dose-related increases in the incidence of hepatocellular adenomas and carcinomas, adrenal phaeochromocytomas and malignant vascular tumours of the liver and spleen have been observed[4].

The International Agency for Research on Cancer noted that 'pentachlorophenol has been mentioned specifically only in reports of two cases of Hodgkin's disease and seven cases of leukaemia of individuals who used it as a wood preservative or handled timber to which it had been applied and of two cases of non-Hodgkin's lymphoma of the skin in men employed in its manufacture. Generic reference to chlorophenols, which probably included pentachlorophenol, has also been made in three case-control studies which showed relative risks of 6.6 and 3.3 for soft tissues and sarcomas, 7.6 for heavy exposure and 2.2 for light exposure for lymphomas in association with exposure to chlorophenols. In none of these studies could exposure to pentachlorophenol be distinguished from exposure to dioxins'[4].

The International Agency for Research on Cancer concluded 'that there is inadequate evidence in humans for the carcinogenicity of pentachlorophenol and there is sufficient evidence in experimental animals for the carcinogenicity of pentachlorophenol'[4].

Carcinogen Classification

The International Agency for Research on Cancer's carcinogen classification of pentachlorophenol is possibly carcinogenic to humans (Group 2B).

Pentachlorophenol is not listed with a carcinogen classification in the National Commission's *List of Designated Hazardous Substances* [NOHSC:10005(1994)][5].

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1. GUIDELINES* FOR HEALTH SURVEILLANCE FOR POLYCYCLIC AROMATIC HYDROCARBONS (PAH)

BASELINE HEALTH SURVEILLANCE BEFORE EXPOSURE TO PAH

1. Collection of Demographic Data

- Name and unique company identification number.
- Date of birth.
- Sex.
- Address.
- Date of starting company service.
- Descriptive job title. To include the Australian Bureau of Statistics' *Australian Standard Classification of Occupations* (ASCO)[1] and *Australian Standard Industrial Classification* (ASIC)[2].
- Places of previous employment.

2. Occupational History

- Past work history, including previous exposure to PAH.
- Potential current exposure.
- Whether suitable personal protective equipment is used for PAH exposure.

3. Medical History

- Presence of symptoms.
- Smoking history.

4. Physical Examination

Conducted only if indicated by occupational and medical history.

5. Health Advice

The appointed medical practitioner should inform the employee of the potential health effects associated with exposure to PAH. In particular, employees should be aware of the occurrence and recognition of photosensitivity and skin changes and the need to report them as soon as possible to the appointed medical practitioner, even if they occur between regular surveillance.

*These guidelines set out in a practical manner the minimum requirements for health surveillance and should be read in conjunction with the Introduction to the *Guidelines for Health Surveillance* [NOHSC:7039(1995)].

DURING EXPOSURE TO PAH

6. Personal Protective Equipment

The availability, type, fit, maintenance and frequency of use of personal protective equipment should be monitored regularly.

7. Photosensitivity

Employees should be reminded to notify the appointed medical practitioner if photosensititivity occurs between regular surveillance. Employees reporting photosensitivity should receive additional counselling on the potential health effects of PAH on the skin.

8. Data for Inclusion in Health Records

The following data should be included with the health records of the individual employee:

- Any formal assessments carried out in compliance with the National Occupational Health and Safety Commission's *National Model Regulations for the Control of Workplace Hazardous Substances* [NOHSC:1005(1994)][3].
- Descriptive job titles, with relevant start and finish dates. Those jobs within areas where PAH exposure occurs should be clearly identified.
- Results of atmospheric and personal monitoring and investigations of results that exceed the national exposure standard.
- Record and review of any photosensitivity which an employee has had, indicating specific processes involved.

9. Medical Examination

If employees are excessively exposed or suspected of being so, or have concerns which may be related to PAH exposure, they should be seen by the appointed medical practitioner.

The employer should be informed when abnormal findings are detected so that control measures can be checked. The employee should be informed of the results of the health surveillance.

AT TERMINATION OF EMPLOYMENT WHERE PAH EXPOSURE OCCURS

10. Data that should be Collected

- Date of termination.
- Reason for termination:
  - ill-health (if 'yes', give details),
  - other reasons, and
  - date and cause of death if in service.

2. SUPPLEMENTARY INFORMATION ON PAH

WORK ACTIVITIES THAT MAY REPRESENT A HIGH RISK EXPOSURE

PAH are organic compounds consisting of three 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; and
- diesel emissions.

POTENTIAL HEALTH EFFECTS FOLLOWING EXPOSURE TO PAH

Route of Entry into the Body

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

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 sex 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.

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.

Carcinogenicity

There is sufficient evidence that PAH are carcinogenic to experimental animals. There is evidence that workers exposed to high airborne levels of some PAH show excess rates of lung, kidney, bladder, gastrointestinal and skin cancers. Some well-known carcinogenic PAH are benzo(a)pyrene, benzo(a)anthracene and dibenzo(a,h)anthracene.

Carcinogen Classification

Benzo(a)pyrene, benzo(a)anthracene and dibenzo(a,h)anthracene are listed in the National Commission's *List of Designated Hazardous Substances* [NOHSC:10005(1994)][4] and are classified as Carcinogen Category 2. According to the National Commission's *Approved Criteria for Classifying Hazardous Substances* [NOHSC:1008(1994)][5], a substance is assigned Carcinogen Category 2 if there is sufficient evidence, on the basis of appropriate long-term animal studies or other relevant information, to provide a strong presumption that human exposure to that substance may result in the development of cancer.

Benzo(a)pyrene is also classified as a Mutagen and Teratogen Category 2[4]. A substance is classified as Mutagen or Teratogen if there is sufficient evidence, generally on the basis of appropriate animal studies and other relevant information, to provide a strong presumption that human exposure can result in the development of heritable genetic damage or may result in non-heritable birth defects in offspring, respectively[5].

3. REFERENCED DOCUMENTS

1. Australian Bureau of Statistics, *Australian Standard Classification of Occupations: ASCO Coding System*, Australian Bureau of Statistics, Canberra, 1993.

2. Australian Bureau of Statistics, *Australian Standard Industrial Classification*, Australian Bureau of Statistics, Canberra, 1985.

3. National Occupational Health and Safety Commission, *National Model Regulations for the Control of Workplace Hazardous Substances* [NOHSC:1005(1994)], Australian Government Publishing Service, Canberra, 1994.

4. National Occupational Health and Safety Commission, *List of Designated Hazardous Substances* [NOHSC:10005(1994)], Australian Government Publishing Service, Canberra, 1994.

5. National Occupational Health and Safety Commission, *Approved Criteria for Classifying Hazardous Substances* [NOHSC:1008(1994)], Australian Government Publishing Service, Canberra, 1994.

4. FURTHER READING

Agency for Toxic Substances and Disease Registry, *Case Studies in Environmental Medicine 13: Polynuclear Aromatic Hydrocarbon (PAH) Toxicity*, Agency for Toxic Substances and Disease Registry, United States Department of Health and Human Services, Public Health Service, Atlanta, 1990.

International Agency for Research on Cancer, *IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans, vols 32-35: Polynuclear Aromatic Compounds*, International Agency for Research on Cancer, Lyon, 1984-1985.

Lauwerys RR and Hoet P, *Industrial Chemical Exposure Guidelines for Biological Monitoring*, 2nd Ed, Lewis Publishers, Boca Raton, 1993.

National Occupational Health and Safety Commission, *Guidance Note for the Protection of Workers from the Ultraviolet Radiation in Sunlight* [NOHSC:3012(1991)], Canberra, 1991.

National Occupational Health and Safety Commission, *National Strategy for the Prevention of Occupational Skin Disorders*, Australian Government Publishing Service, Canberra, 1989.

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

1. GUIDELINES* FOR HEALTH SURVEILLANCE FOR CRYSTALLINE SILICA

BASELINE HEALTH SURVEILLANCE BEFORE EXPOSURE TO CRYSTALLINE SILICA

1. Collection of Demographic Data

- Name and unique company identification number.
- Date of birth.
- Sex.
- Address.
- Date of starting company service.
- Descriptive job title. To include the Australian Bureau of Statistics' *Australian Standard Classification of Occupations* (ASCO)[1] and *Australian Standard Industrial Classification* (ASIC)[2].
- Places of previous employment.

2. Occupational History

- Past work history, including previous exposure to crystalline silica.
- Potential current exposure.
- Whether suitable personal protective equipment is used for crystalline silica exposure.

3. Medical History

- Presence of symptoms.
- Smoking history.
- Administration of a standardised respiratory questionnaire. Two examples are the international Union Against Tuberculosis' *Bronchial Symptoms Questionnaire 1986*[3] **or** the Medical Research Council's *Questionnaire on Respiratory Symptoms 1986*[4].

4. Physical Examination

With emphasis on the respiratory system.

*These guidelines set out in a practical manner the minimum requirements for health surveillance and should be read in conjunction with the Introduction to the *Guidelines for Health Surveillance* [NOHSC:7039(1995)].

5. Investigation

- Standardised respiratory function tests* to be performed. The tests are FEV_1, FVC and FEV_1/FVC. 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.

6. Health Advice

The appointed medical practitioner should inform the employee of the potential health effects associated with exposure to crystalline silica.

DURING EXPOSURE TO CRYSTALLINE SILICA

7. Personal Protective Equipment

The availability, type, fit, maintenance and frequency of use of personal protective equipment should be monitored regularly.

8. Data for Inclusion in Health Records

The following data should be included with the health records of the individual employee:

- Any formal assessments carried out in compliance with the National Occupational Health and Safety Commission's *National Model Regulations for the Control of Workplace Hazardous Substances* [NOHSC:1005(1994)][5].

- Descriptive job titles, with relevant start and finish dates. Those jobs within areas where crystalline silica exposure occurs should be clearly identified.

- Results of atmospheric and personal monitoring, and investigation of results that exceed the national exposure standard.

9. Medical Examination

A medical examination every five years as described in points 2, 3, 4 and 5 above, that is:

- occupational history;

- medical history;

- physical examination; and

- investigation.

NOTE: These requirements could be met by conducting a cross-sectional epidemiological survey every five years.

The employer should be informed when abnormal findings are detected so that control measures can be checked. The employee should be informed of the results of the health surveillance.

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

AT TERMINATION OF EMPLOYMENT WHERE CRYSTALLINE SILICA EXPOSURE OCCURS

10. Data that should be Collected

- Date of termination.
- Reason for termination:
  - ill-health (if 'yes', give details),
  - other reasons, and
  - date and cause of death if in service.

11. Final Medical Examination

A medical examination as described in points 3, 4 and 5 above, that is:

- medical history;
- physical examination; and
- investigation.

2. SUPPLEMENTARY INFORMATION ON CRYSTALLINE SILICA

WORK ACTIVITIES THAT MAY REPRESENT A HIGH RISK EXPOSURE

Quartz, cristobalite and tridymite are the main crystalline forms of silica. Quartz is the most widespread form of crystalline silica. It is a major constituent of igneous, sedimentary and metamorphic rocks, as well as being the major component of sand in locations such as stream beds, beaches and deserts.

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; and
- construction labouring activities.

POTENTIAL HEALTH EFFECTS FOLLOWING EXPOSURE TO SILICA

Atmospheric 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. Early silicosis may have no untoward effects. However, severe forms can result in poor gas exchange, difficulty in breathing and death. Evidence suggests that crystalline silica interacts with other respiratory hazards, such as tobacco smoke, to cause airway diseases.

Route of Entry into the Body

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

Acute Effects

Acute silicosis occurs after a short exposure to very high levels of silica. This could occur in exposure in confined spaces where respiratory protection is not worn. The condition causes rapidly progressive dyspnoea 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.

Chronic Effects

Chronic silicosis may be of the simple type where single nodules are present in the lung or it may progress to massive fibrosis. Workers with advanced massive fibrosis may be expected to have a largely restrictive functional abnormality.

Exposure to crystalline silica can also lead to other respiratory diseases such as chronic obstructive airway disease and bronchitis. These diseases can occur both in workers with and without silicosis.

Renal disease and scleroderma have been described in case-reports where workers were exposed to high levels of crystalline silica.

Carcinogenicity

There is some evidence that people with silicosis have an increased risk of developing lung cancer.

Carcinogen Classification

Currently, the National Commission's carcinogen classification for crystalline silica is under review. For the meantime, the International Agency for Research on Cancer classification for crystalline silica is stated. The International Agency for Research on Cancer[6] classification for crystalline silica is Group 2A, probably carcinogenic to humans (1987). According to the International Agency for Research on Cancer, this category is used when there is limited evidence of carcinogenicity in humans and sufficient evidence of carcinogenicity in experimental animals. Exceptionally, an agent may be classified into this category solely on the basis of limited evidence of carcinogenicity in humans or of sufficient evidence of carcinogenicity in experimental animals strengthened by supporting evidence from other relevant data.

3. REFERENCED DOCUMENTS

1. Australian Bureau of Statistics, *Australian Standard Classification of Occupations: ASCO Coding System*, Australian Bureau of Statistics, Canberra, 1993.

2. Australian Bureau of Statistics, *Australian Standard Industrial Classification*, Australian Bureau of Statistics, Canberra, 1985.

3. Respiratory Disease Committee of the International Union Against Tuberculosis, *IUAT Bronchial Symptoms Questionnaire*, International Union Against Tuberculosis, 1986☎.

4. Medical Research Council Committee on Research into Chronic Bronchitis, *MRC Questionnaire on Respiratory Symptoms*, Medical Research Council, 1986☎.

5. National Occupational Health and Safety Commission, *National Model Regulations for the Control of Workplace Hazardous Substances* [NOHSC:1005(1994)], Australian Government Publishing Service, Canberra, 1994.

6. 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.

☎Reproductions of these respiratory questionnaires can be purchased from Publications Sales at Worksafe Australia. Telephone (02) 577 9555.

4. FURTHER READING

Hilt B, 'Crystalline Silica', in Beije B and Lundberg P (eds), *Criteria Documents from the Nordic Expert Group, 1992*, Arbete och Halsa, vol 35, pp 1-80, 1993.

1. GUIDELINES* FOR HEALTH SURVEILLANCE FOR THALLIUM

BASELINE HEALTH SURVEILLANCE AT TIME OF EMPLOYMENT IN A THALLIUM PROCESS

1. Collection of Demographic Data

- Name and unique company identification number.
- Date of birth.
- Sex.
- Address.
- Date of starting company service.
- Descriptive job title. To include the Australian Bureau of Statistics' *Australian Standard Classification of Occupations* (ASCO)[1] and *Australian Standard Industrial Classification* (ASIC)[2].
- Places of previous employment.

2. Occupational History

- Past work history.
- Potential current exposure.
- Whether suitable personal protective equipment is used for that specific thallium process.

3. Medical History

- Presence of symptoms.
- Smoking history.

4. Physical Examination

Conducted only if indicated by occupational and medical history.

5. Investigation

Spot creatinine corrected urine for thallium to be conducted. Where there is 50 µg thallium or more per gram of creatinine, a repeat spot creatinine corrected urine for thallium should be performed at the same time of the day.

6. Health Advice

The appointed medical practitioner should inform the employee of the potential health effects associated with exposure to thallium.

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*These guidelines set out in a practical manner the minimum requirements for health surveillance and should be read in conjunction with the Introduction to the *Guidelines for Health Surveillance* [NOHSC:7039(1995)].

DURING EXPOSURE TO A THALLIUM PROCESS

7. Personal Protective Equipment

The availability, type, fit, maintenance and frequency of use of personal protective equipment should be monitored regularly.

8. Urinary Thallium

Baseline Level

Spot creatinine corrected urine for thallium to be conducted every 90 days. Where there is 100 µg thallium or more per gram of creatinine, a repeat spot creatinine corrected urine for thallium should be performed at the same time of the day.

Action Level

On confirmation of a level of 100 µg thallium or more per gram of creatinine, a physical examination should be performed with particular attention to the nervous system and noting any hair loss.

The employer should be informed when abnormal findings are detected so that control measures can be checked. The employee should be informed of results of the health surveillance.

Removal Level

Removal from thallium work should be considered if the level is above 150 µg per gram of creatinine. Spot creatinine corrected urine for thallium should be repeated within 30 days and then at regular intervals until the level falls below 50 µg thallium per gram of creatinine.

Return to Work

The person must be medically fit to return to thallium work.

AT TERMINATION OF EMPLOYMENT IN A THALLIUM PROCESS

9. Data that should be Collected

- Date of termination.
- Reason for termination:
  - ill-health (if 'yes', give details),
  - other reasons, and
  - date and cause of death if in service.

10. Final Physical Examination and Investigation

Physical examination and investigation as described in points 4 and 5 above.

2. SUPPLEMENTARY INFORMATION ON THALLIUM

WORK ACTIVITIES THAT MAY REPRESENT A HIGH RISK EXPOSURE

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

- laboratory analysis where thallium malonate-formate (Clerici's reagent) is used for mineralogic analysis of rocks, ores and sand, and separation of diamonds; and
- production of pigments, luminous paints, artificial gems, coloured glass and special optical glasses for lenses and prisms.

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

POTENTIAL HEALTH EFFECTS FOLLOWING EXPOSURE TO THALLIUM

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

Route of Entry into the Body

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

Acute Effects

Thallium and thallium compounds are extremely toxic. For humans, doses of 14 mg/kg and above are fatal. 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.

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 15-20 days.

Chronic Effects

Thallium may act as a cumulative poison with chronic intoxications and any 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.

3. REFERENCED DOCUMENTS

1. Australian Bureau of Statistics, *Australian Standard Classification of Occupations: ASCO Coding System*, Australian Bureau of Statistics, Canberra, 1993.

2. Australian Bureau of Statistics, *Australian Standard Industrial Classification*, Australian Bureau of Statistics, Canberra, 1985.

4. FURTHER READING

Lauwerys RR and Hoet P, *Industrial Chemical Exposure Guidelines for Biological Monitoring*, 2nd Ed, Lewis Publishers, Boca Raton, 1993.

Manzo L and Sabbioni E, 'Thallium', in Seiler HG and Seigel H (eds), *Handbook on Toxicity of Inorganic Compounds*, Marcel Dekker Inc, New York, 1988.

1. GUIDELINES* FOR HEALTH SURVEILLANCE FOR VINYL CHLORIDE

The data required to be collected for health surveillance are in accordance with items at Appendix 6 of the National Occupational Health and Safety Commission's *National Code of Practice for the Safe Use of Vinyl Chloride* [NOHSC:2004(1990)][1].

BASELINE HEALTH SURVEILLANCE AT TIME OF EMPLOYMENT IN A VINYL CHLORIDE PROCESS

1. Collection of Demographic Data

- Name and unique company identification number.
- Date of birth.
- Sex.
- Address.
- Date of starting company service.
- Descriptive job title. To include the Australian Bureau of Statistics' *Australian Standard Classification of Occupations* (ASCO)[2] and *Australian Standard Industrial Classification* (ASIC)[3].
- Places of previous employment.

2. Occupational History

- Past work history, including previous exposure to vinyl chloride.
- Potential current exposure.
- Whether suitable personal protective equipment is used for that specific vinyl chloride process.

3. Medical History

- Presence of symptoms.
- Smoking history.

4. Physical Examination

Conducted only if indicated by occupational and medical history.

5. Health Advice

The appointed medical practitioner should inform the employee of the potential health effects associated with exposure to vinyl chloride.

*These guidelines set out in a practical manner the minimum requirements for health surveillance and should be read in conjunction with the Introduction to the *Guidelines for Health Surveillance* [NOHSC:7039(1995)].

DURING EXPOSURE TO A VINYL CHLORIDE PROCESS

6. Personal Protective Equipment

The availability, type, fit, maintenance and frequency of use of personal protective equipment should be monitored regularly.

7. Data for Inclusion in Health Records

The following data should be included with the health records of the individual employee:

- Any formal assessments carried out in compliance with the National Commission's *National Model Regulations for the Control of Workplace Hazardous Substances* [NOHSC:1005(1994)][4].
- Descriptive job titles, with relevant start and finish dates. Those jobs within areas where vinyl chloride is used should be clearly identified.
- Results of atmospheric and personal monitoring, and investigation of results that exceed the national exposure standard.

8. Medical Examination

If employees are excessively exposed or suspected of being so, or have concerns which may be related to vinyl chloride exposure, they should be seen by the appointed medical practitioner.

The employer should be informed when abnormal findings are detected so that control measures can be checked. The employee should be informed of the results of the health surveillance.

AT TERMINATION OF EMPLOYMENT IN A VINYL CHLORIDE PROCESS

9. Data that should be Collected

- Date of termination.
- Reason for termination:
  - ill-health (if 'yes', give reasons),
  - other reasons, and
  - date and cause of death if in service.

2. SUPPLEMENTARY INFORMATION ON VINYL CHLORIDE

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, in particular, during cleaning of autoclaves.

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

POTENTIAL HEALTH EFFECTS FOLLOWING EXPOSURE TO VINYL CHLORIDE

Route of Entry into the Body

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

Acute Effects

At concentrations of several thousand parts per million, vinyl chloride affects the central nervous system causing symptoms such as euphoria, headaches, dizziness and loss of consciousness.

Dermal exposure to liquid vinyl chloride is possible. Skin burns may result by rapid evaporation of liquid vinyl chloride and consequent freezing of the skin.

Chronic Effects

Long-term exposure to vinyl chloride at concentrations of probably several hundred parts per million may result in scleroderma, Raynauds syndrome, acro-osteolysis (bone reabsorption of the terminal phalanges of the fingers), and fibrosis of the liver and spleen.

Carcinogenicity

Vinyl chloride is genotoxic.

A large number of epidemiological studies[5] and case reports have substantiated the casual 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.

Carcinogen Classification

Vinyl Chloride is listed in the National Commission's *List of Designated Hazardous Substances* [NOHSC:10005(1995)][6] and is classified as Carcinogen Category 2. According to the National Commission's *Approved Criteria for Classifying Hazardous Substances* [NOHSC:1008(1994)][7], a substance is assigned Carcinogen Category 2 if there is sufficient evidence, on the basis of appropriate long-term animal studies or other relevant information, to provide a strong presumption that human exposure to that substance may result in the development of cancer.

3. REFERENCED DOCUMENTS

1. National Occupational Health and Safety Commission, *National Code of Practice for the Safe Use of Vinyl Chloride* [NOHSC:2004(1990)], Australian Government Publishing Service, Canberra, 1990.

2. Australian Bureau of Statistics, *Australian Standard Classification of Occupations: ASCO Coding System*, Australian Bureau of Statistics, Canberra, 1993.

3. Australian Bureau of Statistics, *Australian Standard Industrial Classification*, Australian Bureau of Statistics, Canberra, 1985.

4. National Occupational Health and Safety Commissions, *National Model Regulations for the Control of Workplace Hazardous Substances* [NOHSC:1005(1994)], Australian Government Publishing Service, Canberra, 1994.

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6. National Occupational Health and Safety Commission, *List of Designated Hazardous Substances* [NOHSC:10005(1994)], Australian Government Publishing Service, Canberra, 1994.

7. National Occupational Health and Safety Commission, *Approved Criteria for Classifying Hazardous Substances* [NOHSC:1008(1994)], Australian Government Publishing Service, Canberra, 1994.

4. FURTHER READING

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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.

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