A report on occupational health and safety at the Fruitgrowers Chemical Company remediation site, Mapua

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Department of Labour
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ACKNOWLEDGEMENT

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PREFACE

In 2006, the Parliamentary Commissioner for the Environment (PCE) began an investigation into allegations of poor environmental management of the remediation of a contaminated site at Mapua from members of the public.

The PCE referred issues surrounding human health to the Ministry of Health and workplace health and safety issues to the Department of Labour for investigation.

The Ministry of Health has reported publicly on its findings on health effects of the remediation on residents in surrounding areas.

This report considers:
1. Whether there was evidence of a failure to have in place an adequate health and safety management system at the site.
2. Whether there was evidence of adverse health effects to workers from the FCC site clean-up operations.
3. Whether there was evidence of failures to inform the workers of significant exposures.
4. How the Department of Labour was involved with this workplace.

The initial brief of the investigator was to review the information held on file by the Department of Labour. This included the toxicology assessment and public exposure assessment from the Ministry of Health report and the PCE report. On request, the Ministry for the Environment (MfE) provided its project management documents. The investigation also included:
- Interviews with the individuals who had health complaints associated with the clean-up
- Interviews with the local representatives of the Department of Labour, the Ministry of Health and Tasman District Council
- A commentary on the toxicological profiles of the chemicals on site
- An assessment of whether or not there was a link between the work activities and the health concerns of site workers.

The interviews were carried out in January 2009 and November 2010 in Nelson. The investigation did not include interviews with the site managers but drafts were made available for comment.

I am grateful for the peer-review of Associate Professor Tim Driscoll of
The University of Sydney.

BACKGROUND

The Fruitgrowers Chemical Company was founded in Mapua beside the mouth of the Waimea river estuary near Nelson in 1931. The company produced organochlorine pesticides such as DDT, DDD and Dieldrin and synthetic plant hormones such as 2,4,5T and 2,4D.

By the late 1970s the plant was making 84 different pesticides, herbicides, insecticides and fungicides, using 124 chemicals.

The plant closed in 1988 and became an "orphan contaminated site". An orphan site is one where either no party can be identified as having legal liability, or the liable party is unable to fully fund the remediation of the site.

The site was classified as New Zealand's most contaminated.

Several investigations into contamination on the site, surrounding marine sediments and adjacent residential lots identified the presence of some of the substances known to have been stored or manufactured on-site, including:

- Extensive contamination with organochlorine pesticides (OCPs), especially DDT and its breakdown products (collectively DDX), Aldrin, Dieldrin and Lindane (collectively ADL)
- Occasionally elevated levels of heavy metals (including chromium, arsenic, lead, cadmium and mercury) and elemental sulphur. Arsenic and mercury have been discounted as metals of concern
- Occasionally elevated levels of petroleum hydrocarbons
- Traces of chlorophenoxyacetic acid herbicides, phenoxy herbicides organophosphates, triazines and other related nitrogen-containing pesticides
- Traces of polychlorinated biphenyls (PCBs).

Contamination was typically found in areas used for chemical handling and bulk storage, and within stormwater drains and low-lying areas. Concrete on the site was also contaminated. Topsoil on four neighbouring properties was contaminated to varying degrees, and parts of two of these properties were included in the area to be remediated.

Marine sediment samples from the Waimea Inlet revealed contamination mainly by OCPs, particularly DDT and its metabolites, and to a lesser extent Dieldrin. Metal and OCP levels in groundwater exceeded guidelines for the protection of aquatic ecosystems and recreational water quality.

Decontamination

In 1999, the Ministry for the Environment allocated funding to a decontamination programme.

Two years later, the Tasman District Council awarded a contract for remedial work to a partnership of Thiess Services (an Australian remediation specialist) and Environmental Decontamination Limited (EDL) of Auckland. Thiess was the main contractor and was to hold the resource consents. EDL supplied the remedial technology, Mechano-Chemical

Dehalogenation (MCD). EDL had received more than $450,000 in funding from the Foundation for Research, Science and Technology to help with development of the MCD technology which it had done at its Auckland base

In November 2001, the full scale MCD reactor unit was installed on-site at Mapua under a short-term resource consent for preliminary trials. In 1999, 300 cubic metres of earth (containing 800 PPM DDX) had been excavated for use as a "trial pile" for Proof of Performance (POP) testing of the plant. Initial treatment results were promising, although vibration from the plant was a problem for neighbours. The MCD reactor was moved to its final site and modifications made to the plant.

A resource consent application was made during August 2003 and the appeal process was completed in November 2003. In December 2003 a Hazard and Operability Study (HAZOP) was carried out on the plant. Fugitive emissions of the collected dusts, failure of equipment items and the possible breach of environmental conditions were discussed and a range of 59 actions were generated. Each of these was to be addressed prior to the final commissioning of the plant.

Actual POP testing occurred between February and April 2004. During one of the four trials, a mechanical breakdown led to the formation and release of contaminants that included small quantities of 2,3,7,8 Tetrachlorodibenzodioxin (dioxin) from the dryer of the MCD plant. It was considered that the dioxin emissions did not represent emissions during normal operating conditions, and that the problem could be eliminated.

By mid-2004, the relationship between Thiess and EDL had "broken down" and in August 2004 Thiess withdrew from the project.

Resource consents for the project were transferred from Thiess to MfE, which appointed Effective Management Services Ltd (EMS) as site manager.

EDL signed a contract with MfE to complete the soil treatment part of the remediation works. It appears that one factor in EDL continuing once Thiess pulled out was that the resource consent that had been granted was specifically for the operation of the MCD technology developed by EDL for use on the site.

Remediation eventually began in September 2004. There were problems in two main areas of the plant: firstly with the soil dryer, and secondly with the emissions control system. The dryer ran at too high a temperature at times, resulting in volatilisation of the organochlorine and other pesticide contaminants. The carbon filter in the air emissions system deteriorated rapidly because of acidic emissions or became overloaded. This resulted in a further period in late 2004 to mid-2005 where there were significant emissions which may have included dioxin: these emissions were not monitored for dioxins. In 2005 and 2006 independent investigations were undertaken to solve these control problems. The next testing for dioxins took place in 2007.

The remediation was completed in 2008.

EXECUTIVE SUMMARY

Health and safety management

- MfE adopted part of the Thiess health and safety plan when it took over the site, but the plan was not fully developed until after plant operation started.
- MfE's policy was that contractors should develop their own health and safety plans, which is good practice.
- There were no clear lines of responsibility and poor follow up by MfE to ensure contractors fulfilled their responsibilities.
- The MfE plan required quarterly area and personal occupational monitoring – this did not occur.
- EDL's health and safety plan proceeded in parallel with work on the site, not in advance of that work.
- EDL's health and safety plan was not completed until 2007, three years after the proof of performance plant started.
- Environmental monitoring was being undertaken on, and at locations close to, the site, but this suffered from limitations as some of it was not carried out to recognised standards and some covered only a limited suite of chemicals. The advice from all the scientific advisers was that MfE were monitoring for the correct suite of chemicals – it was the placement of one high volume sampler that was questioned.
- Process emissions were efficiently monitored, but only a limited suite of chemicals was monitored, and this monitoring was not suitable for assessing occupational exposures.
- Health monitoring of individuals was based on a sound plan, but poorly executed – no explicit action levels to trigger executive action were set, results were often delayed so that action could not have been taken until some time after exposure, follow up of employees was inconsistent and administrative procedures poor so that some significant results went unrecognised.
- There was a failure to carry out a systematic hazard identification process at the start of the project.
- Ensuring that a systematic hazard identification process was operating would have identified existing hazards, new hazards as they arose and the hazards identified would have undergone regular assessment to assess their significance.
- Control mechanisms to eliminate, isolate or minimise hazards from the processing of the contaminated soil were inconsistently managed.
- Protection of employees from hazards was achieved using personal protection equipment, the least effective method, and with varying levels of effectiveness – hazard elimination is the desirable method.

Technology

- The MCD technology was new and untried on a major site resulting in a number of technical issues.
- Poor control of the soil dryer meant that in the initial stages of operation, temperatures would have exceeded safe levels and there would have been de-novo formation of unwanted products such as PCBs and dioxins.
- Back pressure is known to have caused "fugitive" chemical emissions which may have caused significant exposures to people working on or near the site.

- Changing the proprietary mix of chemicals which enhances the MCD process reaction caused an excess of ammonia and other, at times unknown, gases and vapours to be emitted.
- The air emissions control equipment at the plant allowed the release of process emissions into the general environment – modelling determined a low risk to neighbours of the plant, but the effect on employees cannot be determined with certainty.

Conclusions

This remediation project was technically complex and accomplished with new technology in a relatively short time frame which is a considerable achievement. This report was written with the benefit of hindsight, and any criticisms made are intended to inform the planning of future remediation projects.

Because of the nature of the project with such a diverse range of pesticides and other chemicals distributed around the site, some of which were known, some unknown, detailed health and safety planning should have been part of the overall management plan for the site.

These plans should have been well advanced prior to the start of the project. Unfortunately this was not so.

The conditions which underpinned these problems may well have been a failure to identify, in advance, resources in terms of personnel and finances to support the health and safety plan. The evidence for this is a failure to appoint a designated health and safety professional to guide the process, changes in the medical support and late payment of invoices for occupational monitoring.

The MFE did not understand, or was not fully aware of, its statutory responsibilities as Principal of the site. Although it is clear that parts of the health and safety management plan by MfE reached an "adequate basic" standard, some of the planning and management by contractors clearly did not. This was the responsibility of the contractor, who was, to all intents and purposes, the expert in how the plant operated and what hazards might result. The failure to manage this relationship effectively meant that MfE assumed responsibility.

The evidence suggests that all practicable steps were not taken on the Mapua remediation project and it was possible that the health of at least four workers was affected to some degree. There were opportunities for adverse exposures to occur. Future work-related health effects cannot be ruled out.

Subsequent monitoring of employees did not reveal exposure to major chemicals of interest at the site. However, the nature of the monitoring meant that some exposures went unrecognised and could not have been detected.

Due to the information gaps and quality and range of the monitoring data it is impossible to carry out a full assessment of worker exposure. There is clinical evidence that four employees involved with either the remediation plant or the associated laboratory

suffered from health effects. The chemicals found on site were a plausible cause of some, but not all, of these health effects.

Health and safety professionals from the Department of Labour (DoL) were involved in giving advice about health and safety at the site, which was appropriate. However their roles and responsibilities were not clearly defined.

There were inherent conflicts of interests in this relationship between two Crown entities.

An internal Department of Labour investigation was carried out into health and safety at the site. This was adequate, but given the unique nature of the site closer contact and more regular site visits may have been of benefit.

Recommendations

1. The Principals to the contract for the remediation (the Ministry for the Environment) should offer all workers from the site a medical assessment.
2. The Department of Labour should ensure that employer responsibilities under the HSE Act are met for any future projects involving remediation of chemically contaminated sites, and particularly, ensure that project plans:
   a. Have clear health and safety plans for workers in the project plans for the remediation
   b. Identify an appropriately qualified individual to have overall responsibility for health and safety for the project.
3. The Department of Labour should develop a policy on how it will provide support to other government departments on health and safety matters and how they should discharge their duties under the HSE Act.
4. The Department of Labour's Guidelines on the Clean-up of Contaminated Sites should be updated and should include recommendations that :
   a. A protocol is developed for each project to encourage workers to self-report symptoms
   b. Persons having control of places of work within the scope of the guidelines should be encouraged to involve an experienced independent occupational health consultant in projects that may involve a significant risk to on-site staff.

Figure 1. Mechano-Chemical Dehalogenation (MCD) process
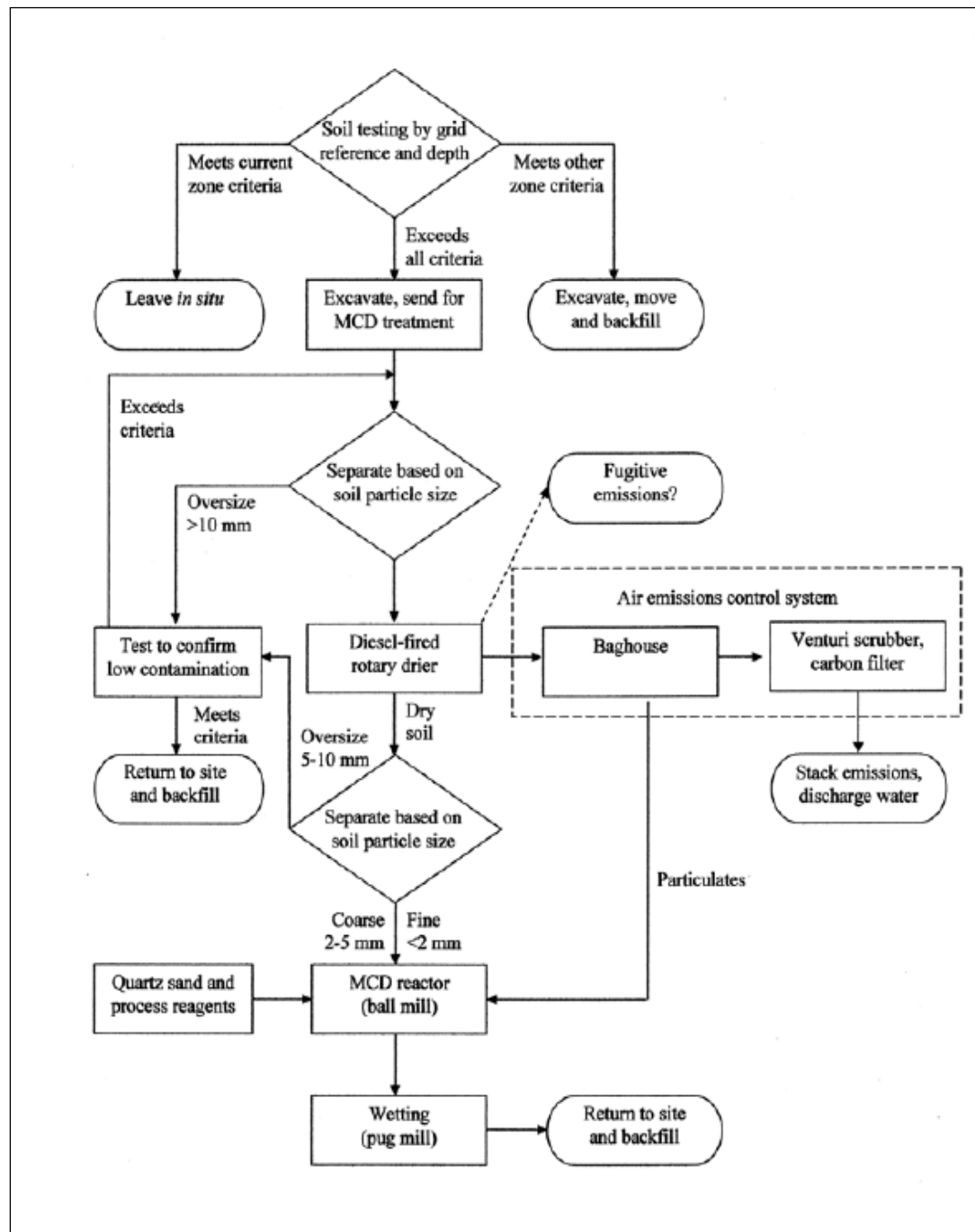
Source: Ministry for the Environment.

APPENDIX 1
HEALTH AND SAFETY MANAGEMENT AT THE FCC SITE

HEALTH AND SAFETY MANAGEMENT AT THE FCC SITE

General legal and regulatory requirements

The Health and Safety in Employment Act 1992 is the principal statute which governs occupational safety and health in New Zealand. The key concept of the Act is outlined in section 5: the intention expressed is for the act to promote "excellence in health and safety management by employers".

In contrast to a raft of early prescriptive legislation, the HSE Act was characterised by the fact that it is what is called "enabling legislation". This means that although it places the obligation on the employer to act regarding health and safety (in legal parlance, places a "general duty" to protect health and safety at work), the employer is also allowed to interpret how the Act applies to a specific workplace and employees. It is therefore up to the individual employer to manage health and safety according to the principles established by the Act.

Section 5 of the HSE Act lays out the principal object, and how this is to be achieved, the key sections being that:

> "The object of this Act is to promote the prevention of harm to all persons at work and other persons in, or in the vicinity of, a place of work by:
>
> (a) promoting excellence in health and safety management, in particular through promoting the systematic management of health and safety; and
>
> (b) defining hazards and harm in a comprehensive way so that all hazards and harm are covered, including harm caused by work-related stress and hazardous behaviour caused by certain temporary conditions; and
>
> (c) imposing various duties on persons who are responsible for work and those who do the work; and
>
> (d) setting requirements that:
>
>> (i) taking all practicable steps to ensure health and safety; and
>>
>> (ii) being flexible to cover different circumstances; and
>
> (e) recognising that volunteers doing work activities for other persons should have their health and safety protected because their well-being and work are as important as the well-being and work of employees; and
>
> (f) recognising that successful management of health and safety issues is best achieved through good faith co-operation in the place of work and, in particular, through the input of the persons doing the work; and
>
> (g) providing a range of enforcement methods, including various notices and prosecution, so as to enable an appropriate response to a failure to comply with the Act depending on its nature and gravity; and

(h) prohibiting persons from being indemnified or from indemnifying others against the cost of fines and infringement fees for failing to comply with the Act."

Section 6 has been described as the "pivotal" section of the Act stating that:

"Every employer shall take all practicable steps to ensure the health and safety of employees while at work."

This pivotal requirement of the Act restates the object of the Act (from section 5) in terms of a general duty for employers.

The section then expands on this general duty by prescribing the following particular duties to:
1. provide and maintain a safe working environment
2. provide and maintain facilities for the safety and health of employees at work;
3. ensure that plant machinery and equipment in the place of work is designed, made, set up, and maintained to be safe for employees
4. ensure that systems of work do not lead to employees being exposed to hazards in or around their place of work
5. develop procedures for dealing with emergencies that may arise while employees are at work.

The key section in this initial part of the Act is that the aim is "promoting excellence in health and safety management, in particular through promoting the systematic management of health and safety".

Most employers (whatever their sphere of expertise) have expertise in managing the hazards of their particular type of work. In many cases they are the experts in their particular type of work and help to develop industry "codes of practice" to help them carry out that work safely. They do so according to best practice, which is the fundamental underlying principle enshrined in the Health and Safety in Employment Act. It should also be recognised that most employees will be well experienced in their work and the conditions of their work, and have valuable contributions to make in planning health and safety.

Planning, organising and managing workplace health and safety

The Act is not prescriptive about "excellence" or the systematic management of health and safety, but there are New Zealand guidelines as to "best practice" in systems designed to manage workplace hazards. The most widely applied is the ACC Workplace Safety Management Practices programme, which provides ACC levy rebates to employers who meet satisfactory audit standards of health and safety performance.

The audit includes sections on employer commitment to safety management systems, including procedures for planning, review and evaluation. An initial stage of the audit involves having a documented health and safety policy authorised by senior management representatives, incorporating:

"management commitment to comply with relevant legislation, regulations, codes of practice and safe operating procedures, including specific understanding of management responsibilities for health and safety."

Specific guidelines for contaminated sites

Specific guidelines for contaminated sites are also given in the Department of Labour publication "Health and Safety Guidelines for the Cleanup of Contaminated Sites". This lists three important steps in planning and organisation.

Developing an overall organisational structure

In the development of an organisational structure, the essential and skilled personnel needed for the operation must be clearly identified right from the start. Establishing a chain of command, accountabilities and responsibilities for the key personnel is an early requirement. A structure that supports the overall objectives of the cleanup should be formed and this should include the following components:

- the appointment of a leader who has the authority to direct activities
- identification of the other key personnel for the project and confirming their functions and responsibilities
- establishment of clear lines of authority, responsibility, and communication
- development of an interface with the appropriate control or regulatory authorities.

As the project progresses, it may be necessary to modify the organisational aspects to recognise changes that take place in each phase. These changes may include downgrading the requirements which experience shows are not necessary, or upgrading the health and safety requirements to take account of new information. It is essential that any changes made are communicated to all key staff and others involved to ensure control is maintained.

Establishing a comprehensive work plan

To ensure a safe response, a work plan should be developed for each site which will include health and safety considerations. This plan is likely to vary according to the conditions and complexity of the job and the type of contamination. The plan should describe the anticipated cleanup activities and be managed in conjunction with the environmental assessment programme. A thorough field investigation programme and a review of all available information will provide the detail necessary to make the initial decisions on how the work should proceed. The preparation of a work plan will be more successful if it involves all appropriate disciplines and input from on-site and off-site personnel. It may be necessary to use occupational health consultants and agencies such as OSH and local authorities in this process.

Establishing a site health and safety plan

A comprehensive site health and safety plan for all workers should be prepared and implemented. This should establish policies and procedures designed to protect workers from the potential hazards at the site. The operator should produce a written health and safety plan to ensure that all personnel can be adequately informed of policies and decisions. Under the HSE Act, general duties for health and safety require the identification of hazards and, once identified, all practicable steps must be taken to isolate, eliminate, or minimise exposure, in that order, to that hazard. As required by the HSE Act, employees shall be involved in the development of procedures for identifying

hazards and dealing with emergencies. Regulatory agencies will also be able to assess the effectiveness of the plan.

On-site performance

Structure and responsibilities
As originally envisaged, the Principal for the contract was to be Thiess Services, an Australian specialist remediation contractor. Subsequently, the site management consisted of the Ministry for the Environment, which was overall manager. Responsibility for site operation was subcontracted to Environmental Management Services (EMS).

Environmental Decontamination Ltd (EDL) \ "was responsible not only for operating and maintaining the treatment plant, but for uplifting excavated soil for treatment, assessing pesticide concentrations before and after treatment, and stockpiling of the treated soils. Earthworks contractors (Highway Stabilisers Environmental (HSE)) carried out excavation and backfilling. Other contractors included the analytical laboratory and an environmental monitoring contractor. These contractors were originally engaged by Thiess, but for most of the remediation period were contracted to MfE." (PCE Report)

The engineers to the contract, MWH, had responsibility to monitor the terms of the contract to ensure that the contractors fulfilled their contractual obligations. This included the requirement for contractors to produce a health and safety management plan.

This relationship between the various parties was therefore that MfE was the Principal, ultimately responsible for health and safety at the site; EMS EDL and HSE were contractors with their own responsibilities. The responsibility of EMS, as site manager, was to monitor the processes of the other contractors. The "Guide to the HSE Act", (Department of Labour, 2003, page 81) says:

> *"A principal is required to monitor a contractor's performance in relation to employees' exposure to hazards. This is in addition to the contractor's responsibilities to their employees. What is practicable for the Principal will often differ from that expected of the contractor/employer in the circumstances. But if there is a step which it is practicable for a Principal to take, then there is a duty to take that step. The Principal cannot distance themselves from what is occurring in the workplace simply because the employer is more directly related to and responsible for the employees carrying out the work. It is a matter of fact and degree in each case, but the positive duty means "wilful blindness" is not acceptable."*

The management responsibilities at the site were laid out in an undated document, The Health and Safety Management Plan: "The MfE site representative has overall responsibility for the effective management of health and safety at the FCC remediation project". Health and safety requirements were also placed on subcontractors and suppliers. Paragraph 4 of the management plan states:

"All subcontractors working at the FCC Remediation Project site are required to develop and implement their own Workplace Health and Safety Plan. These individual plans must be submitted and approved by the MfE site representative prior to commencement of any work on the site."

MfE did develop an interface with regulatory bodies by seeking guidance from the Department of Labour through correspondence with a departmental medical practitioner (DMP). This occurred by means of a series of email communications with a Wellington-based DMP.

Work plan

Tasman District Council was required, under the terms of the resource consent, to appoint a peer review panel to:

"... review, comment and make recommendations on remediation management plans and monitoring reports."

The Peer Review Panel was formed in February 2004, with members selected for their expertise in noise, air quality, vibration, pesticide contamination, water resources and coastal ecology. The panel met at approximately quarterly intervals throughout the remedial works.

Site management meetings were also held (PCE Report):

"…as part of the operational process of managing the site, starting on 10 November 2004, these monthly meetings were between the site managers (EMS), the EDL project manager, the MfE project manager, the engineer (MWH) and occasionally others. The meetings were a forum to report and discuss operational matters such as health and safety, the performance of the plant and any problems. It appears, however, that some major matters – including the complete loss of the carbon filter in March 2005 – went entirely unreported at these meetings".

Minutes of the site management meetings on file at do, at times, contain a discussion of health and safety items. Initially, there was a named health and safety contributor who helped to draft the health and safety management plans.

Site health and safety plans and management

There were several levels of site management meetings. These included the monthly site management meetings previously referred to. It is not clear who chaired these meetings, although they were held at the MWH offices. On occasions the MfE Principal was not present. The first minuted site management meeting was held on the 27th January 2004 (with no health and safety item on the agenda). The meetings did include the contractors (EDL and HSE) who, from the minutes, appeared to attend the same meetings but at different times.

There were also weekly site management meetings between EMS, EDL and HSE.

The earliest record of a safety analysis was the hazard and operability study (HAZOP) carried out in December 2003 on behalf of Thiess by the Qest Consulting Group. A HAZOP study is a systematic analysis of a process (planned or existing) which identifies

problems that might disrupt efficient operation or present risks to operators or plant and equipment. The summary said that: "The main risks identified in the HAZOP study were fugitive emissions of the collected dusts, failure of equipment items and the possible breach of environmental conditions." There were 59 recommended actions recorded in the study report.

Thiess Services produced a management plan for proof of performance and stage 3 works, section 2 of which dealt with health and safety management. The document refers to AS/NZS 4801 "Occupational health and safety management systems – specification with guidance" for use in their health and safety plan. Thiess also required that the occupational health and safety system meet the requirements of AS/NZS/ISO 9001. Some of the statutory authorities quoted in this document were Australian in origin, so it is clear that this plan was under development. The MfE plan was based on this document.

The MfE Site Manager (John Roosen of EMS) had input at an early stage, producing five Standard Operating Procedures for the Site Induction/Safety Plan in November 2004. These related to explosion, fire, evacuation, medical emergency and chemical spill. They also produced a HAZOP dealing mainly with the excavation site, but a second version (deviation) did look at procedures for controlling dust, odours and breach of containers.

Juliet Westbury of MWH produced a draft Health and Safety Site Induction and Policy document on 3rd September 2004. The Health and Safety Policy was as follows:

SECTION 2   **HEALTH & SAFETY POLICY**

The Ministry for the Environment's Health & Safety Policy with respect to activities associated with the FCC Site Remediation project is provided as the first element of the site Health & Safety Management Plan. The key elements of this H & S Policy are summarised as follows:

The Ministry for the Environment (MfE), as project manager, is committed to ensuring the health and safety of the contractor, sub-contractors and visitors. The MfE is also committed to continuous improvement and will pursue best practice in occupational health and safety, and shall comply with the requirements of all relevant legislation.

Health and Safety Procedures:
Effective procedures have been introduced and will be maintained to ensure that all site hazards are identified and appropriate measures are introduced to control these hazards.

Management Responsibilities:
The MfE Project Manager has overall responsibility for the effective management of health and safety at the FCC site.

Employee Responsibilities:
All employees are responsible for carrying out work in a manner that does not adversely affect their own health and safety and that of other workers at the site.

The draft contained details of H&S duties, risk management, safety and environmental meetings, accident reporting and health monitoring.

The MfE were the authors of a "Workplace Induction" plan dated 27 April 2005, and a later, undated, Health and Safety Management Plan for the site. There are seven electronic versions on file, the first being electronically dated to November 2004, and the final version tracked to September 2009.

It appears that MWH also produced a draft of this plan. It was later (September 2005) reviewed by Laura Hurst, the occupational hygienist employed by EMS, specifically to include additional appendices on Health Monitoring (Attachment D) and "contaminant

information" (Attachment E) on organochlorine pesticides (DDT, Aldrin, Dieldrin and Lindane). She comments:

> "It has been interesting spending some time with the chemical info on these substances. They have very low melting points if you consider our 120 C cut off and they also decompose quite nicely in any flame. It will be interesting to see how long the carbon filter lasts with soil that has these contaminants. I have attached the chemical safety card info for all 4 major contaminants for your reference."

The induction plan does have a "general policy" statement, that: "For protection of the health and safety of site workers, the community and for the protection of the environment, all persons coming onto the Fruitgrowers Chemical Company Site at Mapua, New Zealand, are required to comply with these instructions."

The management plan also contains a policy statement "the MfE, as project manager, is committed to ensuring the health and safety of contractor employees, subcontractors and visitors at the FCC Remediation Project site at Mapua. The MfE is also committed to continuous improvement and will pursue best practice in occupational health and safety and shall comply with the requirements of all relevant legislation."

The management responsibilities were laid out in the management plan: "The MfE site representative has overall responsibility for the effective management of health and safety at the FCC remediation project". Health and safety requirements were also placed on sub-contractors and suppliers. Paragraph 4 of the management plan states:

> *"All sub-contractors working at the FCC Remediation Project site are required to develop and implement their own workplace health and safety plan. These individual plans must be submitted and approved by the MfE site representative prior to commencement of any work on the site."*

The EDL site induction document is dated September 2004. This was referred to as a management plan, but it was not.

The weekly site management meeting minutes of May 2005 record a review of the document requested in February and supplied in March. A memo concerning the deficiencies in the programme was given to EDL on the 10th March. A meeting to discuss the review of the plan with Brent Pascoe was postponed due to problems with the plant. The review took place on 8th April 2005. The review followed the details itemised in the memo of 10th March (2005) and centred on the need to adhere to stated procedures as well as to provide evidence that the employer responsibilities were being met in regard to safe work practices and protection of the employees from known hazards. Specifically, it was agreed that:

1)   The current EDL documentation was deficient in identifying the health hazards in the workplace,
2)   Requirements for a Job Safety Analysis for each new procedure were not being followed,

3)   Information about the chemicals used on site was incomplete, MSDSs were not available,

4)   First aid and personal protection equipment needed to be more accessible to all personnel,

5)   The company Health and Safety Committee was dysfunctional and/or inaccessible to Mapua staff and

6)   Information concerning the responsible staff on site required updating and the stated attached documentation needed to be attached.

The minutes indicate agreement from EDL that these items would be addressed as soon as practically possible.

The review made it clear that that the current workplace induction for EDL employees was insufficient and it was requested that all employees be required to partake in an MfE workplace induction.   A copy of the workplace induction documentation was given to Brent Pascoe on 22 April and full workplace inductions were conducted in two sessions for seven employees on 28 April.

The following concerns were raised by employees during the inductions:
1.   PPE was not readily available. Insufficient numbers of respirators, both the dust mask and cartridge half face mask varieties, were available to staff. Gloves were not supplied and a number of employees were providing their own hand protection.

2.   The current overall is effective in keeping out dust at the beginning of a shift but it was requested if a better type of overall that was not as porous as the current cotton ones but not so flammable as the Tyvek suits could be found. EMS agreed to research into alternatives.

3.   They were unaware of Material Safety Data Sheets and the information that could be obtained from them; specifically first aid and emergency information. It was requested that a copy of the pertinent ones be kept at the local medical centre in case of emergency treatment needs.

4.   There was a lack of recent first aid training in the group. It was agreed that [ ] and [ ]would apply to update their skills.

5.   The potential exposure to fine silica dust due to the sand dryer and other activities was a concern.   The risk of silicosis is dependent upon the quartz concentrations in the sand as well as the duration and level of exposure. EMS offered to attempt to obtain this information from the suppliers of the sand.

6.   First Aid Kits were in need of restocking.

7.   The lack of information on the Biological monitoring of 9 Feb. was a concern. Results were expected to be received at Dr [ ] office next week, 3 May 2005.

During discussion of the safety requirements for the site, it was stated that employees on site were not certified electricians or welders. It was considered unnecessary by EDL for the repair and maintenance work carried out by staff to have certified workers.

There was also disagreement concerning the last item in the induction regarding drugs and alcohol on site. It was considered not to be a safety concern to have a few beers on site after a work shift. Discussion about the inability of the

employee to react appropriately in the case of an emergency if they are intoxicated or the disruption to the other employees did not reach any agreement in the meeting.

It is pleasing to report that since the inductions last week, there is a marked improvement in the use of personal protective equipment. Dust masks and cartridge respirators are available to the staff and they are being worn.

The availability of a clean rest area to EDL staff has also been a concern. At the recent site management meeting, 2 May, it was agreed that the smoko container would be relocated outside the existing fence line and oriented away from the plant.

ACTIONS REQUIRED:
An extensive revision of the EDL H&S documentation to reflect the current level of health risk and the required safety practices.
Agreement to the no drugs no alcohol policy on the worksite.
Set a date for a refresher of Workplace induction for 6 weeks from the initial one – refresher to focus on potential health hazards and their association with specific work practices.
EMS to research into overalls and sand constituents.

There was no health and safety item on the EDL site management meeting minutes until the 2nd June 2005 when it was minuted that:

" [   ] indicated that there was a marked improvement in the health and safety aspects at the EDL plant, particularly with individuals wearing their respiratory equipment. However, the health and safety plan still needed updating. [   ] indicated that the original health and safety plan that he had provided was reduced because it had too much detail. It was indicated that [   ] had received a list from the site management team on what health and safety items needed to be included. [   ] indicated that [   ]should submit health and safety items to his contact at Fonterra who will complete the health and safety plan and resubmit it back to the site management team next week. [   ] indicated that it was extremely important that the health and safety items be completed as he has recently noted that individual managers are personally liable for any health and safety as infractions."

This plan seems to have taken some time to formulate. In fact it was not produced until the 20th October 2005 when it was presented to management.
 There is no record of this initial plan on file.

Section 3 of the EDL management plan is the health and safety plan, which is also undated, but the PDF document data identifies it as being produced on 20 March 2007. The introductory application statement says:

"This section of the management plan is related to health and safety management and is designed to satisfy the requirements of the Health and Safety

in Employment Act and amendments. The plan provides a management strategy to effectively manage all significant health and safety risks as deemed appropriate for operations. The objective of this section is to ensure that all risks to people, plant, property equipment and the environment are adequately identified, assessed and managed to the lowest acceptable level."

The policy statement is:

"It is the policy of EDL – Environmental Decontamination Ltd – to ensure that risks in the working environment are managed to ensure the safety of people, plant, property, equipment and the environment. In meeting these objectives, a systematic and planned control system for health and safety management will be implemented."

There is no written evidence that employees actually had formal input to the health and safety planning process. This is usually achieved by having a health and safety committee with employee representatives on it.

HSE were developing their plan (cited in site management minutes) in January 2005. They produced a Staff Manual dated February 2005 containing information on Health and Safety programme guidelines, emergency procedures, recording, reporting, investigating accidents, management of hazards in the workplace, personal protective equipment, health and hygiene control, OSH consultative process and general safety policies. The document lays out the H&S responsibilities of the project director, compliance manager; project manager site superintendent and employees.

The policy statement (Statement of Purpose) is: "To have a healthy, safe and accident free and productive work environment for all employees." There is a hazard register, but this is generic. At the 9th July 2005 site management meeting there is a minute to the effect that the manual was to be distributed to staff.

Summary of gaps identified

Thiess Services were initially responsible for the remediation works and for formulating a Remediation Action Plan and Site Management Plan with a health and safety section.

The MfE then took control. The PCE report states the project team included "legal and accountancy expertise, but the evidence suggests that they lacked operational experience with contaminated land issues or with civil engineering projects." A site management team was formed, and the MfE website does identify a person with a health and safety job title. The weekly site remediation team minutes identify an Industrial Hygienist [ ] as part of the Environmental Management Services team.

There are several weekly minutes in which she contributed to health and safety items, and she was the author of several reports. A site auditor was appointed, with no health and safety remit. The Peer Review Panel did not have a health and safety expert on it.

The role of the panel was to ensure compliance with the resource consent conditions. As such, it was not the role of the Peer Review Panel to provide health and safety expertise to the consent holder and their contractors. If however there are emissions to air,

ground or water then it could be argued that environmental health (or occupational and environmental) expertise should be available to the panel in order to evaluate potential risks to the community. There could also be some collateral advantage to employees

The first evidence of health and safety planning was the Health and Safety Management Plan electronically dated to November 2004. This does contain a policy statement, sets out responsibilities, defines the applicable legislation, defines the contaminants likely to be found on site, the likely exposure pathways, the personal protective equipment to be used and a health monitoring statement. The MfE project and site managers are personally identified, with the MfE site manager proposed by MWH as having the responsibility for health and safety. The person taking overall responsibility for health and safety was to be the MfE project manager, but this information does not appear on the documents, which are signed neither by the project manager [ ] nor the "site representative" [   ]. There are a number of versions of the document but it is not clear which one was in operation at which time.

The safety management plan states that "employees have the opportunity to participate in the development of health and safety policy at the site". However, there was no formal process (a health and safety committee) by which they could do so. The plan still does not identify the person responsible and remains unsigned. A health and safety person is identified as such on the MfE website, apparently part of the EMS team, but there is no formal description of the role or responsibilities of this person.

The evidence from the minutes of the June 2005 site management meeting indicate that there were significant problems with the EDL plan. It appears that these were not resolved until October 2005.

There is therefore no strong evidence that health and safety planning was a priority. The induction plan (which is not a health and safety plan) is dated one year after proof of performance testing started, the undated management plan presumably some time later. Neither is signed, and the latter is undated. The management plan does imply that employees were to be involved, but there are no details of how this was to be achieved. Although subcontractors were to develop their own health and safety plans, there is no documentary evidence that this was ever followed up. The use of the term "subcontractor" in this document is not correct strictly speaking because EDL and EMS seem to have been directly contracted to MfE and would have been contractors.

EDL provided their completed health and safety plan to MfE in April 2007. The signatory was the EDL project manager. This document was produced some three years after proof of performance testing started.

The HSE health and safety management plan was comprehensive and well thought out but lacked some detailed information on chemical risks.

Implications of gaps

The most serious implication is that there does not seem to have been a provision for health and safety planning to form part of the management plan. Health and safety, as far as can be ascertained, was not included in the Remediation Action Plan or the Site Management Plan (except in the original Thiess document), the site auditor is not

mentioned as having a remit in this area and there was no health and safety expert on the peer review panel.

The effect was that health and safety was not formally recognised at the start of the project and had neither management commitment nor a high profile. The outcome was a failure in forward planning and a health and safety management system that did not keep pace with the operational tempo.

The persons responsible for health and safety were identified generically, but clear lines of reporting were not established. Most seriously, the plans were not endorsed by a senior manager, so there was no senior management responsibility. It is well recognised that senior management commitment to health and safety is a major force in providing resources and ensuring that health and a safety is considered in management plans.

The resource issue was an important one. It was later recognised that health monitoring would be required, an expensive and time consuming process. This was decided at the management level in MfE, but was not initially identified as part of the contract. As a result there was dissent about paying for the monitoring with delays in processing the samples and interpreting the results.

Because no one had direct responsibility, the health and safety planning documents were tardy in production and, by implication, application. Workers were not aware that the MfE site representative was also the designated site health and safety representative

That is not to say that effort was not put into health and safety by the MfE site management team. EMS had a health and safety person on their team, identified as an occupational hygienist, who seems to have made a particular effort to address the health and safety management plans. They also put significant effort into helping the other contractors recognise their health and safety responsibilities under the Act. In particular they required one of the principal contractors, EDL, to produce their own health and safety management plan. The fact that they did not do so until 2007 and the initial poor quality of the document eventually produced indicates that MfE did not fully understand their roles and responsibilities.

Although EDL's health and safety performance was being monitored, failures were occurring during the period that plans were being produced. During this period detailed hazard identification and assessments were not carried out as required, there were failures to monitor the workers' environment to an acceptable standard and employees received health surveillance that was haphazard.

MfE therefore appears to have left it up to EDL to comply with their health and safety duties correctly, and there is no concrete evidence that they were monitoring this compliance. MfE also delegated responsibility to EDL in the matter of providing their workers with entry medicals and site induction training. Apparently several workers did not receive these.

In light of the number of hazards on site, their unknown distribution and potential significance, Thiess were prudent in basing their health and safety plans on international standards. It appears that MfE initially required EDL to meet ISO 9001 quality

management system requirements, but they were not required to provide third-party certification of the system and no formal accreditation audits were undertaken on the laboratory.

There was little evidence that employees were to be involved. For example there are no records of site health and safety planning meetings which involved employees.

Record keeping appears to have been very poor, including the accident reporting requirement. It appears that a worker had a serious harm accident which was neither reported to the Department of Labour nor investigated by anyone on-site.

Bearing in mind the complexity of the project, careful management of health and safety should have been a priority, but these oversights caused a failure to recognise that fact.

Hazard identification, assessment and control

Legal and statutory requirements

Sections 7-10 of the HSE Act place legal obligations on the employer to identify, assess and control hazards at work.

Hazard identification and assessment

Section 7 places the obligation on employers to ensure that they have in place effective methods for systematically identifying hazards, including existing hazards and new hazards as they arise. They are also required to assess each hazard regularly and assess if it is a "significant" hazard.

The Act does not prescribe how this process is to be carried out, but the steps must be systematic.

A systematic assessment requires a framework for assessment, the simplest approach being to visit the work site, observe the processes and activities, document the hazards (and how they are controlled) and use this as a basis for forming the health and safety management plan.

The process has been formalised by occupational hygienists as the "Walk Through Occupational Hygiene Survey", a survey technique which is fairly simply carried out by walking through a process (or workplace) from start to finish – or "goods in, product out" – while documenting the hazards. The technique is especially useful for new workplaces (which have not had a comprehensive health and safety assessment carried out) but is also useful as a periodic routine measure in workplaces to assess process-variable factors, which can change the nature of the hazards.

This provides the basic information to establish a framework on which to base future investigations, helps to prioritise hazards, determines the requirements for measurement and establishes some immediate controls of potentially hazardous exposures if they exist.

The survey is designed to address the following questions:
- What hazards are present in the workplace?
- Are these hazards significant?
- If so, what control measures are in place, and are these adequate?
- What procedures are implemented to maintain the control measures?
- What monitoring may be required?

In order to comply with the HSE Act, it is essential to be able to answer these questions. A matrix designed to assess performance in these areas is shown below.

The description of an "adequate basic" audit of the hazard identification structure and process is as follows:

> *"Ad hoc or basic hazard identification with major hazards identified. Limited documentation including hazard register. Evidence that significant hazards have been identified. Employees aware of hazard register."*

An "adequate fair" audit of the hazard identification structure and process is described as:

> *"Hazard identification shows evidence of systematic approach and is documented. Significant hazards identified and documented. Evidence of employee input."*

Table 1.   Health and Safety Performance Matrix

Assessment	Inadequate	Adequate basic	Adequate fair	Adequate sound	Adequate excellent
Hazard identification and assessment	Hazard identification non-existent or limited to basic safety. No hazard register. Limited or no assessment of significance.	Ad hoc or basic hazard identification with major hazards identified. Limited documentation including hazard register. Evidence that significant hazards have been identified. Employees aware of hazard register.	Hazard identification shows evidence of systematic approach and is documented. Significant hazards identified and documented. Evidence of employee input.	Systematic hazard identification by area process or activity with majority of hazards identified. Well documented hazard assessment with evidence of review. Led by experienced person with evidence of significant employee input.	Systematic and comprehensive hazard identification by area process or activity with all hazards identified. Documented and peer reviewed hazard assessment. Led by person with formal H&S qualification. Employee input at each stage.
Hazard control and monitoring	Not all hazards subject to control, and no evidence of monitoring.	Basic control such as general ventilation with provision of PPE for significant hazards. Limited but ad-hoc monitoring.	Some significant hazards eliminated or isolated. PPE for all significant hazards. Targeted environmental and health monitoring.	Most significant hazards eliminated isolated or minimised with PPE for residual control. Regular environmental monitoring with health surveillance.	All significant hazards eliminated isolated or minimised with PPE for residual control. Regular environmental monitoring with health surveillance.
Information and training	No hazard-specific hazard information available. No documented evidence of training.	Basic hazard information available (MSDs). Ad-hoc training on safety.	Information available on all major hazards. Evidence of health and safety training on induction.	Comprehensive information on all hazards. Formal Induction and on-going health and safety training.	Comprehensive information on all hazards. Formal Induction and ongoing health and safety training with regular assessment.
Accident and incident investigation	No accident register.	Accident register with evidence of review and action.	Accident register and "near-miss" reporting system. Evidence of review, action and follow up.	Comprehensive reporting and analysis system. Designated accident investigators.	Comprehensive reporting and analysis system. Designated accident investigators. Evidence of senior management follow-up and injury prevention initiatives.

On-site performance

The initial hazard identification document was produced by Thiess, material from which contributed to the MfE "Workplace Induction" manual dated 27 April 2005.

This does identify, in a table, "Contaminants likely to be found on-site" (taken from section 2.9 of the Thiess plan), as follows:

Table 2.   Contaminants likely to be found on site

Group	Substance	Media[1]	General Toxicity Information
Inorganics	Asbestos	S	Proven human carcinogen
Heavy Metals	Chromium (Cr)	S/L	Proven human carcinogen (Cr(VI))
	Arsenic (As)	S/L	Proven human carcinogen
	Lead (Pb)	S/L	Reproductive and central nervous system hazard
	Cadmium (Cd)	S/L	Suspected human carcinogen, kidney toxin
	Mercury (Hg)	S/L	Toxic to nervous system and many other organs
Organochlorine Pesticides (OCPs)	DDT	S/L	Central nervous system hazard, headaches, nausea
	DDD	S/L	Central nervous system hazard, headaches, nausea
	DDE	S/L	Central nervous system hazard, headaches, nausea
	Aldrin	S/L	Central nervous system hazard, headaches, nausea
	Dieldrin	S/L	Central nervous system hazard, headaches, nausea
	Lindane	S/L	Central nervous system hazard, headaches, nausea
	Phenoxy Acid Herbicides (2,4 D 2,4,5 T)	S/L	Central nervous system hazard, headaches, nausea
Organophosphorus Pesticides (OPPs)	Mevinphos	S/L	Central nervous system depression, headaches, nausea
	Dichlorovos	S/L	Central nervous system depression, headaches, nausea
	Hexamethyl-phosphoramide	S/L	Central nervous system depression, headaches, nausea
	Various Others	S/L	Central nervous system depression, headaches, nausea

Note: 1. S = Solid (soil or solid waste); L = Liquid (groundwater, surface water)

The table is presented with a caveat in that:

> "….The information in the above table does not provide a complete review of all contaminants that may be encountered on the site. Rather, the

information is presented to reinforce the potentially hazardous nature of the most significant and probable site contaminants."

Section 2 of the management plan also contains a policy statement:

"Effective procedures have been introduced and will be maintained to ensure that all site hazards are identified and appropriate measures are introduced to control these hazards."

Procedures will be reviewed and monitored to take account of changing conditions and circumstances at the FCC site.

Records will be kept of the hazard management programme.

Employees have the opportunity to participate in the development of H&S practices at the site.

All relevant documentation relating to occupational health and safety issues is made available to employees.

Hazard identification and risk assessment procedures are in place, are monitored and are regularly updated."

The Department of Labour did make specific recommendations to MfE in January 2005 regarding an occupational health monitoring policy which was under development at the time, recommending (sic):

"Full hazard identification on site, quantifying ALL RISKS, not only chemical ones, before commencement of work. This includes actual time exposed, i.e. length of shift, length of work week, personal safety, food and water safety, safety during lunch and rest breaks, personal protective gear, decontamination procedures, washing and disposal of contaminated clothing and coveralls, washing and personal cleansing protocols and facilities (contaminated clothing can put family members at risk) before commencement of work."

Reference is made to "unknown hazardous substances" and a standard operating procedure to deal with these. Other hazardous substances stored or used on site are referred to, as is a hazardous substances register, material safety data sheets (MSDS) and the reagents held by EDL, as follows:

Table 4.3.3 Extract from induction manual

The potential or confirmed contaminants are listed in the MSDS register held on site. This includes a summary of Workplace Exposure Standards for the various contaminants, the physical and chemical properties of the substances, acute and chronic health effects associated with these contaminants, and first aid procedures to be followed subsequent to exposure to these contaminants.

Unknown hazardous substances

Besides the contaminants listed above there may be unknown hazardous materials on the site that have either been buried in containers or may have been spilled onto the soil.

These unknown contaminants would include fuels, caustic material that may burn skin or the lungs, and chemicals that have not been discovered during the initial site investigation.

All workers, especially those involved in excavation activities, must be constantly vigilant during times when chemical containers, including drums, cans, pails, sacks or dumped product, may be discovered. An SOP [standard operating procedure] on "unknown chemicals or containers" is in development.

Other hazardous substances stored or used on-site

In addition to the contaminants that may be encountered as a result of site remediation and landfilling operations, a range of other hazardous substances are stored and used on-site. A Hazardous Substances Register for these substances is maintained on site, together with a Material Safety Data Sheet for each substance. This includes caustic cleaners, motor fuel, diesel or gas oil, solvents and other industrial materials. There are also a number of reagents and other materials held by Environmental Decontamination Limited. These materials are included in the Hazardous Substance Register. (See 10.1 for further details.)"

Material safety data sheets

As noted in 4.2, each chemical approved for site use must have a Material Safety Data sheet on file and accessible to all workers during an emergency. A complete file of MSD sheets will be maintained with the site's main first aid kit. MSD sheets provide information about the chemical in question including use, storage and handling as well as health effects, and first aid for exposure.

Section 5 of the document then deals with "Principal health and safety hazards", and deals with chemical contamination, routes of contamination, mechanical and operational hazards and treatment plan operational hazards.

Section 5.1 mentions the "unknown chemicals" used in the manufacture of pesticides.

Section 5.2 mentions the principal routes of absorption, (inhalation, ingestion, skin absorption and percutaneous). Section 5.3 mentions the mechanical and general operational hazards and 5.4 the treatment plan hazards. Page 8 mentions, in regard to treatment plan hazards, that: "A few of the specific hazards include: ….dust and vapours from equipment including that generated by the soil reactor, soil dryer, bag house and the pummel."

Attachment E of the Health and Safety Management Plan does give information in MSDS (Material Safety Data Sheet) format on the "principal contaminants" on the site – DDx, Aldrin, Dieldrin and Lindane for example. The presence of an MSDS, providing that it is adequate, is a first step in deciding upon the significance of a hazard. Section 4 of the workplace induction document does state that:

"A Hazardous Substances Register for these substances is maintained on site, together with a Material Safety Data Sheet for each substance. This includes a summary of Workplace Exposure Standards for the various contaminants, the physical and chemical properties of the substances, acute and chronic health effects associated with these contaminants, and first aid procedures to be followed subsequent to exposure to these contaminants."

Such an MSDS would be an adequate document on which to base a hazard assessment. However, there is little evidence that the process has been carried out to the second stage and the significance of the hazard assessed.

A further significant omission was a failure to put in place a system to deal with "new" chemicals as they become known to MfE.

Part of the information on file is documents regarding the effective procedures for identifying hazards requested from MfE under the Official Information Act by a site employee. A reply by the Acting Secretary states:

> *"Section 2 of the Ministry for the Environment's Health and Safely Management Plan for the FCC Site Remediation does refer to effective procedures. The wording implies that there are other procedures which are separate from the plan. This is, however, not the case. There are no additional procedures."*

In the November 2004 management meeting minutes there is a reference to previously undetected pesticides, herbicides and other hazardous materials being detected in the soils. The item was related to health and safety, regarding a peak that had occurred in the chromatograph test results which was not characteristic of anything that had been found on-site, but was thought to be phenothiazine. The MfE project manager stated that the issues would be dealt with as they came up, but would have to be accommodated "since the site may contain other surprises as far as chemical contamination was concerned". The MfE site manager stated that a standard operating procedure (SOP) would be developed to deal with materials as yet not found or characterised on the site previously.

There is no evidence that this SOP was developed.

The weekly site management meeting minutes of May 2005 (Page 18) have already been referred to and illustrate some of the difficulties that EDL were having in producing their health and safety Plans.

The EDL Site Induction document, undated but electronically tagged in September 2005, does contain an MSDS section.

The EDL health and safety management plan contains pro-forma hazard identification sheets but they are not completed. A job safety analysis sheet lists the plant operation hazards as: "Air quality, dust, fumes, digestive [sic], explosion of gas/fumes".

The recommended actions or procedures were "face masks must be worn at all times; full decontamination uniforms to be worn when plant operating; approved foam fire extinguishers to be in place; evacuation procedures to be in place."

Section 3.4.3 identifies hazard types, noting that chemical hazards "can affect the skin or the body by contact either through skin absorption, the digestive system or via the lungs if air is contaminated with chemical, vapour mist or dust. Note! POPs – Persistent Organic Pesticides, are DANGEROUS – ensure the correct PPE at all times."

Gaps identified

The principal evidence that a hazard identification process has been carried out is the table from the induction manual, later reproduced in the management plan with the addition of a "toxicity" attachment labelled E.

The table does identify some of the hazards on site, but is by no means comprehensive, as admitted by the caveat. There is little or no evidence that the significance of the hazards has been assessed: all the potential hazards on site are simply labelled as being "potentially hazardous" without any further qualification.

MfE's Health and Safety Management Plan Policy Statement (section 2) stated that hazard identification and control procedures existed but MfE could not provide copies of the procedures through the Official Information Act. An integral part of the health and safety plan was therefore not implemented.

There was a failure to consider individually and comprehensively the operational areas on site – the remediation area, the treatment plant and the laboratory. There was also a failure to assess the activities and processes within each area, identify the contaminants likely to be found, and determine if the hazards were significant or not.

The induction plan does specifically identify, in one sentence, the treatment plant hazards: "Dust and vapours from equipment, including that generated by the soil reactor, soil dryer, bag house and the pummel." However, it fails to state what the dust and vapours actually were and whether or not they were significant.

The main contaminants on site were known, but other potential exposure went unrecognised at this stage, firstly because of the nature of the site and its varying levels of contamination, and secondly because of the varying conditions under which the plant operated, including the significant temperature variations in the dryer.

Neither the laboratory nor the treatment plants had specific hazard identifications carried out, and only much later were laboratory solvents, process chemicals (the "proprietary mix" of chemicals and sand) and plant emissions recognised and assessed for significance.

Section 4 of the workplace induction document does state that:

> "A Hazardous Substances Register for these substances is maintained on site, together with a Material Safety Data Sheet for each substance. This includes a summary of Workplace Exposure Standards for the various contaminants, the physical and chemical properties of the substances, acute and chronic health effects associated with these contaminants, and first aid procedures to be followed subsequent to exposure to these contaminants."

Such an MSDS would be an adequate document on which to base a subsequent hazard assessment. It was not in itself a hazard assessment because the information was generic and does not apply to actual workplace conditions

Evidence indicates that MfE were aware of numerous hazards, including benzene, PCBs, dioxins, PM10, ammonia and other volatile organic compounds at various points in the project, but failed to include them in the monitoring.

Section 5.2 (page 35) of the MoH report specifically identifies other chemicals of concern that may have been discharged from the site. The summary and conclusions also include the following:

> *There is evidence that members of the public were likely to have been exposed to dioxins discharged from the site during the remediation. However, in the absence of exposure information from ambient air monitoring, along with the uncertainty regarding the dioxins emission rate used in the dioxins dispersion and deposition modelling undertaken by AES, no conclusion can be reached on the health risk, if any, associated with possible dioxins exposure (Section 5.2.5).*

> *It seems likely that low levels of ammonia discharged from the site may have caused some of the many odour complaints and symptoms of eye and throat irritation, but this level of exposure is unlikely to have caused lasting or serious adverse health effects (Section 5.2.1).*

> *PCBs and benzene are both chemicals of concern that the public may have been exposed to as a result of discharges from the site. However, there is no monitoring data for PCBs and hence no conclusion can be made on health risk (Section 5.2.7). For benzene there is evidence that benzene was formed in the dryer and also discharged from the MCD reactor. It was reported to the PRP that based on the concentrations detected in the carbon filter, the levels being discharged from the site may be well above the ambient air guideline value for benzene. Although subsequent testing using PIDs [photo-ionisation detectors] at various locations round the site were reported as not showing benzene apart from next to the pug mill, there was disagreement recorded in the PRP minutes over the accuracy of this testing. It is possible that the public may have been exposed to benzene arising from the site due to stack emissions, particularly during the period when the AECS was malfunctioning, and also due to fugitive emissions from the MCD reactor. No conclusion can be made on health risk due to the lack of robust monitoring data (Section 5.2.3).*

> *There is considerable uncertainty about the number of days the public was exposed to PM10 levels that breached the NES due to lack of robust*

exposure data and the non-representative location of the Tahi Street monitoring site for assessment of exposure for residents to the south of the site. However, it is likely that PM10 levels breached the NES on numerous occasions. Therefore it is likely that there was an increased risk of adverse health effects from inhalation of dust during the remediation. The lack of robust exposure data and health data means the extent of the public health risk cannot be determined with certainty but is likely to be low to medium.

The MoH report makes reference to:

.... non-compliance with some of the conditions in the resource consents that may have led to public health risk or compromised the ability to assess whether there was any public health risk. These included the following: the soil dryer did not have an automatic cut-off if the temperature at the dryer inlet exceeded 120°C, TSP/PM10 monitoring during the PoP trials/first month of operation was not completed as specified, the location of the ambient air monitoring sites were not as specified, dust was discharged from the site at levels that were at the very least offensive and objectionable and may possibly have been noxious, and the PRP did not meet at least quarterly during the first two years of the remediation. Other factors, such as the AECS and the process reagents used, were considered substantially different by the PCE from what was presented to the resource consent hearing in the 2003 AEE, meaning that these could also be considered non-compliance."

In summary, although there is a list of chemicals, there was no system for identifying all the existing or new hazards, nor was there documentary evidence of a systematic assessment of all the hazards. The MfE hazard ID for the Mapua site falls into the adequate basic category for the plant, and was inadequate for the laboratory.

The EDL plan is, at best, generic. It does not contain any information about specific hazards bar the fact that persistent organophosphate pesticides (POPs) are dangerous and that PPE should be worn. There are no notes about possible health effects or controls other than PPE. There are pro-formas for hazard identification, but in 2007 these should have been completed, with full details of all hazards. The statement that "POPs are dangerous" without any further detailed assessment indicates a lack of awareness of basic health and safety principles.

Although the MfE representative followed up on EDL's responsibilities to fulfil their health and safety requirements more urgency was required. Some procedures seem to have been missed out, for example those in the laboratory. A detailed analysis does not seem to have happened. Indeed it seems to have been avoided by defining the laboratory as being "off-site" based on a strict definition of the site boundary.

The EDL hazard ID was inadequate.

The HSE plan was adequate to a basic standard.

Implications of the gaps

As a result of the incomplete hazard assessment, there was no clear idea of what the significant hazards actually were, and when and where they were to be found.

The specific implications were that the solvents in use within the laboratory were not included in a hazard identification, which led to subsequent failure to put in place adequate controls.

The failure at this initial stage to identify the contaminants emanating from the process, their form (dust, gas vapour or otherwise) and whether or not they were significant – in other words whether they were likely to be a source of serious harm – led to exposures. These exposures included the products of the reactions of the proprietary mix of chemicals in use by EDL and the likely emissions from the reaction, which later proved to include volatilised pesticides and dioxins. Sand can pose a risk of silicosis if it is in excess of 0.2 mg/m^3 in air. The actual concentration is not known in this case, as no initial hazard identification was carried out.

As hazard identification is the basic step in controlling workplace hazards, this failure of systematic identification meant that existing hazards were unrecognised, and made the recognition of new hazards less likely.

There is specific evidence that this did happen. In November 2004, phenothiazine was identified by the contract lab in two "cells". This was a previously unidentified hazard, although it was listed in the FCC inventory and has health effects similar to organophosphates.

The circumstances were such that workers were exposed to an unknown range and level of chemicals, making it impossible to assess health risk. What is known, however, is that the majority of chemicals involved were significant hazards and the data regarding the effects of multiple chemical exposure is very limited. It is not possible to ignore a potential for future health effects in those who were occupationally exposed on the site.

Statutory requirements for hazard control

Sections 8 to 10 of the HSE Act describe the control techniques which are to be used to address any significant hazards which are identified. The hierarchy of control techniques is: Hazards to be eliminated (section 8), Hazards to be isolated (section 9) or Hazards to be minimised (section 10).

The hazard control process outlined in the HSE Act is a process of elimination, isolation and minimisation, but can also be considered from the point of view of source, path and receiver (Figure 2).

Dealing with the hazard at source includes elimination, substitution (for a lesser hazard) and isolation. Dealing with the path is also a form of isolation. Dilution

ventilation is a control method which is usually achievable by general workplace ventilation (open doors etc) without any more specific intervention. Where point sources of contamination are present, it is usual to apply local extract ventilation or LEV, which has to be designed for the particular source or contaminant. To be efficient it has to be adequately designed, usually involving flanges or a hood.

Figure 2. Movement of a contaminant from a source to a receiver with control techniques for each component (After Olishifski, 1988.)

Source	Transmission path	Receiver
Elimination	Shielding	Eliminate the need for the worker
Substitution	Increase the distance	Reduce duration of exposure
Enclose the process	Dilution ventilation	Enclose the worker
Change the process	Housekeeping	Change work process
LEV	Maintenance	Reduce number of workers
Suitable and sufficient maintenance programme		Education and training to alter behavioural influences
		Maintenance
		Personal protective equipment

Dealing with the receiver involves education and training to reduce exposure by behavioural means, and lastly, usually when residual protection is required, personal protection equipment (PPE) should be supplied.

PPE is usually considered for residual protection when other control methods have failed, because PPE use relies heavily on behavioural factors.

For an adequate basic assessment according to the HSE audit framework, the management plan should include:

> "Basic control such as general ventilation with provision of PPE for significant hazards. Limited but ad-hoc monitoring. Some significant hazards eliminated or isolated. PPE for all significant hazards."

An adequate fair plan would include:

> "Some significant hazards eliminated or isolated. PPE for all significant hazards. Targeted environmental and health monitoring".

On-site performance

Isolation (or segregation) is considered in section 7 of the induction plan, which deals with contamination zones and additional health and safety measures which might be required. The areas are an exclusion, or hot, zone, a contamination reduction zone (or corridor) and a support or cold zone.

Section 6 of the induction document does state that:

> "Site operations, procedures and work in general will be arranged or staged) so as to reduce or eliminate exposure to workers. Where this is not possible, site personnel are required to wear appropriate protective clothing and equipment in order to prevent injury or health effects from hazards on site. Protective clothing and equipment will be matched to the hazards encountered during a particular job or operation."

The selection of PPE is based on a "level of protection" (page 9), as follows:

Table 3.   Selection of personal protective equipment

Level of Protection	Equipment	Areas Required
Base Level	Steel-toed boots Hard hats Safety vests Hearing protection (if required)	Exclusion Zones and Contamination Reduction Zones (subject to results of air monitoring programme)
D	Coveralls with long sleeves, or Tyvek overalls, or Kleenguard coveralls Steel-toed boots Hard hats Hearing protection Safety glasses or goggles Face shield (optional) Gloves of leather or nitrile rubber	As above
C	Level D equipment, plus Half facepiece air purifying respirator, Type AB2, Class P2 cartridges, meeting AS1716 requirements	As above
B	Level D equipment, plus Full facepiece positive pressure-demand, self-contained breathing apparatus (SCBA), meeting AS1716 requirements **or** Full facepiece positive pressure-demand airline respirator, with a 5-10 minute escape bottle, meeting AS1716 requirements	As above

The induction document also states:

> "The selection of the appropriate PPE level will be based on the nature of the activity being undertaken. The level of PPE required may be upgraded if air monitoring results exceed relevant action levels. A requirement to stop work may eventuate should air quality monitoring results exceed relevant action levels. Some areas of highly contaminated waste will be identified, excavated and segregated within the exclusion zone. All personnel involved with the excavation, transport and treatment of contaminated waste stockpiles are required to wear Level C protection. Training will be carried out in the requirements of PPE during site induction and toolbox talks. Male personnel working in Level C or B environments are required to be cleanly shaven for the purposes of ensuring the proper

fit of respiratory protective equipment. All personnel are required to perform positive and negative pressure fit tests prior to entering a contaminated area requiring the wearing of respiratory protection."

Section 7 deals with site contamination and zones of protection. Areas of a site containing hazardous waste or contamination are divided into three control zones. These control zones included:
1. Exclusion or hot zone
2. Contamination reduction zone or contamination reduction corridor
3. Support zone or cold zone.

The purposes of establishing control zones were "to prevent the unwanted movement of contaminants from the site to uncontaminated areas. Further effort is made to reduce the possibility of contact with any contaminants present with personnel working on the site and finally removal of contaminants by personnel or equipment leaving the site."

Section 9.1 deals with dust control:

9.1 Contamination and Industrial Activity Control Measures on Site

There are a number of methods employed to eliminate chemical contamination from migrating from the site into the surrounding residential and business neighbourhoods, as well as reducing the impact of the site remediation including excavation and processing of soils and the treatment plant.

9.1.1 Dust Control

Contaminants identified in the Site Investigation and outlined in the Remedial Action Plan are present in varying concentrations of the soils at both FCC East and West. There may also be unknown or not identified contaminants most likely in the soil. It is critical that all site activities focus on eliminating or reducing the amount of dust produced on site and to prevent that dust from migrating off site. Measures include:

- Daily use of the site sprinkler system and water cart to wet soils at both FCC East and West prior to vehicle movement or excavation
- Covering of excavated stockpiled soils and excavations with tarpaulins
- Reducing the speed of vehicles including excavators, diggers, tip trucks and other equipment on site
- Travelling only on established roadways on the site and not cutting across cell areas
- Routine maintenance of the bag house and other filters at the treatment plant.
- Planning operations around inclement weather particularly high North East of North West winds
- Use of soil fixiants including shade screen, mulch or straw, or chemical non toxic soil fixers

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There were no control methods specific to the laboratory or processing plant. The EDL plan is once again generic. Section 3.3.2 states: "respirators are located in the site office and issued to individuals, and hung on the employee's labelled hook in the canteen. ... There are different masks for different jobs and the correct one must be worn. Respirators only work properly if they are correctly fitted and worn. If the mask becomes clogged and breathing difficult, replace it."

Gaps identified

The failure to carry out a comprehensive hazard identification means that all the hazards were not identified, and control mechanisms could not have been specific to the process or contaminant in question.

Most, if not all, laboratories will have fume cupboards or hoods to either isolate or minimise hazards from chemical vapours or fumes, and personal protective equipment will be available to protect the eyes and skin of laboratory staff. In this case, there was no fume cupboard, and no efficient form of ventilation. The ventilation fan high up on the wall would have almost certainly have been ineffective, and the arrangement whereby the fan was ducted downwards was designed by EDL and not close to the area where solvents were used. The fan reportedly reversed on windy days. A face velocity of 0.5 ms^{-2} is necessary for the capture of vapours, and the modification was highly unlikely to achieve this.

Initially, respiratory protective equipment was not available in the laboratory. It was made available later but would not have been necessary had a fume hood been installed.

Respirators were provided to lab workers in July 2006 as a reaction to their complaint of illness but no fit testing was performed and dust filters were not provided. Lab workers seemed to have been discouraged from wearing their masks by EDL management as they were being worn in public areas when going back and forth to the site. They were also wearing masks going to and from the on-site toilet so were denied access to this and told to use the facility in the MFE office further down the street. It appears that management felt that was "not a good look for the company".

Latex gloves, which were provided as PPE in the laboratory, are permeable to hydrocarbons, and are suitable to use with aqueous solutions, but not otherwise. They were not suitable for use with solvents.

Similarly, the failure to identify and assess all the hazards at the plant itself meant that the respiratory protection was not specific to the hazards, including sand and uncharacterised fugitive emissions from the process.

The selection of the appropriate PPE level was based on the nature of the activity being undertaken, and the level of PPE required was to be "upgraded" if air monitoring results "exceeded relevant action levels". A requirement to stop work "may eventuate" should air quality monitoring results exceed relevant action levels.

The EDL advice about PPE is incorrect. Respirators should not be hung on hooks in the canteen because they will contaminate the area and the harnesses will become damaged. The advice to replace the mask if breathing becomes difficult is dangerous (the advice should have been to replace the air purifying filter). The mask will become ineffective long before this.

At least two plant workers spoken to felt "overcome" by vapours in their workplace while wearing respirators. One of the EDL staff reportedly had a full beard for a period while working on the EDL "pad". This is not compatible with a well-fitted respirator. When respirators were eventually supplied to lab workers, no fit test or dust filters were provided.

Regarding the wearing of PPE, the 2006 Interim Medical Report also states:

> *"Protective Equipment: Even though the evidence does not come through strongly that wearing or not wearing a mask was critical, there was little emphasis on PPE wearing in general – a degree of casualness was apparent. The Kiwi "she'll be right" attitude prevailed. While it would appear that chemical exposure on this site was not a significant risk, this may not always apply."*

Figure 3.   An EDL worker, with PPE but not wearing it, at exit from the reactors

Although dust mitigation measures were specified, it was an ongoing problem and a possible health issue.

> *"It seems unlikely that dust emissions from the site were "minimised", and were likely to have been in breach of the conditions much more often than it appears from the record."* (PCE report)

Tasman District Council has an alternative view that the dust was minimised and that this was achieved to "quite a large extent". It does not necessarily follow

that a breach of the condition "dust beyond the boundary" (resource consent wording) did not mean that the condition to minimise using "best practicable options" was not undertaken. The issue will probably remain contentious.

The MoH report states that:

"… the discharged dust may contain a significant portion of PM10".

And:

"…dust was discharged from the site at levels that were at the very least offensive and objectionable and may possibly have been noxious".

"…it is likely that PM10 levels breached the NES75 on numerous occasions. Therefore it is likely that there was an increased risk of adverse health effects from inhalation of dust during the remediation."

Implications of the gaps

Exposure to contaminated soil (from soil samples and from wind-blown dust) and solvents took place in the laboratory with the dermal absorption route being the most likely source of significant exposure to solvents, bearing in mind that, initially, the gloves provided were inadequate.

The interior of the laboratory was exposed to the stack and reactors' emissions (gases vapours and dust) from the plant as they were downwind (northeast) of the plant. After lab workers were provided respirators in July 2006, one experienced nausea due to the strong odour of plant emissions while inside the lab with the door open for ventilation. The worker later found out that the supplied respirator was too large, so a smaller size was provided.

On another occasion, a worker is said to have been exposed to a great deal of dust while sweeping out the lab and was distressed that the respirator had failed to prevent this. The worker later found out that a dust filter had to be added to the respirator to prevent this problem.

It seems that, even after these control failures in the lab were pointed out to EDL, they failed to take action to correct them. In March 2005 the Cawthron Institute was commissioned to assess quality control of the gas chromatograph results in the lab. It was found that that ventilation was inadequate and dust was entering the lab from outside. Some important items of equipment were lacking and "the bulk preparation of the solvent mixture used for extraction is not satisfactory; the use of hexane in this way is also not desirable due to safety/volatility concerns."

At the April 2005 EDL management meeting the EDL CEO reported on the results of the Cawthron visit to the lab:

> *"Cawthron Institute had been on site to examine their lab procedures. Bryan summarized the results of the audit. It was reported that better calibration was required, extraction procedures were adequate, ventilation was inadequate and there were concerns about ambient dust entering the lab from the open doors and affecting results. [   ]indicated that a copy of the report had been provided to Site Management."*

No action was taken regarding the ventilation issue for nine months until the lab staff specifically brought the issue of solvent exposure to the notice of management.

Exposures on the plant were therefore taking place, but as these were not specifically identified there was no way to decide what protective equipment was needed. Environmental monitoring was identified as a method to raise protection levels, but the action levels were not identified, and in any case the monitoring method could not have identified significant exposures soon enough because of the delays in analysis and reporting. Significant exposures were therefore likely to occur and go unnoticed.

In summary, the MfE hazard control plan was adequate basic for the site and inadequate for the laboratory. The EDL plan was inadequate, showing a failure to appreciate some basic principles of occupational health and safety.

Hazard monitoring: statutory requirements

If significant hazards cannot be eliminated or isolated but they are minimised, then section 10 of the Act requires the employer to take the actions under subsection 2. This includes the requirements to monitor the employees' exposure to the hazard; to take all practicable steps to obtain the employees' consent to the monitoring of their health in relation to the hazard; and with their informed consent, to monitor the employees' health in relation to exposure to the hazard.

Occupational health monitoring therefore requires the hazard itself to be measured by the appropriate method. In this case, as most of the hazards were airborne, this means sampling for the substance in question to ensure that the levels are within the occupational exposure standards set. This monitoring must reflect the exposure of the individual. It may also be appropriate, if certain conditions are met, (principally that all hazards have been identified and controls put in place) to monitor the levels of substances actually in the body of an exposed individual. Failing that, the effects of some substances can be detected by monitoring the health effects in the individual, for example liver function.

Types of monitoring

The monitoring process for the occupational environment (as distinct from the general environment) may either be a part of, or follow on from, the hazard identification process. For chemical substances the hazard identification should identify the form of contaminant (vapour, dust or other form), the nature or chemical composition of the hazard and whether or not it is significant. There are Workplace Exposure Standards (WES) published by the Department of Labour for

many contaminants, and although exposures should be "as low as reasonably practicable", monitoring can identify the level of the substance in the atmosphere, and the need for control measures. It then forms a baseline data point for assessing control strategies.

There are many instruments available to carry out air sampling.

Direct reading instruments have an electrochemical sensor which provides a continuous reading on a meter. The sensors are specific to a particular contaminant (usually a gas or vapour) but some instruments, for example the photo-ionisation detector (PID), will detect total organic vapours. "Grab sampling" can be carried out by using detector tubes (Gastec or Draeger) through which the contaminant is drawn, the level during the sampling being indicated by a colour change in the detection medium.

The other option involves drawing the sample through either a detector tube or a filter using a metered flow pump.

Figure 4.   A "Draeger" sampling pump with detector tube

There is no single 'best' strategy for monitoring occupational exposure. In each case the sources of exposure, the characteristics of the contaminants, the location and work patterns of the worker and the reasons for sampling will differ. Constraints are also imposed by the methods of collecting and analysing samples, practical logistical problems such as the portability of equipment and cost.

The optimum strategy is the compromise which best combines the choice of method and sampling scheme (i.e. location, time and number of samples), to provide results which are adequate for the decisions which follow. In almost all cases, estimates of exposure which truly represent actual exposure should be obtained.

On a site such as Mapua, there are essentially two choices of location of sampling on an individual worker: personal sampling in the worker's breathing zone, and area or static sampling in a fixed location in the work area.

- **Personal sampling:** Personal sampling can be further subdivided into a true personal sample where the sampling device is directly attached to the employee and worn continuously during all work and rest operations, and a breathing zone sample where a second person holds a sampling device as near as possible to the worker's breathing zone. This second type of sampling is difficult to perform, but may be useful where a direct reading instrument can provide an instantaneous readout and show the exposure distribution as it relates to changing work procedures.

- **Area sampling:** Personal sampling is the only valid method of estimating personal exposure, and area or static sampling is the best method for obtaining information on the sources of emissions (which will generally be in an area where workers pass through) to direct control efforts. The focus is on the sources of contaminant emissions, and samplers are usually placed at fixed locations to monitor major emissions regardless of whether employees are normally present.

As a general rule neither method will give valid or useful information for the other purpose. There are, however, instances where, because appropriate personal sampling devices do not exist for all substances, it is necessary to conduct area sampling at the sites frequented by the employee and calculate time-weighted averages on the basis of the amount of time spent at each site.

Other elements of the sampling strategy also differ significantly for this type of sampling, for example length of sample time and pump flow rate will often be varied to reach a compromise between sampling identifiable production intervals and the minimum time required to collect sufficient sample for the detection limit of the analytical method.

Another reason for performing area sampling may be on-going surveillance of a process or operation which has been shown to be 'under control', i.e. it does not produce significant exposure when operating within known limits. In this case area sampling may be used to signal local increases in emissions, caused for example by leaking valves or a fault in a ventilation system, before they result in excessive exposure. But apart from this it should be remembered that results from area sampling will normally provide information which is of limited or no use for assessing employee exposure. This all requires careful planning.

Sampling strategies for personal monitoring
The two main methods of selecting the sample to monitor are worst-case sampling and random sampling.

- **Worst-case sampling:** Selecting the maximum risk or worst-case employee involves a subjective determination based on careful observation during a walk-through survey. A number of clues will generally point you in the right direction, but considerable care is needed. These clues include closeness to the source of emissions, time spent near the process when workers are mobile, and air movement patterns within the workplace –particularly where the work process itself contributes to these, e.g. a hot process or moving machinery. Differences in work habits of individual workers may also have a significant effect on levels of exposure experienced, even when performing essentially the same process. For example, welders adopt different postures subconsciously, either close to the weld and directly in the path of the plume of contaminants produced, or with the head held further away. The obvious advantage of worst-case sampling is the degree of certainty with which group exposures can be said to be "safe" when maximum-risk employees have been shown to have exposures within recognised standards.

- **Random selection:** Where there is any doubt about the ability to select maximum-risk employees to perform worst-case monitoring, then the only scientifically valid method is to select a sample in a purely random manner. Random sampling will also have application where employees have been grouped according to the similarity of exposures, and a sample from each group has to be selected for monitoring.

Number of samples
There is no set rule for determining the number of replicate samples required to fully evaluate a worker's exposure. A minimum number must, however, be taken to characterise the exposure in space and in time, and to provide the level of confidence required.

Regular monitoring is of diminishing value as exposures become further removed from the exposure limit. There is little to be gained, for example, from repeated measurements of exposure when contaminant concentration in the breathing zone is below one-tenth, or above twice the exposure limit. However, in either case a change of process, of materials handled or of the ventilation, would provide sufficient justification for further sampling.

Sampling in time
When to sample, and the length of time over which to sample, are also decisions that must be made in formulating a sampling strategy. The random environmental fluctuations of contaminant concentrations during the day can be very high (as in this case, with levels between 175 and 9700 ppm). The causes of this variability include randomly occurring fugitive emissions, changes in production rates, the distribution of contaminants by bulk flow, and uneven diffusion in both time and space. The worker moves in an often random and unpredictable manner within this work environment. This variability has important implications for sampling if a meaningful measurement is to be obtained.

Obviously any sample taken at a single point in time could give a result which may be almost two orders of magnitude (or 100 times) different from a sample taken at any other point in time. It is therefore not possible to draw conclusions from such a sample.

Day-to-day variations in exposures are also significant and should be accounted for in any sampling strategy. Apart from the random inter-day variation expected of any environmental exposures, there is also systematic variation as seasons affect concentrations.

So another aspect of sampling time to be considered in devising a strategy is the duration of the survey or the number of different days on which samples are taken. The inter-day variation may be as significant as the intra-day, and if one particular day's exposure was found to be acceptable it could not be concluded that all other days' exposures will be. The true daily exposure average is itself drawn from a distribution of all other true daily exposures over a period of time, and a sample taken on one day may have come from a low portion of the distribution.

The variation in the environment between days can only be established by the taking of a sufficient number of samples on different days to enable "power calculation" to be carried out. This is rarely done in practice because of the resources needed, but an appreciation of it should be incorporated into any interpretation that is made of the significance of any single day's sampling.

Biological monitoring

Biological monitoring is the measurement, in a biological sample, of a contaminant or metabolite of a contaminant to which a person is occupationally exposed.

Biological monitoring needs a set of values to act as a reference standard. These values are known as biological exposure indices (BEIs), and, like WES, they are published annually by the Department of Labour.

BEIs are used as a guideline in evaluating potential health hazards. They represent the levels of determinants that are most likely to be found in biological specimens collected from a healthy worker who has been exposed to chemicals to the same extent as a worker with inhalation exposed to the chemical at the WES concentration.

BEIs do not represent a sharp distinction between hazardous and non-hazardous exposures. Due to biological variability, it is possible for an individual's measurements to exceed the BEI without causing an increased health risk. If, however, measurements in specimens from a worker on different occasions persistently exceed the BEI, or if the majority of measurements in specimens obtained from a group of workers at the same workplace exceed the BEI, the cause of the excessive values must be investigated and proper action taken to reduce the exposure.

In practice the biological samples where the indicators may be determined usually consist of blood or urine. Which sample is chosen depends on a knowledge of the metabolism of an exogenous substance in the human organism, and of the alterations that occur in the critical organ.

Routine monitoring of exposure

For the routine surveillance of exposure to some substances, biological monitoring may be preferred over air sampling. If the substance has a long half-time in the body (for example lead), the biological monitoring assay will give a result that reflects an integrated exposure. In other cases the equivalent air sampling procedure may, because of the typical work practices or sampling difficulties encountered, give less reliable results.

With biological monitoring, information can be obtained that would not otherwise be available by environmental monitoring including:

- the evaluation of absorption and/or exposure over a prolonged period of time (not only of the amount of substance present in the working environment at a given time, when the analysis of environmental pollutants is carried out)
- the amount of a substance absorbed as a result of movements within the working environment or of accidental causes, which often cannot be checked (not only on the amount normally present in the workplace)
- the amount absorbed by the organism via various routes (not only via the respiratory route, as is presumed in environmental monitoring)
- the evaluation of the overall exposure, as the sum of different sources of contamination, which may also exist outside the working environment
- the amount absorbed by the subject, taken as an individual, as related not only to their workplace, but also environmental factors, the subject's particular way of withstanding physical effort, age, sex, individual genetic characteristics, the functional condition of the organs responsible for the metabolism and elimination processes, etc
- on whether the subject has been exposed to a risk which could not be proven in any other way and, in some cases, when.

Requirements for biological monitoring

Conditions necessary for successful biological monitoring include:

- the existence of indicators
- the existence of sufficiently accurate, sensitive and specific analytical methods that will guarantee technical reliability in the use of these indicators
- the possibility of using readily available biological samples on which the indicators can be measured
- the existence and knowledge of dose-effect and dose-response relationships
- if the assay is done at a remote laboratory, then the specimen must be stable in the biological fluid
- while the concentration of the major metabolite may be very high, and therefore easily detected, if it is a metabolite that is common to several substances the determination of the unaltered substance or minor metabolite may be preferable.

Biological monitoring and the use of the BEIs cannot be used as a means of worker surveillance when acute exposure conditions exist or when biologically inert substances, local irritants, infectious agents, allergens, mutagenic, teratogenic or carcinogenic substances are involved.

In situations where biological monitoring is applicable, then it is essential to use indicators with sufficient predictive validity. This is to say the validity of the test is the degree to which the parameter under consideration predicts the situation as it really is or as it would be using more accurate measuring instruments. Validity is given by the combination of two properties, sensitivity and specificity. If a test possesses a high sensitivity, this means it will give few false negatives; if it possesses high specificity, it will give few false positives.

On-site performance

General site environmental monitoring

The main form of environmental monitoring carried out was ambient air monitoring using high-volume sampling pumps. Initially these were aerosol (particulate) sampling heads, but polyurethane foam (PUF) filters were substituted later in order to measure volatile organic compounds (including gases and vapours). These were situated at three areas on site, one of which was located next to the laboratory. One was sited to obtain "background" levels. There were numerous problems with this monitoring, which are detailed in the PCE report.

The results were assessed using a total hazard index (THI). The ratio between the exposure estimate for a pollutant and its reference dose is the hazard quotient, and the sum of the hazard quotients over the range of exposures that occur is the THI. It should not exceed unity. Because not all contaminants were included, the THI is also technically suspect.

Stack emissions monitoring

Proof of performance plant testing was carried out in the early part of 2004. Dioxins were released from the stack in April, and high levels of organochlorine pesticides (OCPs) were also noted. This was due to the soil dryer operating at too high a temperature with volatilisation of pesticides and probable de-novo formation of dioxins. Between August 2004 (when the plant started up again) and January 2005 there was no stack monitoring. The emissions results over the period of operation showed variable results, with peaks in many of the compounds being tested: Aldrin, Dieldrin, Lindane, hexachlorobenzene, DDx and the metals.

The PCE main report refers to the stack emissions and the monitoring regime as follows:

> "The stack measurement emissions programme was designed to sample,
> at certain intervals, emissions from plant under normal working conditions,
> to test whether all the various components of the system were acting to
> prevent emissions of key toxins. The results were reassuringly low for the

substances measured, indicating that the THI estimate of toxins dose to people might give a reasonable picture of the situation from early 2006 onwards."

The Air Technical Annex provides further insights:

"From the chemical perspective, a combination of the hotter drier temperatures, insufficient characterisation and/or insufficient blending of the soils being run through the dryer, and the routing of the diesel exhaust through the system probably led to the unforeseen formation of large amounts of acid gases (probably SO_2 and HCl) as well as larger amounts of volatile organic compounds [VOCs] than expected. All these factors, along with poor management of the plant, contributed to the likelihood and potential extent of fugitive emissions."

And:

"The air emissions control system failed completely on a number of occasions and did not work effectively to control emissions during some periods of the consent. The extent of these emissions is uncertain, but is likely to have been higher than was discussed in the resource consent hearing. The effect on people and the environment cannot be readily determined from currently available data."

And:

"At various points in the project (e.g. at times during 2005) they were good indicators of problems occurring – so rising levels of OCPs (in large excess of those encountered in the Proof of Performance) in February 2005 indicated problems with the AECS."

Personal monitoring

A document requested under the Official Information Act does have a section ("Personal air monitoring"):

"5 Personal air monitoring

One of the greatest risks of exposure on site is from inhalation of dust contaminated with OCPs. To provide a check on the levels of risk originally assessed for the site and on whether the safety measures are adequate, personal air monitoring will be conducted.

5.1 Testing frequency

Personal air monitoring will be conducted very three months,

5.2 Testing methodology

Staff working in selected locations including excavations and processing plant will wear personal monitoring equipment attached to their protective equipment that will measure airborne OCP exposure. Monitoring equipment will also be placed in staff smoko rooms and inside work vehicles. Personal air monitoring will be conducted in accordance with the appropriate analytical method from the National Institute of Occupational Safety and Health (NIOSH) Manual of Analytical Methods (NMAM) or a suitable alternative.

5.3 Interpreting and action

The occupational physician will consider whether the levels of OCP detected from the personal air monitoring are cause for concern. If the occupational physician decides that measured levels of workplace exposure are of concern, then the site supervisor in consultation with the occupational physician will determine appropriate actions. These actions may include halting work onsite, increasing the amount or grade of personal protective equipment and conducting further blood tests.

After conducting the first round of personal air monitoring the occupational physician and the site supervisor will review the adequacy of personal protective equipment.

Personal sampling
There is information to suggest that a more detailed sampling strategy was proposed by the Occupational Hygienist [   ] in an undated document as follows:

> **"Purpose of workplace personal/area sampling**
>
> Workplace/area monitoring provides an opportunity for MfE to show a pro-active responsible approach to the potential hazards by actively seeking to define the workplace environment to a high standard.
>
> Use of Air Purifying respirators requires real time air monitoring under all workplace standards including NIOSH. Since there are 4 to 5 work faces on site where respiratory protection is required, to properly monitor these work areas will require multiple personal air sampling pumps.
>
> 1)   Personal:
> Determination of potential exposure levels during a working shift and comparison of those exposure levels with existing standards for dust and OCP in air
> Confirmation of the PPE requirements

Confirmation of the presence or absence of other contaminants such as Volatile Organic Compounds and Ammonia

2)   Area:
Determination of proper work practises to reduce personal and environmental contamination

3)   Community Complainants:
Reassure that home and/or personal exposure is minimal or define what the exposure may be.

Rational (sic) for a 3 pump sampling strategy:

Characterization of the workplace environment is best done by sampling multiple locations on the same day. With multiple locations sampling a measure of an emitting source i.e. the reactor or screening operation can be made in conjunction with a measure of the personal work shift exposure of at risk employees. Conclusions can then be drawn as to the potential level of exposure, the actual recorded level of exposure and possible work practise modifications that could reduce exposure based on the sampling results and the detailed record of work activities during the sampling period.

Single point sampling on separate days does not easily allow for this type of data interpretation. Instead, assumptions as to the homogeneity of the working environment over the different days have to be made and consequently any conclusions about work place exposure and work practises are much less robust. If multiple samples are attempted on the same day from a single sampling pump, sampling times are greatly reduced and consequently detection limits for the compounds in question may not reach the required standard for workplace exposure assessment.

The following is a list of potential sampling events which would fulfil the purposes outlined above:

HSE employee exposure characterization:

Once each quarter or more often if results or changes in work practises warrant it, when screening and excavating is occurring a personal sampling pump will be attached to-
the monitor/supervisor of the excavation;
another in the vicinity of the sceener and
another in the cab of the excavator.
In areas where no volatile organics compounds, VOCs are suspected the sampling train will consist of a preweighed filter cartridge which will be analyzed for total dust and OCP concentration and the results compared to existing standards for workplace exposure. If VOCs are suspected, an absorbent charcoal sampling tube can be placed in the line with the filter

cassette. Results can be related to each other as the sampling will have been conducted simultaneously.

It is anticipated that during operations at the landfill area a measure of the VOCs and the dust for other types of pesticides will be required to better characterize the exposure of the workers.

EDL employee exposure characterization:

Once each quarter or more often if results or changes in work practises warrant it, when the plant is in full operation a personal sampling pump will be attached to the-
Reactor near the vent shafts
Reactor near the mixing bowl
Plant personnel responsible for sample collection and maintaining the moisture content of the mixing bowl
Initially the sampling train will include a silica gel sorbent tube specific for Ammonia as that is the contaminant of interest at the moment. The results of this full day shift sampling will provide an indication as to the source and the concentration of Ammonia in the workplace air and the level of exposure of the plant personnel.

Community Complainants:

In response to multiple complaints from an individual concerning home air quality as a result of the project activities or pro-actively to reassure neighbours, sampling pumps could be placed
In the home
On the occupant
At the boundary of the site.
This type of sampling strategy would allow us to define the home environment and compare it with potential contamination coming from the site."

The document is undated, but includes costings.

Personal and ambient monitoring samples were taken in July, August, October and November 2005 during "a variety of different plant and excavation operations". A total of 23 samples were taken using PUF sampling filters for gaseous OCPs and 6 samples for total dust. These consisted of 13 personal samples (3 of these from the EDL plant engineer on a single day); 11 area samples (concentrating on 13 and 18 [   ] St and the EDL plant) and 5 field blanks.

The method is stated as:

"Gaseous OCPs were sampled using an MSA ELF low flow personal sampling pump set at a constant flow rate of 3 lpm utilizing the PUF filter sampling head manufactured by SKC as detailed in the above method. Total dust was sampled

either in series with the PUF sample as a pre-filter as per Method 10A or as a separate sample as a measure of total dust. Samplers were either attached to an individual worker during an entire work shift or portion of work shift or the samplers were placed in areas of special interest. This might include the cab of the excavator or fence lines of the site at 13 and 18 [   ] St."

The samples were taken "during a variety of different plant and excavation operations. The sampling included DDT, Aldrin, Dieldrin and Lindane (collectively ADL). With the exception of the Plant Engineer "during a day when he was working in extremely dusty and OCP laden atmospheres (baghouse maintenance), all samples were reported to have been below the detection limit of the assay."

The excessive dust required additional caution:

"Three separate samples were taken of the Plant Engineer's work shift due to concern of overloading of the sampling medium (i.e. PUF filter) from the excessive dust during his shift. It should be noted that the Plant Engineer was involved in a special maintenance project during the day of sampling involving repairing the bags in the bag house. Therefore, his results are not indicative of a regular work day. The dust in the bag house is very fine and highly contaminated with OCPs. During this operation the plant engineer was fully suited in a tyvek, boots, gloves and a full face piece air purifying respirator.

There is a possibility that the sample for the plant engineer was overloaded due to the high dust levels as there was evidence of breakthrough of the dust to the back filter. Taking the worst case scenario that the entire 99.3 ug of the sample during the worse exposure in the bag house was deposited in the first 15 minutes, the ceiling or Short Term Exposure Limit would be 2.31 mg/m3 which is still within the guidelines. The effects of the possible additional exposure by skin absorption are not measured by this monitoring. The effect of the combination of the exposures is seen from the biological monitoring."

The results were however reported conservatively, assuming the results were at the detection limit (generally less than 5µg). The results for the Engineer included values for pp-DDT of 5.7 and 6 µg for the first two samples and values for pp-DDE of 12µg, pp-DDD 8.3µg op-DDT 13µg and pp-DDT of 66µg. The samples were adjusted for total concentrations of DDX and working hours. The DDX Time Weighted Average (TWA) for the plant engineer was 0.091mg/m^3, the WES being 0.5mg/m^3. All the other samples showed undetectable levels of ADL. Total dust measurements were also obtained from 3 personal samples and one area pre-filter sample. The levels, between 0.06-0.96 mg/m^3 were below the relevant WES's (10mg/m^3 inspirable "nuisance" dust and 3mg/m^3 for inspirable dust (not containing free quartz).

Workplace survey, 3 August 2006

There is a report headed Fruitgrowers Chemical Remediation Mapua Air Sampling Event 3 August 2006 (Air sampling event). The instrument used was a photo-ionisation detector. The survey was stated to have four aims:

"The first was to measure volatile organic gases as part of its general three-monthly reviews of respiratory protection measures on site. This is a requirement with the use of air purifying cartridges in respirators.

The second was to use the PID to take air samples from several specific areas of the decontamination plant including the pug mill.

The third use of the PID was to conduct a general perimeter survey to determine whether or not volatile organic gases or vapours were moving beyond the site perimeter.

The fourth purpose was to survey the EDL off site soils laboratory at the corner of Iwa Street and Aranui Road at the request of the EDL site designated health and safety officer."

The PID measures volatile organic compounds (VOC) and other toxic gases in concentrations from as low as parts per billion (ppb). The detector is sensitive to a range of chemicals, and does not typically distinguish one chemical from another. The PID uses ultraviolet light to ionise the sample gas in positive and negative ions, which are then counted by the detector. The charged particles produce a current that is amplified and displayed on the meter in parts per billion (ppb) or parts per million (ppm).

The meter is calibrated against a known concentration of isobutylene, which has a baseline correction factor of 1.00. When measuring a single gas, a specific correction factor is applied to the reading to give an accurate record of the atmospheric concentration. For example, methyl bromide requires a correction factor of 1.70 to be applied to the displayed isobutylene equivalent reading to give the actual concentration in air. The difficulty arises when multiple or unknown gases are being measured. No single correction factor can be applied to the mixture, and the reading can only be expressed as a total VOC concentration.

The PID is useful if looking at one specific gas or vapour, for example ammonia or toluene, when a correction factor can be applied. It is not useful for chemicals adsorbed onto, or absorbed by, particulates as the reading will be erroneous.

PIDs can be run continuously, and the data logged, in order to assess the variability of emission levels, but in this case a series of "grab" or convenience samples was taken. Data logging was undertaken, but I have not seen any results.

The results are tabulated in the document as follows.

Table 4.   Photo-ionisation detector survey respiratory protector programme results

FCC East	Non Detect	
FCC West support area	Non Detect	
EDL Plant pug mill at the paella main mill.	175- 9700 ppm 200 ppm from a hot pile of residual treated fines.	.5 to 1 meter from the pug mill exit. Detection drops off dramatically past 1 meter from the mill exit.
EDL Plan pug mill at the Series III mill	135 ppm	Series III was not operating. Sample obtained approximately 10 cm from the surface of a residual pile of treated fines
FCC West Soil Storage Area Screening operation	35-50 ppm at storage pile M7/N7 24 ppm M8/M9 Non screened solid waste storage pile mixed with soil 10 ppm 10 cm from a CMT screened storage pile	
FCC West landfill and foreshore	Non Detect	
EDL off site soils laboratory corner of Iwa St. and Aranui Rd. Area surrounding the laboratory confined to a vacant lot holding the EDL off site soils laboratory (container) Area adjacent to the fire house just north of the laboratory	Non-detect at the GC MP, general area of the lab and sample preparation counter 75 ppm inside a beaker located in the fume hood containing a clear liquid with sample cells. Approximately 2 cm from the liquid surface. Non-detect 15ppm localized at a stained area of soil	PID set initially between the fume hood and scintillation bath. PID later moved by lab technician from the area and placed in a cardboard box immediately adjacent to the fume hood. Located next to a bottle of solvent. Primary solvents are acetone and hexane Appears to be a localized spill of and unspecified solvent material.
Head Space sampling from a specially prepared sample jar used by MfE	150 ppm + from the head space of approximately .25 liters at the top of a sample jar.	Sample jar was drilled with a sample port installed. Fresh sample from the pug mill was placed in side and set for 30 minutes before it was sampled with the PID.

The commentary is included below:

Pug mill at the end of the paella mill reactor

The PID indicated organics at approximately 0.5 to 1m from the outlet of the pug mill varying from 175 ppm to 9700 ppm. The PID does not identify or differentiate what organics are present, but based on observation and odour, at a distance from the mill, we could pretty well stipulate that the organic is ammonia from the decomposition of urea that is used as a reagent in the paella mill. The workplace exposure standard for ammonia is 25 ppm (NZ standard WES). Thus I have continued issue of a site safety notice in the "Weekly Planning Document" and at our safety meeting that anyone working in that area must wear respiratory protection. (They are doing this on the plant pad.)

The output augers from the top and bottom mill were surveyed from the exit of the augers from the paella mill to the connection with the pug mill. At several small 8mm bolt openings, the PID registered 135 ppm. This was also seen at the base of the augers as they output from the top and bottom paella mills. The infeed belt inside the poll bard was sampled, with the PID registering approximately 25 to 75 ppm from untreated material moving toward the primary dryer. The output pug mill for the Series III reactor was not operating but the residual pile of treated fines at the base of the mill was sampled. This area registered 130 ppm 20 centimetres from the surface of the pile. The area of the soil storage silos, perimeter of the dryer and the baghouse were surveyed approximately one metre from the equipment with negative results.

Laboratory sampling
The results from the lab survey are as follows:

Non-detect at the GCMP, general area of the lab and sample preparation counter. 75 ppm inside a beaker located in the fume hood containing a clear liquid with sample cells. Approximately 2cm from the liquid surface.

PID set initially between the fume hood and scintillation bath. PID later moved by lab technician from the area and placed in a cardboard box immediately adjacent to the fume hood. Located next to a bottle of solvent. Primary solvents are acetone and hexane

"Head space" sampling was also carried out, from a "specially prepared sample jar" containing a fresh sample from the pug mill. The VOC was 150 ppm in this head space.

Gastec ammonia and benzene monitoring, June-August 2006

A further survey was carried out at the request of the Peer Review Committee by MfE using Gastec benzene and ammonia dosimeters. These dosimeters are glass tubes containing reagents which detect the presence of benzene and ammonia.

The manufacturer's instructions say:

> "Place the tube on a worker near the breathing zone, except when measuring carbon dioxide (see second Tech Tip below). No pump, chart, or analysis is needed. Read the printed calibrated scale on the tube for exposure measurement in parts-per-million-hours (ppm-hours). Divide the reading by hours sampled for ppm levels."

Benzene monitoring was carried out at the EDL plant pad adjacent to the main soil dryer and pug mill, the [   ], [   ] Street and EDL laboratory air monitoring stations and the support area at FCC West.

Ammonia monitoring was carried out in the laboratory, the pug mill, the poll barn near the dryer and the support zone at the EDL smoko.

The tubes were left in location for 10 hours and read at intervals, then left for 60 days and "occasionally monitored".

None of the benzene monitors recorded any activity.

Average readings for ammonia were 175 ppm at the pug mill. No ammonia was detected at the dryer or in the laboratory.

The recommendations were that ammonia exceeded the WES, and that respirators were required in all areas of the EDL pad. Future work planned included further monitoring for organic vapours with passive absorption samplers and also personal air monitors (Elf Escorts). There is no record of this monitoring being carried out.

Gaps identified

An important point to grasp is that general environmental monitoring is not the same as occupational monitoring: the two have different methods and aims, and the former cannot assume the role of the latter.

The environmental sampling that was carried out does give a reasonable estimate of air concentration of contaminants for resource consent purposes. It also gives exposure information for the local community. Workers are, however, exposed differently, because they are closer to the sources of contamination, more likely to be exposed in their breathing zone, more likely to be exposed to fugitive emissions and less likely to be subject to a dilutional or ventilation effect.

The environmental sampling carried out did give some information about these exposures. However one of the main gaps was that the monitoring did not give timely and adequate information about the activities and conditions giving rise to

hazardous exposures. The opportunity to identify discrete "high risk" conditions and work activities was lost.

A further technical difficulty with the occupational environmental monitoring for pesticides at Mapua was the range of substances present, differences in form (dust or vapour) and the likely mode of exposure (respiratory or skin), all requiring different forms of monitoring. Air monitoring alone is insufficient, because dermal exposure to pesticides may have been significant which requires a different form of assessment.

Biological monitoring has the advantage that skin and inhalation exposure are integrated. Providing that the pharmacokinetics are known, a dose can be estimated and multiple exposures considered using suitable methods. This is why biological monitoring through blood or urine sampling, in some cases along with health surveillance, are the most preferred methods, provided that there is no likelihood of acute exposure. Unfortunately acute exposures (to fugitive and stack emissions) did seem to be occurring.

The acute and changing nature of the exposures meant that biological monitoring was not the method of choice for identifying exposures. The method was subject to the constraint that the activity and conditions giving rise to the high levels found in individuals were subject to a delay in analysis and reporting and were remote in time from the exposure. This can be seen where the baseline levels of a contaminant were compared to results measured months later. There was no opportunity to influence the exposures in the meantime.

Environmental monitoring
The USA Environmental Protection Agency Method TO-4 is an accepted method of environmental monitoring for pesticides. This captures contaminants in both the particulate and the vapour phase through the use of a combination sampling head (or combination technique) with a particulate (usually glass fibre/quartz) filter and a polyurethane foam (PUF) filter to absorb vapours.

The TSP reference method is also used as a reference standard for sampling procedures, calculation and data reporting, maintenance, and the assessment of data for accuracy and precision.

At Mapua, as stated in the PCE report (see the information below), this method was only loosely followed because the PUF filters were used to measure particulates, over-estimating the smaller particles and under-estimating the larger. The samplers were also run for much longer than intended; there were no blanks for reference purposes; only a proportion of the sample was actually analysed; the suite of chemicals that was measured was limited; and there seemed to have been a degree of dissent as to where the sampling units in the laboratory were to be sited. The report states that the samplers were initially sited by the supervisor but later moved by the employee. There has been conjecture in various subsequently commissioned reports about the effect of all these factors, but the methods are there for a reason, and the results are, strictly speaking, not valid.

The full details are given in the appendix to the PCE report "Investigation into the remediation of the contaminated site at Mapua", the Air Technical annex to which states:

a. *The limited range of the substances measured means that we cannot rule out the fact that people may have been exposed to a range of toxins, most notably dioxins as well as mercury compounds, especially between September 2004 and November 2005.*
b. *The design and management of the plant meant that from June 2004 until November 2004, the risk of the generation and emission of a range of toxins, most notably dioxins, was elevated.*

The MoH report also states:

"As well as the limited range of chemicals included in the monitoring programme, there were methodological issues in the ambient air monitoring undertaken which affected the data being used for the THI. Adjustments have been made in the revision of the THI calculations since completion of the remediation to address these issues, but the quality of the data weakened the value of the THI for monitoring immediate risk to public health during the remediation. Further, the location of the monitoring sites did not adequately assess risk to the public south of the site and also appears to be contrary to the resource consent conditions (Sections 5.1, 5.2.2 and 7.1)."

Stack sampling was also carried out, but once again this was technically compromised. In order to influence the process conditions, monitoring would have been required in "real time". With the technology available at the time this was not possible.

Occupational environmental monitoring

The occupational monitoring strategy for the PUF sampling carried out for the site manager, engineer and laboratory worker are not stated, but would neither have been worst case nor random. The former would have selected environmental conditions (with strong winds or in "dusty" conditions). The latter mode of selection would not have been truly random but was obviously made on some unstated criteria of selection by the plant management. This, along with the non-standard method that was used, limits the utility of this monitoring to the extent that it would have been of no value in assessing exposure apart from the day in question.

The other occupational environmental sampling carried out was with a PID to assess the ammonia in the plant area (specifically the pug mill) and for VOCs in the laboratory.

Although ammonia would have been detectable at between 1 and 20 ppm (normal variability in the threshold of detection) it does not mean that other organic substances could not have been present, leading to the high variability (175-9700) in the PID readings. As a result of the variable nature of the feed and

the "proprietary mix of chemicals" in the MCD process, very little is known about the nature of fugitive emissions from the plant and from the pug mill.

The laboratory sampling might have been useful had the details of the sampling strategy been specified and the logging carried out over a more prolonged period, with personal sampling. Other significant limitations were that acetone and hexane were not specifically targeted, but some chemicals such as HCB, OCP and benzene were monitored (by PUF sampling), which would not be expected Also, workers were not consulted prior to the monitoring to get information about their practices, i.e. number of samples per day and volume of solvent used, leading to incorrect assumptions about work practices. Non-standard methods and devices were used for this monitoring.

These aim of these August 2006 tests – performed by MfE to "survey the EDL off-site soils laboratory at the corner of [   ]Street and [   ] Road at the request of the EDL site designated health and safety officer" – were to follow up on the laboratory workers' complaints about ill health reported in July. There is no evidence that this type of monitoring was conducted at other times between September 2004 and July 2006. When one of the concerned workers asked why there had never been any monitoring in the lab previous to 2006, the MfE health and safety representative is reported to have said "because you're off-site", implying that the lab did not "qualify" for any. On the other hand an interviewee familiar with the site and personnel maintains that the occupational hygienist did visit the lab. The laboratory staff maintain that the MfE site health and safety representative made no assessment of the lab, or indeed visited it, in the 2½ years prior to this event.

Another EDL worker, who was working in the shed and dryer area, known to be an area where hazards were particularly significant, does not remember being the subject of personal monitoring in three years. Evidence indicates that the other plant workers were provided with only four sessions of monitoring over the course of the project, not every three months as specified in MfE's health and safety management plan. This was insufficient, based on the continually changing conditions and hazards.

Implications of the gaps

The failure to carry out adequate monitoring of the occupational environment meant that workers were exposed to a mixture of poorly characterised toxic contaminants at largely unknown levels. Although ammonia may have been a primary contaminant by odour, it does not exclude the presence of other substances.

Based on prevailing wind direction and deposition modelling undertaken for the MoH report, EDL workers, including the lab staff who were downwind of the plant, would have been subject to some of the highest exposures of environmental toxins (gaseous and dust) emitted from the plant.

Evidence from later sources (MoH report, 2010) indicates that MfE were aware of numerous hazards, including benzene, PCBs, dioxins, PM_{10}, ammonia and other

volatile organic compounds at various points in the project, but failed to include them in the monitoring. The report makes a number of references to the subject:

> *"The lack of information on the possible by-products of the mechano-chemical dehalogenation (MCD) process meant that there were notable omissions to the range of chemicals included in the monitoring programme that may be associated with public health risk, particularly ammonia, arsenic, benzene, chlorobenzene, dioxins, mercury, PCBs and two isomers of DDX"*

> *"The limited range of chemicals included in the monitoring programme, methodological issues in the ambient air monitoring undertaken which affected the data being used for the THI, and lack of representativeness in the location of the monitoring sites have compromised the ability to assess health risk for the public during and subsequent to the remediation. There appeared to be a lack of appreciation on the part of the Ministry for the Environment (MfE) as resource consent holder that the purpose of the THI was not primarily as a compliance tool but was to assess public health risk. There is evidence that members of the public were likely to have been exposed to dioxins discharged from the site during the remediation. However, in the absence of exposure information from ambient air monitoring, along with the uncertainty regarding the dioxins emission rate used in the dioxins dispersion and deposition modelling undertaken by AES, no conclusion can be reached on the health risk, if any, associated with possible dioxins exposure (Section 5.2.5)."*

> *"PCBs and benzene are both chemicals of concern that the public may have been exposed to as a result of discharges from the site. However, there is no monitoring data for PCBs and hence no conclusion can be made on health risk (Section 5.2.7). For benzene there is evidence that benzene was formed in the dryer and also discharged from the MCD reactor."*

> *"There is considerable uncertainty about the number of days the public was exposed to PM_{10} levels that breached the NES 75 due to lack of robust exposure data and the non-representative location of the Tahi Street monitoring site for assessment of exposure for residents to the south of the site. However, it is likely that PM_{10} levels breached the NES75 on numerous occasions. Therefore it is likely that there was an increased risk of adverse health effects from inhalation of dust during the remediation. The lack of robust exposure data and health data means the extent of the public health risk cannot be determined with certainty but is likely to be low to medium."*

The MoH report makes reference to "non-compliance with some of the conditions in the resource consents that may have led to public health risk or compromised the ability to assess whether there was any public health risk." Most notably, "the

soil dryer did not have an automatic cut-off if the temperature at the dryer inlet exceeded 120˚C" and "dust was discharged from the site at levels that were at the very least offensive and objectionable and may possibly have been noxious". "Other factors, such as the AECS and the process reagents used, were considered substantially different by the PCE from what was presented to the resource consent hearing in the 2003 AEE, meaning that these could also be considered non-compliance."

There are particular instances of failure to monitor, for example when phenothiazine, a previously unidentified chemical, was found on site.

The lack of a complete monitoring suite and the method of sampling – with the delay in the results becoming available (in the order of weeks or so) – meant that the results could not, as stated in the management plan, have informed the use of protective measures and triggered an increase in the levels of PPE. Firstly, although action levels were mentioned in the management plan there is no evidence of their existence. Secondly, by the time that the monitoring results were available the exposures would already have taken place, with the conditions giving rise to them going largely unrecognised. This is why biological monitoring cannot be used to assess acute exposures. The monitoring regime was more suited to a well-established process with much less dynamic conditions and stability in feedstock and processing conditions.

The statement in the air sampling event report: "we could pretty well stipulate that the organic is ammonia from the decomposition of urea" is therefore misleading. These high levels should have led to steps to identify exactly what the organic contaminants were, and should have led to a review of the control methods.

This "grab sampling" strategy obviously identified a problem, as stated in the PCE report:

> *"Within the MCD, there are two main sources of emissions – one from the soil dryer, and the second from the reactor itself. Both have short and direct paths to the atmosphere (via screw conveyors), although the reactor seems to have a design feature enabling conveyors to act as devices to relieve transitory air pressure rises in the reactor. In terms of emissions from the reactor or the dryer, under expected operating conditions the design of the system would seem to minimise any emissions that could occur from the reactor except via the AECS. Under normal operation, about 2m³s⁻¹ of air was passing through the system and the flow was fan forced (the fan was at the front end of the process). In addition, on the far side of the bag-house there is an additional fan to induce flow. However, in the first year of operations there were back-pressure issues within the system, where the resistance of the AECS was such that the pressure within the system increased. This seems to have been caused by the substantial back pressure from the baghouse and carbon filter. In this situation, air can be forced back through the dryer. Under these circumstances, it is likely that fugitive emissions from the*

dryer could have occurred. These emissions would likely have been discharged into storage shed ST120. It is also worth noting that the 'effective seals' between the storage shed ST130 and the dryer do not mean 'pressure tested'. So some of these fugitive emissions from the dryer may have been emitted into the open. No routine measurements seem to have been taken in these areas (particularly important in the first year of operation) to be able to check the extent of these emissions."

The lack of a system to deal with new hazards led to some recognised, and most likely some unrecognised, exposures. In November 2004, phenothiazine was identified by the contract lab in two remediation "cells". This hazard was not formally identified, even though it was listed in the FCC inventory and has health effects similar to organophosphates. Workers were not informed of this.

Although experts were involved in other aspects of monitoring, there is no evidence that an occupational hygienist ever reviewed the plans, assessed the site or advised on any aspect of the control or monitoring techniques. This was a crucial shortcoming, as workers on site believed that their exposures and health were being monitored adequately, when in fact they were not. There is also no evidence that air monitoring results were discussed with the workers or occupational physician, as stated in the health monitoring policy, or that the results were used to take action.

Biological and health monitoring

Biological monitoring is a useful adjunct to environmental monitoring in that it takes into account all routes of exposure (skin, ingestion and inhalation) and individual variability of exposure. It can also detect when minimisation procedures, particularly the use of personal protective equipment, has failed. The requirements for successful biological monitoring are described on page 42.

Health monitoring is an allied procedure in that it monitors the function of the target or end-organ system, or systems, affected by the exposure and may give an early warning of excessive exposure.

Biological and health monitoring performance on site

The undated MfE management plan document (sections 5.4 and 5.5) contains the plans for medical examinations and health monitoring, further qualified by information in attachment D, the health monitoring record. The management plan states the following:

> ### *Section 5.4*
>
> *Medical exams – all employees who are expected to work on site for a period in excess of one month over the duration of the contract must complete a medical examination.*

Prior to beginning work on the site, all workers will be examined by an occupational physician to assess their current (baseline) state of health and fitness to work. The medical exam will include: complete physical including assessment of limbs, respiratory function and fit test (repeated every six months), establish baseline biological indices in sec 5.5, eyesight, colour blindness and hearing."

Exit medical exam – once workers have finished working on site, an exit medical exam will be conducted. At this time the occupational physician will decide whether follow-up questionnaires and future medical testing are required.

"Fitness for duty" reports prepared by the occupational physician will be forwarded to MfE site rep prior to commencing work on the site.

Section 5.5

Biological monitoring will be undertaken to check for any effects on workers from

OCPs. This will involve full blood counts, testing for levels of OCP in blood, renal (kidney) and hepatic (liver) function.

Workers will be tested before starting and after finishing work on site (during medical examinations), and every three months in between.

Blood tests will test for all OCPs that are likely to be present on site.

Biological monitoring may also be required to check for any effects on workers from OPPs, organophosphate pesticides. This blood test monitors the cholinesterase activity of the worker which is directly affected by exposure to organophosphate pesticides, OPPs.

The occupational physician will liaise with the site supervisor and testing laboratory to arrange for tests to be taken at appropriate times in the work cycle and to ensure samples are stored and transported correctly.

The OPP monitoring was added in August 2005, when it became clear that these were to be found in the landfill part of the site. A memo at this time stated that this testing would be carried out and funded on an initial basis by MfE. EDL

indicated that it would not pay for this additional testing and would be submitting a variation for the blood testing already carried out.

Medical examinations

It appears entry medicals began in late 2004 and were only provided to selected workers and management. Some of the workers, including lab staff, did not receive an entry medical exam. There is no evidence that "fitness for duty" reports were ever issued to workers, contrary to a requirement in the plan.

The health surveillance programme was not ready at the start of the project. The biological monitoring did not begin until February 2005, six months after the project started. This means that it was not "pre-employment" surveillance for some workers. One worker had already been on site for 14 months when the February tests took place, several other workers had been there for almost five months.

Similarly, MfE did not provide baseline organophosphate tests as specified in their plan. These were suggested as being necessary in August 2005, as above. There do not appear to be any records kept of worker health monitoring to ensure everyone received all the tests.

An occupational physician (by definition a specialist physician so registered by the New Zealand Medical Council) was engaged, and an independent occupational health provider, Ramazzini, was contracted to MfE for "day to day" clinical occupational health management, (for example to arrange for medical examinations and blood tests). There seems to have been a degree of confusion as to the medical role and what the monitoring was about. The site management minutes (March 2006) record that: "[   ] provided details on changes to the Occupational Health Programme now that [   ] has left Ramazzini". [   ] is talking with the occupational physician, who will be on site 16 March to understand the work aspects. The role of Ramazzini is also being defined. There has been little study on OCPs and what is [sic] deemed to be high levels". The occupational physician reported on his visit and the OCP monitoring in June 2006 (q.v.).

The occupational physician was officially contracted to MfE from 13 March to 31 August 2006 only. There was no site physician contracted to the project for the last 16 months. It is uncertain how results of OCP blood tests would have been assessed at other times. Attachment D specifies the frequencies of, and responsibilities for, testing. OCPs were to be monitored prior to starting work, every three months during employment and at termination of employment. OPPs were to be monitored prior to starting work after any known intense exposure and at termination of employment if required. Lead was to be measured prior to starting work, every three months during employment and at termination of employment. The responsible persons were the supervisor/occupational physician. There is limited evidence that that this schedule was actually adhered to, but this cannot be assessed, as participation would have been voluntary.

MfE apparently contemplated another type of biological monitoring in 2006 for laboratory workers after they voiced concerns about solvent exposure. Based on

those concerns, MfE wrote in their monthly report (January 2006) that "the option of biological monitoring was explored for the hexane exposure but found to be unavailable in a reasonable timeframe."

Evidence indicates that EDL workers had their OCP blood results held up on more than one occasion. At the July 2006 site management meeting this was minuted as being due to "... non-payment of invoices by EDL relative to the blood testing". The length of the delay is not stated but a debt collection agency had taken over the resolution of the issue. The most notable example of delay was that the May 2006 test results were not received by workers until October 2006. It is difficult to say whether this delay would have had an effect on the test results themselves, but the delay in interpretation would have made them of no value in hazard management and control. (Management meeting minutes – June, August, September 2005, March June, July, September 2006)

One of the managers at the site brought up an issue relative to the personal monitoring. He wanted to know what became of the results provided to the employees and if employers would be able to get a copy. Another stated that the results for the general blood work were provided to the employee and the employee's designated doctor as well as held by the occupational physician. The results of the OCPs in the blood for the 9 February test had been received and the results of the May test were expected mid-June. The occupational physician had been developing a cover letter to assist the individual employee in the interpretation of the results. Discussion ensued as to the distribution of the results to the principal and the employers. The employee indicated that the current medical information release document, which had been signed by all employees, allowed for the release of an anonymous list of the results to the principal. It was requested that the site management team provide a memo on the biological monitoring programme and the rationale for the current frequency of sampling

Another manager expressed concern about the high costs of the analysis and wanted to know where the requirement was for him to pay for the lab costs. [   ] indicated that it was each employer's responsibility as part of risk management to ensure the health and safety of their employees was covered. [   ] wanted to make sure that the document that the site management team was developing can indicate what each employer needed to do satisfy their responsibility. [   ] stated that he wanted to know what the blood information meant. .... [The occupational physician] will be asked to provide some interpretation on the results. Minimally the site management team will provide some details on the tests and general results.

Results of health monitoring

Acetylcholinesterase (AChE) monitoring

The monitoring of AChE is, an established and well-recognised form of health monitoring for the effects of OPP exposure. Some initial tests appear to have been carried out, but there is no record of these on file.

Routine blood tests

There is evidence that routine blood tests were carried out at periodic intervals. However, only the results from the individuals on site who were interviewed were made available for review.

OCP blood tests

The results of tests are on file from the 11 February 2005, AqriQuality Lab reference 4894 (21 tests); 21 February 2006, AqriQuality Lab reference 11265 (lipid and non lipid adjusted) (four tests); 20 September 2006, AqriQuality Lab reference 17368 (two tests); and 11 October 2007, AqriQuality Lab reference 31131 (two tests). Thirty employees had at least one test, four had two, and two had tests on a third and fourth occasion.

Some discussion of the tests is available in the monthly reports.

In June 2005:

> "Collation of the OCP in blood results has been completed by the Occupational Physician, Dr Ryder, and all results have been distributed to individual employees. Collated anonymous results for all employers have not yet been released to the various companies involved. This is expected to be accomplished within the next month. Dr Ryder has not indicated to any of the companies that there is any particular concern with the body burden of OCPs for any individual or worker group as evidenced by the baseline tests in Feb and the 3 monthly test in May The next blood test for the workers is scheduled for the week of 8 August 2005."

In September 2005:

> "The results for the August round of blood tests were available this month. However, the schedule of the Occupational Physician has not allowed him sufficient time to collate these results and provide feedback to the individual employees. The results have been made available as anonymous data to the Management of each company and the EMS Site Management Team. A trend of increasing blood levels for pp DDT was observed throughout the workforce.   This trend may not be significantly greater than the potential error in the analysis and further investigation by the Occupational Physician into its significance will be necessary. The Management Team, however, has responded to this potentially significant increase by retraining staff in the use of PPE, increased vigilance in PPE use, designation of higher levels of PPE for more areas of the site, improved cleanliness in lunch room and toilet block and requiring that employees do not smoke while working in the contaminated areas. These measures address the areas over which individuals have some control. These measures do not address the plant failures during this period which may have contributed to this increase. It is expected that the complete

retooling of the plant with the improved safety features and the improved air emission systems will contribute to a reduction in exposure for the workforce in the subsequent 3 month period."

In November 2005:

"This month was the 4th round of biological monitoring sampling. In all, 22 employees provided samples for analysis of liver function, cholinesterase function, OCP concentration and other general health parameters. The results of this sampling, excluding the OCPs, were provided to the employees within 2 weeks of the test which was a good improvement of service delivery. Further analysis of the results to ensure that employee cholinesterase activity has not reduced during this period of exposure to OPPs has been requested. Initial OCP results are expected before Christmas but complete results and analysis will be available in January.

OCP and other blood parameter results for the August test were also released this month by the Occupational Physician. A trend of increasing DDT levels over most of the workforce was observed. Site management had been aware of this trend from the preliminary results received in September and had ensured that work practices were improved and that all employees remained vigilant concerning potential avenues of exposure. The trend was evident in this series of samples but also a trend of great variability in results between sampling events was also observed. In discussions with the analytical lab, it was determined that there is a 30% error possible in the analysis. Discussions with the Occupational Physician concerning the need for determining potential action levels for increases in the blood serum of the workforce were also initiated this month. Further research is being undertaken with national experts."

The November 2005 monthly report states:

"4.3 … a trend of increasing DDT levels over most of the workforce was observed. Site management had been aware of this trend from the preliminary results received in September and had ensured that work practices were improved and that all employees remained vigilant concerning potential avenues of exposure. Discussions with the occupational physician concerning the need for determining potential action levels for increases in the blood serum of the workforce were also initiated this month. Further research is being undertaken."

In the December 2005 – January 2006 monthly report:

"The site uses a survey conducted of a cross-section of residents in New Zealand which indicates an "average body burden" of OCPs in the general population. Action levels are compared to combination of OCPs found in workers during their pre-employment baseline biological sampling (pre-

site historical exposure) and levels found in the general population. Health professionals have established that when an individual worker exceeds their baseline by 25% their particular case will be reviewed and work practices evaluated, including work area, personal protective clothing, respiratory protection and general hygiene".

The unsigned "Health and Safety for Dec and Jan 06" states the following:

"The results of the November blood monitoring tests were available to staff by the end of January. Currently there are 23 staff on the 3 monthly tests of whom 15 have been on the program since Feb 05. A potential trend of increasing body burden of on site contaminants of workers with the longer work history on site has been observed. Site contractors have implemented programs to reduce or eliminate this trend. Some the methods have involved switching to respirator cartridges which filter out a greater variety of compounds; ensuring all skin surfaces are covered while working in contaminated or potentially contaminated zones and improving the hygiene within various plant operations areas. The Occupational Physician has been retained to closely monitor the situation. Action levels will be based on the individual's own baseline levels and rate of increase."

There is no documentation of how the 25% elevation from baseline was selected as an "action value". The site occupational physician was not aware of it.

The statistics of the first, baseline, test are shown in the table below, and, for those POPs within the 0-60 $\mu g.kg^{-1}$ range, as a box plot in the figure.

Table 5.   OCP statistics test 1

OCP	Number detected	Mean	Min	Max
α HCH	13	1.94	0.16	8.30
ß HCH	21	13.26	1.60	55.00
HCH	21	5.06	1.10	18.00
HCB	21	17.89	9.60	37.00
Aldrin	2	15.85	5.70	26.00
Dieldrin	19	21.69	7.00	65.00
Heptachlor	15	1.76	0.23	10.00
Heptachlorep	5	11.50	2.20	21.00
alphachlor~e	17	4.85	1.40	19.00
ppDDE	21	891.90	300.00	3100.00
ppTDE	20	1.84	0.20	6.30
opDDT	19	7.70	0.59	26.00
ppDDT	21	39.73	4.70	160.00

Figure 5.   Results of test 1
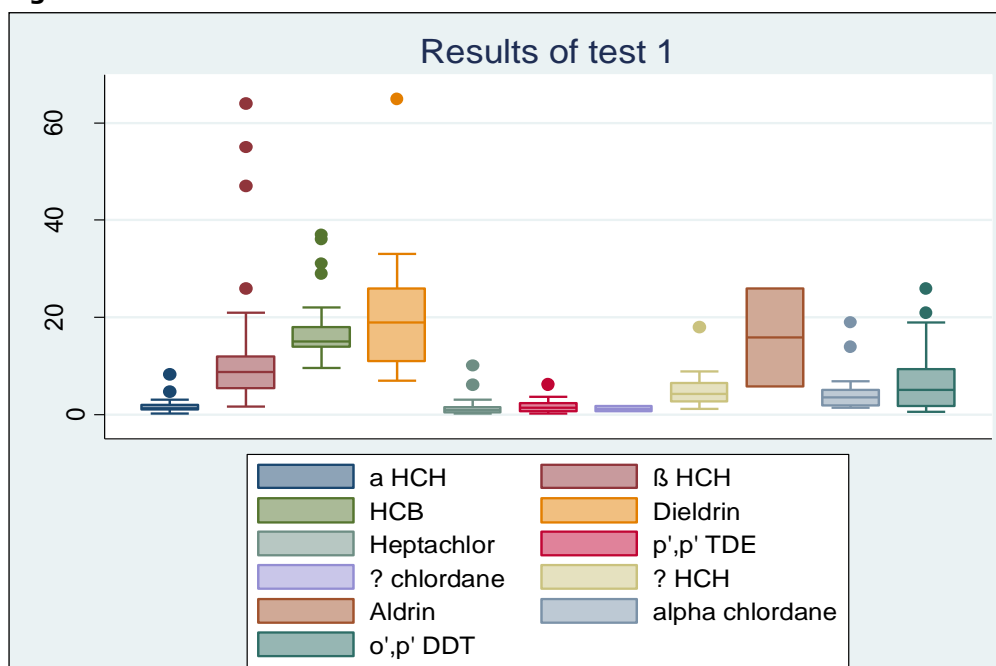
There is evidence of other non-identifying monitoring results specifically from EDL employees, as follows:

Table 6.   Non-identifiable monitoring results

No	11.02.05 pp-DDT (20-50)	pp-DDE (410-2780)	Dieldrin (8-28)	16.05.05 pp-DDT (20-50)	pp-DDE (410-2780)	Dieldrin (8-28)	31.08.05 pp-DDT (20-50)	pp-DDE (410-2780)	Dieldrin (8-28)	30.11.05 pp-DDT (20-50)	pp-DDE (410-2780)	Dieldrin (8-28)	21.02.06 pp-DDT (20-50)	pp-DDE (410-2780)	Dieldrin (8-28)
E1	35	1400	26	28	1100	27	42	1100	ND	70	1800	29	65	1400	31
E2	73	500	33	68	410	36	89	490	34	110	580	33	94	410	30
E3	21	360	14	47	500	36	73	540	33	110	660	30	106	480	30
E4	97	570	31	110	650	45	130	630	35						
E5	16	3100	65	120	2700	70	190	2800	74	270	4800	59	190	2700	44
E6				16	790	19	24	840	19						
E7				7	250	19	13	260	20						
E8	16	300	22	19	250	20	32	310	28		400				
E9	4	910		11	500	9	14	620	7	13	700	7	11	490	9
E10										20	2900	7	58	1700	12
E11										25	2400	11			
E12										12	730	7	36	750	14
E13	26	540	25												
E14	71	550	27												
Mean	39	914	27	34	794	33	67	843	31	78	1663	23	80	1133	24
Range	4-97	360-3100	14-65	7-120	250-2700	9-70	13-190	260-2800	7-74	13-270	400-4800	7-59	11-190	410-2700	9-44
Number	9	9	9	9	9	9	9	9	9	8	9	8	7	7	7

Results shown in ng/g on a lipid weight basis. ND = not detected. NQ = not quantitated.

Comparison data comes from the Organochlorine Programme of the Ministry for the Environment carried out in 1996 and 1997 as part of the National Nutrition Survey {Bates, 2004 #12}.

The most frequently detected pesticides in serum from the New Zealand population were:
- pp'-DDE, which was detected in all strata: concentrations were in the range 413–2780 µg kg-1 lipid, with median and mean concentrations of 919 µg kg-1 and 1080 µg kg-1 lipid respectively
- •Dieldrin, which was detected in 57 out of 60 strata: concentrations were in the range
- < 8–28.4 µg kg-1 lipid, with median and mean concentrations of 11.5 µg kg-1 and 14.2 µg kg-1 lipid respectively
- β-HCH, which was detected in 40 out of 60 strata: concentrations were in the range
- < 7–73.1 µg kg-1 lipid, with median and mean concentrations of 10.7 µg kg-1 and 19.7 µg kg-1 lipid respectively.

Of the remaining pesticides analysed, pp'-DDT was detected in 18 strata at a maximum concentration of 49.2 µg kg-1 lipid. All other pesticides were either not detected, or detected on less than 10% of occasions (i.e. detected in less than six strata). Of these, γ-HCH was detected in one stratum at a concentration of 91.1 µg kg-1 lipid, HCB was detected in four strata at a maximum concentration of 53.6 µg kg-1 lipid, and trans-nonachlor was detected in three strata at a maximum concentration of 8.4 µg kg-1 lipid.

The data from the first test at Mapua are shown below, along with the median and range of these population values for which there is sufficient population data (pp'-DDE, Dieldrin and β-HCH.

Figure 6.   Range and median levels of OCPs in staff (s) compared with population (p)
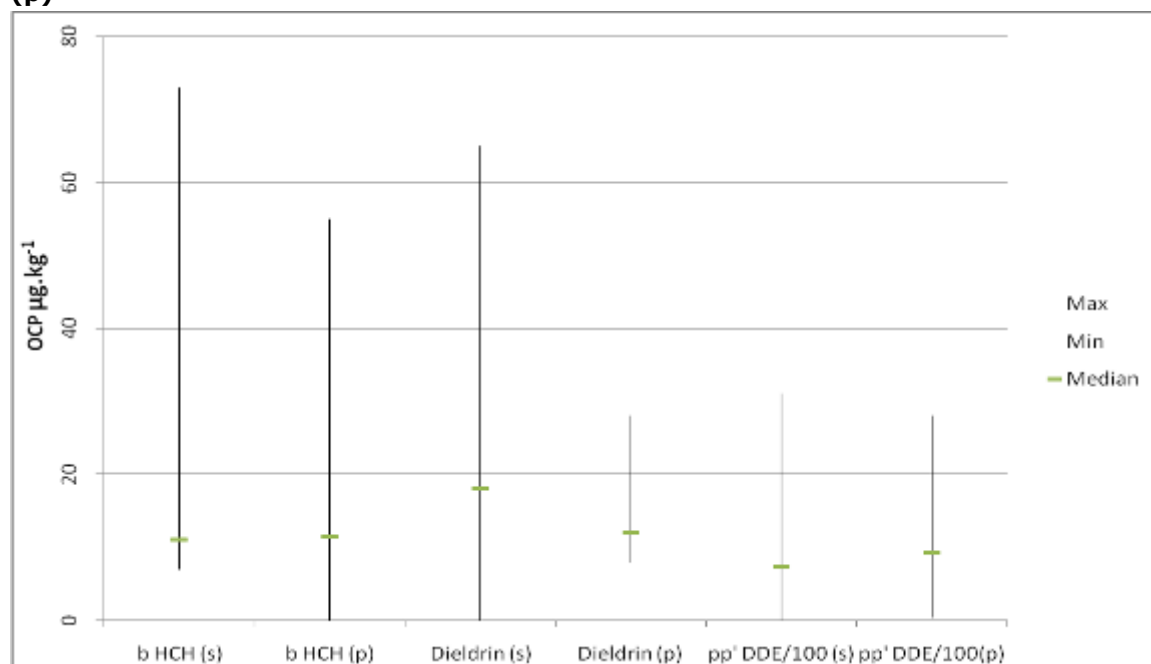
β HCH is in line with the population values, as is DDE. The Dieldrin levels are slightly increased.

There are, unfortunately, too few serial tests to make valid comparisons between individuals or as a group.

Analysis of OCP results

An occupational physician was contracted to MfE to produce a report assessing OCP blood results for the workers.

The Interim Medical Report, 2006, assessed worker OCP blood test results only.

The occupational physician commented that: "There were some difficulties with the testing programme, which I was told was complex technically and had a variation of up to 20% in the results. There was also some time delay in getting results back"."

On the results, he comments that: "All results for Dieldrin, pp-DDE and pp-DDT (except for one pp-DDE) were within the range for non-occupationally exposed New Zealanders as indicated by the Ministry's study. The one exception was a pp-DDE level of 3000. This individual had a relatively high initial test of 2300 on 11 February 2005, which is of interest in that he was not on-site until April of that year."

He also commented that pp-DDT appears to have had the most sensitive response to exposure. The remainder of the report is difficult to comment upon, in that the on-site groups that he was commenting on have been censored under section 9.2(a) of the Official Information Act. In the discussion he says:

"In general, as far as Dieldrin and pp-DDE were concerned, most results were within the range of the Ministry's study and were of no cause for concern. There was no pattern of increase with time on site, and no pattern which related to the wearing or not wearing of protective equipment."

He did, however, specifically comment on groups of employees in a final report (June 2008). The MfE employees' results all fell within the normal range, with no increase in time for pp-DDT. Three Highway Stabilisers Environmental (HSE) employees showed rises in pp-DDT with time and two a temporary rise in Dieldrin. EDL employees generally had pp-DDT levels at the final test which were greater than baseline. Employee numbers 1-5 had "increases with time on-site which exceeded the upper limits of normal. This can be seen in the figure below. There is, in general, a rise with time. There are outlying results: employee 5 had a sharp rise with the November 2005 test, and there was also a peak for some employees in the May 2006 test.

Figure 7. Levels of pp-DDT in EDL employees with time
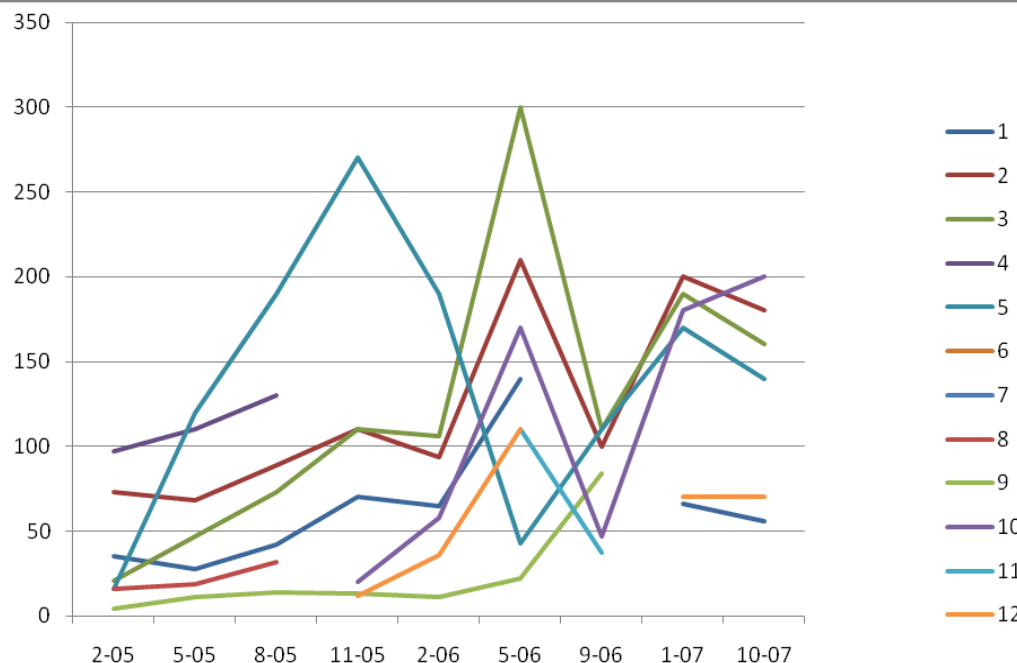
The occupational physician also comments:

"This group of workers in general were closest to the highest dust levels. Wearing of protective masks was variable, and hours of work were long (six days by 8-10 hours a day). In all, this group had a higher intensity of exposure or 'dose'. Their response was a clear rise in pp-DDT levels with time, regardless of their baseline levels."

The conclusions of the report were that the level of OCP in staff blood did not pose a concern, although there is no indication of whether or not the 25% action level had been exceeded.

Gaps identified

The reporting lines for the health monitoring process are not clear. According to the plan, the MfE site manager (EMS) was responsible for checking the "fitness for duty" reports and also for liaising with the site supervisor and testing laboratory to "arrange for tests". It is not clear whether the MfE person was also responsible for the other contractors: EMS say that this responsibility was not delegated to them. This responsibility specifically delegated or not, should have been clear. However, the occupational physician was only contracted for a period of six months, March to August 2006. It is not clear who the occupational physician was prior to (or indeed after) this, but for day-to-day management local general practitioners were also involved.

There is no evidence that, apart from planned blood tests, reporting and investigation of health complaints was encouraged.

The eventual appointment of a specialist occupational health company, Ramazzini, had the potential to add value had they been allowed to manage the occupational health process (in the manner, for example, of scheduling appointments). Oral evidence suggests they were not.

The biological and health monitoring on site was, in theory at least, a sound plan marred by some deficiencies. One of these was the complexities inherent in the interpretation of the results, which required specialist input – that is to say, an occupational physician. The second was the expense, which would have been considerable and, as minutes of meetings suggest, were difficult to keep pace with. The site management minutes record several instances where results were delayed by non-payment of invoices by EDL.

Although the tests were supposed to be done every three months, there was inconsistency in the testing and very few serial follow-up tests were in fact done. As it has health effects similar to those of organophosphates and it was being treated through the MCD, acetylcholine tests should have been provided. It would have been prudent for MfE to provide the baseline acetylcholine tests as indicated in Attachment D to their health and safety management plan. There may have been some exposure at this point to cholinesterase- limiting chemicals which went unmonitored.

Like the environmental monitoring, there was a limited number of chemicals being monitored in the testing suite.

There are inherent difficulties in using the biological monitoring approach in this case.

The first is common to all methods of monitoring in that there are no WES set for the pesticides, so there are no BEIs with which to compare them, thus no explicit

"action" level. Baseline levels can be established, with arbitrary "action levels" set on excursions from baseline, but the baseline measurements were not taken until five months after the project started (which was 14 months after one of the workers started).

The second is that, in reality, there is limited information on toxicokinetics of some of these compounds. Biological half lives are reasonably well characterised in blood for Dieldrin (267 days) chlordane (10-20 days) and Lindane (18-21 hours). The remainder are most likely to have long half lives like Dieldrin. For some of the compounds the serum levels would have reasonably represented the long-term exposure to these agents, as the persistent organochlorine pesticides (POPs) have biological half-lives in the order of years rather than weeks or days. For others, particularly chlordane and Lindane, three-monthly testing would not have given useful information unless the exposure took place days or weeks previously.

The third significant problem with biological monitoring of these persistent agents is that very low levels of the substances must be detected, in the order of nanograms (ng). This requires strict laboratory procedures to avoid contamination (of glassware and other apparatus). These procedures are technically complex, requiring considerable set-up time and usually involve extensive quality assurance procedures to ensure repeatability. To ensure a lack of contamination, laboratory blanks are assayed (and should not show elevated levels of analytes) and samples with known levels of analytes ("spiked" samples) are assayed and should detect the analyte at the spiked concentration. Lastly, POPs are fat soluble, and the lipid component of serum provides a good indication of the level of exposure. This does, however, mean that the lipid component of serum must be known, and the level of POP "lipid adjusted" to take into account normal population variation with the results expressed in micrograms per kilogram of lipid ($\mu g.kg^{-1}$) or nanograms per gram ($ng.g^{-1}$). Both are equivalent to parts per billion.

If the lipid component degrades, then the value of serum POPs will be in error. This is why serum must be separated shortly after the sample is taken, centrifuged, stored at (typically) minus 70ºC and transported on dry ice to the laboratory for analysis.

Lastly, laboratory variation and error also plays a part. Agriquality were only able to guarantee the results with a margin of error of 20-30%, which is important when a 25% increase is selected as the action level.

As regards analysis of the monitoring, it would have been preferable for an assessment and medical report to include all pertinent health surveillance information, including routine blood tests, entry/exit medicals and acetylcholine test, which would have provided a more comprehensive assessment of worker health.

Another limitation of the Interim Medical Report is that 14 chemicals were analysed in worker blood but only three were considered in the report. One

isomer of DDT, o,p'DDT, was omitted, which would have low-biased the total. Some workers' OCP results were available but not included in the report for some reason. Similarly, some worker start dates were omitted.

A "25% trigger level" for OCP exposure wasn't selected until January 2006, but should in fact have been set prior to implementing the programme. No reference was made to this action level in either of the health reports assessing OCP blood test results written by the occupational physician. According to MfE, the occupational physician does not recall being informed of the 25% trigger level and they hold no records as to how this level was developed.

The options and criteria for action should be established before undertaking health surveillance as should the method of recording, analysis and interpretation of the results of health surveillance (HSE blue guide).

In 2008, the occupational physician's findings were outlined in a report. (Review of the Role and Actions of the Ministry for the Environment New Zealand, undated, CJ Bell):

> "In June 2008 [   ] completed his Final Report on Organochlorine Blood Monitoring, reporting this aspect of the health monitoring of site workers from the period 2005-2007. The key finding was that the levels of pp- DDT in blood tended to respond to on-site exposure in some staff. There was a high degree of variability in both the baseline conditions of staff, and their response over time. He indicated the importance of collecting baseline information for each individual, so that change can be measured from this baseline. It would be very difficult to interpret OCP results without baseline measurements."

Implications of the gaps

It seems, from the MfE management plan, that the site supervisor (or manager) was responsible for day-to-day management of medical matters on site, including entry and exit medicals and liaison concerning biological monitoring. It is not possible to say whether or not the responsibility was delegated. If this is so, then this person had responsibility for all the workers on site. This would have removed responsibility from on-site contractors and may have led them to believe that they did not have any responsibility.

At the July 2006 site meeting a "recent problem with the lab personnel relative to perceived high thyroid problems" was discussed. "The local GP is planning on contacting .... [the occupational physician] relative testing. The MfE rep stated that she can write a letter from MfE indicating that employees have the option of stopping their work at the site if they feel uncomfortable about staying there".

The site occupational physician was only appointed for six months between March and August 2006. Important opportunities were therefore missed. During the period prior to the appointment, important decisions were made about the biological monitoring programme. Occupational medical input at the planning

stage would have again stressed the need for a thorough hazard ID and monitoring programme before embarking on a relatively sophisticated programme of biological monitoring, which is unlikely to have been carried out in New Zealand before.

The opportunities for miscommunication did in fact manifest themselves. When an EDL worker had an abnormal full blood count in February 2006, the fact was not immediately recognised and there was a four-week delay before it was reported. This abnormal blood test was unusual, but advice later received from two consultant haematologists cannot rule out the fact that it may have been related to exposure to toxins present on the site. The worker involved was having health problems at the time, and further prompt blood tests may have helped in the clinical management and diagnosis. In the meantime the worker was attempting self-treatment that was not appropriate and could have been harmful in the long term.

This incident was highly unusual in that there is little evidence that full blood counts would have given any useful information for health monitoring purposes, except in the case of acute poisoning with some of the substances on site. An acute change did, however, take place but went unrecognised.

It is clear from the written evidence and interviews that several of the workers began to experience ill health while working on the site and the health assessments failed to identify them. The purpose of such health surveillance is to identify any health effects at an early stage, review the effectiveness of control measures and provide medical care if necessary. The sudden appearance of health problems in employees in a work area may indicate a breakdown in safety precautions, procedures or supervision, and self-reporting of symptoms is a prudent part of health surveillance.

The remainder of the health effect monitoring was the form of testing usually carried out in health checks, with possible recognisable effects in acute exposures. The possible exception is induction of liver microsomal enzymes. However, this was not done, reference levels and an action level being necessary.

As it transpired, the tests were not carried out often enough for this approach to yield useful information. The samples also took time to process, the combined effect being a lag in receiving the results which, like the environmental monitoring, would have allowed exposure to continue in the meantime.

The MfE Occupational Health Monitoring Policy states that: "The results of monitoring under this plan will feedback into the health and safety plan, and adjustments to policy and procedure will be made where required."

At baseline, the results show that, for the suite of substances measured and after some degree of exposure, most individuals appear to fall within "normal" NZ population values. This does not mean that some individuals may have had lower levels at baseline with a subsequent rise. There were also some substances which were not part of the monitoring programme.

In the absence of baseline information for all workers with prompt analysis and reporting of results it was, and is, very difficult to identify exposure effects. The ad-hoc nature of the process did not and does not allow this.

Occupational health monitoring should have been expanded to include other chemicals of concern besides OCPs that weren't included in the environmental monitoring. The policy also wrongly indicates that dust is the only exposure route, when inhalation of gaseous chemicals was a concern from the start of the project, necessitating the addition of the PUF filter in the environmental monitoring. The policy also states that the worker's physician would receive a "certificate of exposure to hazard" but there is no evidence of this. The 25% trigger level for OCPs is not identified in this policy.

Regarding worker concerns about solvent exposure, acetone and hexane monitoring tests were readily available and accessible to staff as a urine test at the local health centre, contrary to the statement made in the MfE Monthly Report. In addition, MfE did not inform the lab staff about these findings or that they were even researching the issue, or of the existence of the monthly report. In the absence of any occupational monitoring in the lab, biological monitoring of hexane and acetone would have provided useful exposure information. Exposure to these solvents can cause neurological and other health effects.

This evidence indicates that MfE did not implement several components of their health and safety management plan. There are inconsistencies between MfE's own health and safety documents. EDL supplied their finalised health and safety plan to MfE in 2007.

The lack of baseline information, lack of systemic hazard identification and assessment, inadequate hazard control methods and personal monitoring, and the limitations of biological and environmental monitoring meant that workers were exposed to an unknown range of toxic chemicals which may have health effects.

Information, supervision and employee involvement: statutory requirements

Under sections 11-14 of the HSE Act, employees have the right to receive information about monitoring that has taken place, emergencies that may arise and hazards to which employees may be exposed. If either environmental or biological monitoring is carried out, employees have the right to receive the results of monitoring regarding conditions in the workplace (Section 11).

Employers also have a liability to give information for employees generally (Section 12). This means that employers must give employees information about the substances that they work with, particularly if the substance has health effects. If so, these health effects should be detailed, and the steps that employees should take to minimise the risk of the effects occurring should be outlined for them, particularly the use of any safety equipment or clothing.

On-site performance

The most specific monitoring carried out was the biological and health monitoring. It is not absolutely clear, from the audit material supplied, how individuals were given the results of their monitoring. The responsibility (Attachment D to the Health and Safety Management Plan) lay with the supervisor/occupational physician. The occupational physician made site visits, but there is no record of an example of the oral advice that he gave. One staff member was routinely not at work on the days that he visited and believes that the advice was not made routinely available to workers. It appears that occupational history information was elicited from some workers, but not lab staff, at some point well into the project. This information should be included in the entry medical questionnaire to ensure workers are fit for work on a contaminated site.

The routine blood tests (for MfE employees) were the responsibility of an independent occupational health provider, Ramazzini of Nelson. Clear lines of responsibility were not established – EDL did not, apparently, have a contract for these services. It is not clear how these test results were followed up. The evidence from at least one employee suggests that there were delays in following up a set of significant monitoring results. The employees interviewed were not aware of their own doctors receiving results of the monitoring.

It also appears that workers were not routinely made aware of the results of environmental monitoring at any time during the project, nor were the results discussed with workers. In one instance a worker asked for results of monitoring because of health concerns and was told to go to the Mapua library to look up the information in the MfE monthly reports archived there as part of the communications plan for the public.

On inspection by the worker, this document provided monthly summaries of all aspects of the project. The same worker eventually had to make official information requests to obtain comprehensive monitoring results.

General information
The hazard information is contained in the Health and Safety Management Plan, with specific reference to (and MSDs type information about) DDx; Aldrin; Dieldrin and Lindane. Otherwise there is a list of "contaminants likely to be found on site". There is information in the Health and Safety Management Plan on the routes of exposure to contaminants and the PPE to be used (pp 11-12). PPE selection was to be "dictated by the results of the occupational health monitoring programme" with the proviso that "the level of PPE required may be upgraded if air monitoring results exceed relevant action levels." Unfortunately, workers were not provided with a copy of this document. The occupational physician, in his final report on the OCP monitoring, gave "some other general thoughts/suggestions". He said under the heading "advice to staff": "When I was asked to become involved I found little had been done to advise employees of the chemicals and possible hazards/health risks. Experience shows that if this is done from the beginning there is likely to be greater appreciation of health risk and better compliance with health and safety requirements."

Training and supervision

Section 13 of the HSE Act lays out the duties of the employer in this respect to ensure that employees are properly trained and supervised. This includes training in the use of plant, the uses and properties of substances at work and the use of any protective clothing and equipment with which the employee has been provided. The aim is to ensure that all employees have the necessary knowledge and experience to work without either harming themselves or anyone else in the workplace. Those under training must be adequately supervised by someone who does have the knowledge and experience required to do the job safely.

Documentation is available on respirator "fit testing" carried out by "3m" representatives in November 2005. Nineteen employees had masks fitted, with two failures due to beards.

Induction for four on-site contract workers took place in January 2005, with Health and Safety meetings for these staff taking place in April 2005. HSE distributed the Health and Safety manual to their staff in July 2005. In the same month EMS were to provide details of the "biological monitoring and toxicology".

A Safety Induction presentation dated "14th No last modified 1st February 2005" forms part of the documentation. This gives a good overall view of the operation and the health and safety issues at the site.

Noitceboards were made available by EMS in October 2005 for "topical and timely" information to be posted. An example of what was made available on the noticeboard is reported in an unsigned document "Health and Safety for Dec and Jan 06" as follows:

"During this period the following topics were covered by means of the noticeboard information system: Practical Considerations of Respiratory Protection; OSH Noise Fact Sheets; extremes of Temperature; High Risk Industries and Occupations; Preventing Injuries when working with Hydraulic Excavators and Backhoe Loaders; Avoiding Falls from heights and MSDSs for Lindane and DDT."

Health and Safety "refresher" training sessions were held in April 2005 with a brief on biological monitoring in July 2005. An "education plan" was documented in November 2005 because "several people had high levels".

In July 206 an "urgent health advisory notice" was promulgated. This outlined the highly contaminated nature of the soil in the storage piles, and advised that individuals "pay particular attention to health and safety requirement for staff and the general public and this was a reminder to the short term sub-contractors as well." All employees and contractors were to use level C protection while on the EDL plant pad.

The training given to all staff should have included the hazards present, their effects and how to recognise them, and the steps that they needed to take to protect themselves. This is, in general, industry and industrial process specific:

many industries are involved in developing codes of practice to ensure that safe systems are in placed with their particular type of work.

Laboratories should have health and safety standards, and are a specific instance of where codes of practice are available for the handling of hazardous substances. A staff member writes that neither of the lab technicians had formal laboratory experience prior to taking on the job, and the on-site training given was not by an experienced practitioner but a PhD student. A subsequent audit (to assess quality control) identified significant problems with analytical procedures and issues related to health and safety.

In February 2006 there was apparently induction training carried out, but the staff interviewed for this report were not invited to any related meeting and did not receive induction training. There was no training given regarding wearing the masks and no fit tests were given in the lab. One employee found out that their mask was too large only after they could still smell chemicals with the respirator on. This worker's prescription spectacles broke in half after wearing the respirator for a few days because they "didn't fit over it".

There are mentions in the documentation of workplace induction refresher training held for EDL, but no documentary evidence.

There are mentions of "tool box talks" which is the generic name given to ad-hoc or periodic health and safety discussions at the work site, but no formal record of proceedings is available.

Employee involvement

Employers must give employees an opportunity to develop procedures for hazard identification and control, and employees must also have input into the procedures for dealing with emergencies or other dangers in the workplace. There is no mention in the documentation about the procedures to involve employees, but a health and safety committee (to which, apparently, none of the staff interviewed contributed) did seem to exist. On the 8th July there is a documented "Review of Health and Safety Meeting with EDL employees" The record states that "This meeting was initially organized in an effort to help employees interpret the OCP in blood data that had been provided to them on 1 July 2005. EMS Health and Safety rep had offered to provide this service at the site management meeting of 1 July 2005. A first attempt on 6 July had been aborted due to the early arrival of ministry staff on a site visit. EMS staff were completely occupied with sampling events on 7 July of the 13 [   ] Street property and so the meeting was reconvened at the direct insistence of the EDL staff for 8 July at 1 pm.

The OCP in blood results were discussed and the staff were particularly interested in the limitations of this type of biological monitoring.   All staff indicated that their results had shown minimal change.

The staff were very interested in discussing the other compounds that may be in their environment that could be a hazard to their health as well as the many routes of exposure that they encountered during their normal work week that

involved a potential exposure to OCPs. Staff drew diagrams of the process and provided examples to explain the areas of concern

Specifically, the areas where they are concerned unknown contaminants may be encountered are:

1   In the dryer. The flame coming in direct contact with some of the soil may be producing a variety of organic compounds that may be hazardous to their health. Since, they enter the dryer and do not wear any additional protective gear for this maintenance work, they are concerned that they are exposing themselves to additional risk.

2   When repairing augers or doing maintenance on the section of the plant that transports the coarse and fine silo output along with all the reagents. This material is often as hard as concrete and requires quite a strenuous effort to remove and repair. Again they conduct work with this material without any increased personal protection.

The other area of major concern was the availability and suitability of personal protection equipment. Specifically,

1.   Gloves supplied were not impermeable to the dusts.
2.   Overalls supplied were not impermeable to the dusts.

Implications of the gaps

Apart from this meeting there is no record of how individual feedback was given to employees about the results of their health monitoring, apart from the employees interviewed for this report and the documents to hand. These consultations with a health professional are important opportunities to discuss concerns, specifically the results of health monitoring and their implications. There seems to have been a problem with routine: although the site is a small one, failure to have a specific system which is available routinely to all employees will allow individuals and problems to remain un-noticed.

General practitioners are an extremely important source of advice, but relying upon them to follow up on the results of occupational monitoring and recognise the implications is not advisable unless they have specific training, understand the site operations and have been given specific responsibility. It enhances the likelihood of delays and confusion as has happened, according to the evidence, on at least one occasion.

There was no evidence that the results of environmental monitoring were made routinely available to staff. According to the management plan, a change in the level of PPE protection depended specifically on information on changing levels of contaminant being available. It relied upon supervisors to act on the monitoring information. This was not a "fail safe" mechanism. Had employees known about this information, they could have taken action themselves. It also led to employees being unknowingly exposed to hazards, which greatly affects how they

appreciate the risks and leads to significant stress and concern and the potential for adverse health effects.

Workers should have been well-informed of all the known and expected hazards and their health effects prior to starting work on the site. This includes information regarding the emission of dioxin in 2004 and other chemicals as they were identified, i.e. phenothiazine and benzene. That way they could have made informed decisions about whether they wanted to work under those conditions.

Three out of the four workers interviewed reported they would have ceased their employment immediately had they been advised of the dioxin release and any on-going concern with de-novo dioxin formation. Management did not explain to EDL workers the concept of volatilisation and the requirement for keeping the dryer temperature below 120°C. When workers finally found out about these issues some years after the fact it was a source of significant concern to them.

There was an apparent failure to provide systematic training in health and safety and make information on hazards readily available to all staff. This resulted in lost opportunity for individuals to take responsibility for their own health and safety. Preventable exposures therefore took place.

Effective supervision also involves leadership and responsibility. The narrative of several reports written by one staff member suggests the existence of serious communications problems between the laboratory staff, EDL and MfE.

Although personal monitoring results were communicated, there seems to have been a particular problem in communicating the results of environmental monitoring to staff. If results were withheld throughout the project, MfE were wrong to do so. It appears that one worker, who had valid reasons to be worried about their health, experienced difficulty in getting this information. This led to an element of mistrust. In the words of this employee "the workers were used as guinea pigs".

Conclusion

The Mapua remediation project took place on a site contaminated with numerous significant hazards, where a novel remedial technology was being trialled. From a health and safety perspective, it was a complex situation requiring experienced management. This was initially available through an experienced remediation contractor, but subsequent changes in plan meant that a government ministry with little or no experience in this specialised field took over. The contractor responsible for the decontamination process, EDL, appears to have had little experience with health and safety matters.

An adequate health and safety management plan (informed by people with the appropriate expertise) would have reduced the risk, but the plan developed by Thiess was not further developed and contractors were tardy in producing their own plans, which were inadequate.

The most serious consequence was a failure to consider, individually and comprehensively, the operational areas on the site, carry out an adequate hazard identification, assess the significance of those hazards and plan control measures for each. Some hazards (particularly on the plant and laboratory) went largely unrecognised for a significant period of time. As a result, workers were exposed to a range of potentially toxic substances including volatilised pesticides, dioxins and benzene.

As the project went on, further hazards were identified such as phenothiazine, benzene, and on-going concern about dioxin by the Peer Review Panel. However, these were not added into the environmental or personal monitoring, any actions that were taken to address them did not take place in a timely manner and inappropriate methods and tools were used. Monitoring should have been reviewed and adjusted during the project to include newly identified hazards. MfE displayed a lack of appreciation of the significance of the hazards.

MfE did not have suitable management systems in place to guide and track health and safety activities. Workers interviewed were not involved in the development of procedures. A worker's serious harm accident was not recorded in an accident report and reported to the Department of Labour as required. Pre-employment medicals and baseline biological testing were not available at the start of the project, so several workers did not receive baseline tests. Several workers' exit medicals identified symptoms consistent with exposure to site hazards but did not receive medical follow-up, and not all the workers received an exit medical.

After repeated failure of the air emissions systems and significant increases in toxic air emissions from the smokestack, management should have become extra cautious with respect to worker health and safety. Workers certainly should have been advised about the status of the plant and the existence of the MfE monthly report, which contained a monthly summary of the project.

The hazard controls in the EDL lab were inadequate and not improved, even after a consultant report concluded that ventilation was inadequate. Worker concerns about solvent exposure were not addressed. After lab workers made claims that their health was affected by their work, EDL provided respirators to them without fit testing or dust filters. They then made comments to those workers suggesting that the respirator shouldn't be worn in public.

Workers were not adequately informed about the nature of the hazards, either on the site or about those that could potentially be produced by the process. Results of monitoring were not provided to workers and there is evidence that MfE withheld this information from a worker when asked for it. Part of the explanation for this failure may have been that the environmental monitoring for resource consent purposes formed a considerable body of data. A notable omission was that workers were not advised about the presence of dioxin in the 2004 emissions monitoring and concerns from the Peer Review Panel about continued dioxin generation. The recent revelations to workers about the presence of dioxin, benzene and other hazards have proved to be a source of tremendous stress, which is ongoing.

Although there was a copy of the resource consent conditions on site, the employees interviewed were unaware of this. They were also unaware of the effect that temperature had on the soil contaminants. They were aware that the emissions were being monitored because of the public health risk, but not about the specific constituents (particularly dioxins). They all believed they were making important and positive contributions to their community and the environment, but would not have been involved had they known of the risks

The occupational monitoring policy was not ready at the start of the project, so some workers did not receive baseline biological tests. It is very difficult to assess subsequent levels in the absence of a baseline. Cholinesterase tests were not provided as baseline or when the phenothiazine was found. Adequate investigation of a worker's abnormal blood test was not undertaken. There is also evidence of resistance on the part of MfE to provide biological monitoring of solvents for lab workers, for some unknown reason.

There are some limitations regarding the accuracy of organochlorine blood tests. Only a small subset of the available data was used in evaluating worker exposure in the 2006 interim medical report, so this document has limited usefulness as an assessment of worker health. Workers were not advised of the existence of this report, which identified a casual attitude of workers toward the wearing of respirators.

It is unclear what training was available and who received it. The lab workers did not receive any induction training or training in the use of respirators or emergency procedures.

The likelihood of exposure of workers must be considered. Based on prevailing wind direction and deposition modelling performed for the MoH report, EDL workers, including the lab staff who were downwind of the plant, would have been subject to some of the highest exposures of environmental toxins (gaseous and dust) emitted from the plant. One worker, who worked in the soil dryer area, was subject to fugitive emissions directly from the dryer in addition to the reactor emissions. No personal occupational monitoring was provided to this worker at any time, nor was there any in the lab in over two years.

Occupational monitoring on the site was inadequate and it has been concluded that there were numerous problems associated with the environmental monitoring, making it of limited value.

> *"This investigation into public health risk is limited by the lack of exposure data for members of the public living or working in close proximity to the site during the remediation to several chemicals that remain of concern, namely dioxins, benzene, PCBs, and for those living south of the site only, OCPs and arsenic."* (MoH report)

The legislation requires that the plant and equipment are safe and workers are provided a safe working environment. However, breaches of the resource consent

conditions and operational failures caused the air emissions control system to fail on several occasions. This led to significant increases in emissions and hazards that were not adequately characterised or monitored. The working environment may therefore have been unsafe during periods of the remediation.

Another major problem was MfE's lack of understanding or awareness of their statutory responsibilities as principal of the site. It is clear from the evidence that all practicable steps were not taken on the Mapua remediation project. The health of at least four workers was affected to some degree but, due to the information gaps and quality and range of the monitoring data, it is impossible to assess worker exposure. Future health effects cannot be ruled out.

APPENDIX 2

OCCUPATIONAL MEDICINE INVESTIGATION

OCCUPATIONAL MEDICINE INVESTIGATION

Plant employees

During the course of the project there was a natural turn-over of staff, but this section of the report focuses on the occupational medicine aspect of four individuals who worked at the plant and were known to have health complaints. Two of these employees worked in the plant ("EDL pad") itself, and two in the on-site laboratory. They made themselves available for interview, but were not clinically assessed.

Of the employees who worked on the plant, one was the plant engineer and the other a "dryer operator" but was also a qualified fitter/welder, carrying out these duties as necessary. All worked on the site in the early period of operation of the plant, referred to in the PCE report as being of concern:

> "In the early period of the remediation (up to November 2005), the situation was potentially of much more concern". (p40 PCE main report)

During the soil drying process, the soil to be remediated from the site was fed into a hopper, and then carried to the dryer on a conveyer belt.

The dryer operator describes this as a "very dusty" job, requiring the use of a half-face air purifying mask as personal protective equipment (PPE). Additional PPE was required when in the bag house associated with the dryer. When the dryer was in operation it was common to see fugitive "steam-like" emissions coming from the seal around the dryer along with the dust emissions.

His job involved taking the door off the drier and unblocking the inlet by shovelling it out several times per day. He doesn't recall an odour, but was "knocked down" several times while doing this. At the end of the day when the fans were shut down, cleaning was again required and the emissions would come back through the dryer. No personal or environmental monitoring was performed in this area or for this worker. Before shifts started it was quite common to find a number of dead birds in the drier area and outside on the concrete pad.

There were also back pressure issues acknowledged in the PCE and MOH reports:

> "The PCE report states there were back pressure issues with the MCD plant during the first year of its operation that may have led to fugitive emissions from the rotary dryer. Such emissions are likely to have been into the storage shed for the contaminated soil awaiting infeed to the rotary dryer or into the surrounding open air. There is no record available of any monitoring done in these areas to assess if these emissions were occurring. These emissions may have been OCPs, intermediaries such as dioxins or any other compound in the soil that was volatilised in the rotary dryer." (MOH report)

After drying, augers carried the dry soil to storage silos. An additional part of this job required emptying the silos when "stoppages and blockages" occurred in the equipment. This the operator describes as a "fine brown dust" which had to be emptied with a wheelbarrow.

From the silos there was a feed to the ball mill reactor where the chemical reactions took place. In this area an ammoniacal gas was given off, and within the dryer there was an access or inspection door which had to be opened to clear debris from a "grill-like" plate within.

There was also occasional exposure to what seemed to be quantities of pure pesticides. On one occasion he observed a "big lump of blue stuff" in the in-feed to the plant.

Figure 8. An example of in situ contaminated soil, dye, pure product, and debris

Figure 9.   A jar of solid material collected after the dryer and particle size separation

In addition to this, in the fitter turner role, the dryer operator was involved in welding. The materials that were deposited on the structural work or machinery being worked on may have been vaporised.

No respiratory protection was worn while welding. The coveralls he was provided caught fire once because they were made of flammable material. He asked for cotton coveralls after that incident but they were never provided to him. He spent considerable time flame cutting during decommissioning of the plant. This also worsened his symptoms.

The plant engineer was closely involved with supervision and maintenance of the plant, being exposed to similar working conditions but also with much more fabrication, including welding, on the plant, carrying out many of the modifications required.

Health effects

It seems that the plant emissions, particularly from the dryer, caused one of these operators to experience a sensation of dizziness and occasionally to suffer from respiratory problems. This included "air hunger", when the operator found it difficult to get a breath at times. Neither of these sensations was associated with any particular or significant odour.

He also experienced headaches, sinus problems, tingling, fatigue, visual disturbances, poor memory, wide-spread musculoskeletal pains and a productive cough. His hearing became worse during this period of employment, during which time he may have been exposed to excessive noise on the plant. Some of these problems are on-going: he has developed an increased sensitivity to chemicals and dust which exacerbate his symptoms. His entry medical was normal, but his

exit medical noted several of the abnormalities described above. He worked on the site for 3 years but has been unable to work since.

The other employee had two episodes of syncope (collapse) while working at the plant, at least one of which required hospital admission. On one of these occasions on the stack he experienced burning droplets on his back. He subsequently experienced headaches, tingling, nausea, slurred speech, irritability, anxiety, poor memory, visual disturbances, localized alopecia, and left side weakness. After this accident he was extremely fatigued and was off sick for three weeks. He was not provided with an exit medical. His health currently seems reasonably good. During the period that he worked there (1½ years) the plant was processing soil with large amounts of Dieldrin.

Laboratory working conditions

Laboratory environment

The laboratory itself was a converted shipping container and it does not appear to have had any "laboratory standard" extraction or plenum air replacement systems. Ventilation was provided by opening the door. However, because of the wind patterns the lab was, more often than not, downwind from the dryer stack and the reactor, thus the "chemical" smell inside the laboratory was quite strong.

Figure 10. Laboratory interior

Laboratory work involved sampling and testing unprocessed and processed soil samples to check for organochlorine pesticide levels. The sample bottles were opened on the bench. Those which had been through the remediation process were still hot and had a strong chemical odour. In-feed soils containing high

levels of pesticides were handled several times a day and, occasionally, "prills" of concentrated pesticide formed part of the sample. An extremely fine, powder-like dust came off the sample bottles when opened.

Initially no personal protective equipment was made available, and the gloves in use were latex gloves, which were unsuitable for handling solvents.

The laboratory process involved solvents, including acetone and n-hexane, with daily exposures to both. This exposure was described as "almost constant" during operational hours. Ventilation of solvent vapours was accomplished by opening windows and doors which allowed the smoke stack and reactor emissions and contaminated dust into the lab.

The Lab technicians were required to visit the plant site several times per day to pick up samples, and that necessitated walking directly into the plant emissions and onto the plant pad with no protective gear.

Figure 11. Plant site

It is difficult to evaluate the intensity of the exposure, but laboratory workers described a strong odour, a combination of the odour from the soil samples and the solvents. This solvent smell was noticeable on clothing when going home after work.

After both lab technicians were diagnosed with illness (July 2006), EDL provided respirators but did not carry out any training or evaluation such as a "fit test". One of the employees was found to need a smaller size of respirator in August of that year after the odour from the smokestack caused the lab technician to be nauseous while wearing a respirator inside the lab.

Health effects

Both laboratory employees were symptomatic. One worked there for 2 years 8 months and the other for close to 2 years, mainly part time hours but also some full time work. Both were in very good health when they started work on the site, but neither were given an entry medical.

Symptoms in common were that both had fatigue and malaise. Unusually, both experienced hypothyroidism and cystitis and both had neurological symptoms. Perhaps the most unusual health effect was an acute haematological event (an anaemia) detected on blood screening on one of these employees in early 2006. The pattern of blood indices on this report is unlikely to have been due to laboratory error.

The worker that was there longer and worked full time during April 2005, a month when stack emissions were significantly elevated, had more symptoms including nausea and vomiting; other gastrointestinal problems; irritability; attacks with tremors and loss of perception; slurred speech; left sided weakness; photophobia; dermatological changes; widespread pain; sinusitis and eye infections.

Clinically, such an unexpected result might at first have been interpreted as being due to a laboratory error, for example a dilutional effect. Two haematologists were later consulted about this abnormal test. Both agreed that the pattern of blood indices on this report was unlikely to have been due to laboratory error, but that this could not be completely excluded.

One suggested that more detailed testing should have been done at the time in order to understand the cause and that the patient would have shown haemoglobinuria (as distinct from microscopic haematuria on two occasions). The occupational physician who assessed the blood test indicated, on the test report that it "needs investigation" but did not undertake any. The other haematologist said a brief episode of haemolysis cannot be excluded – and "this could be a real event as the result of exposure to the agents listed" in other words the organochlorine pesticides present at Mapua.

Relationship with exposures

Emissions from the plant were highly variable, and there seems to have been significant exposure to dust at times.

It is important to note that the smokestack was extended from 5 meters to 9 meters in September 2005. The reactor emissions (ammonia etc.) were at ground level.

The plume dynamics also played a part. The dispersion modelling reports for the 2004/05 stage of the project indicated that the "maximum ground level concentration" (mglc) occurred a little more than 100 meters from the plant, virtually on top of the lab site, and after the stack height had been raised in September 2005 the modelling showed the mglc moved to the SW side of the plant, away from the lab.

The figure below indicates the theoretical deposition rates for dioxin and the deposition pattern in 2004/05 for any of the chemicals emitted from the plant (MOH report). The EDL lab is the uppermost green dot, across the street and downwind from the plant, and the modelling indicates that the lab workers were exposed to some the highest levels of chemical emissions. The lab workers also walked directly into those emissions to go from the lab to the plant site several times per day (when the wind was from the south west).

The plume was also affected by atmospheric stability. On cool mornings (winter and summer), the prevailing wind was from the south west, directing the plume at the lab and keeping odour close to the ground until the afternoon when it would start to rise (unless there was an inversion layer present).

Figure 12. Theoretical deposition rates for dioxin and the deposition pattern for any of the chemicals emitted from the plant
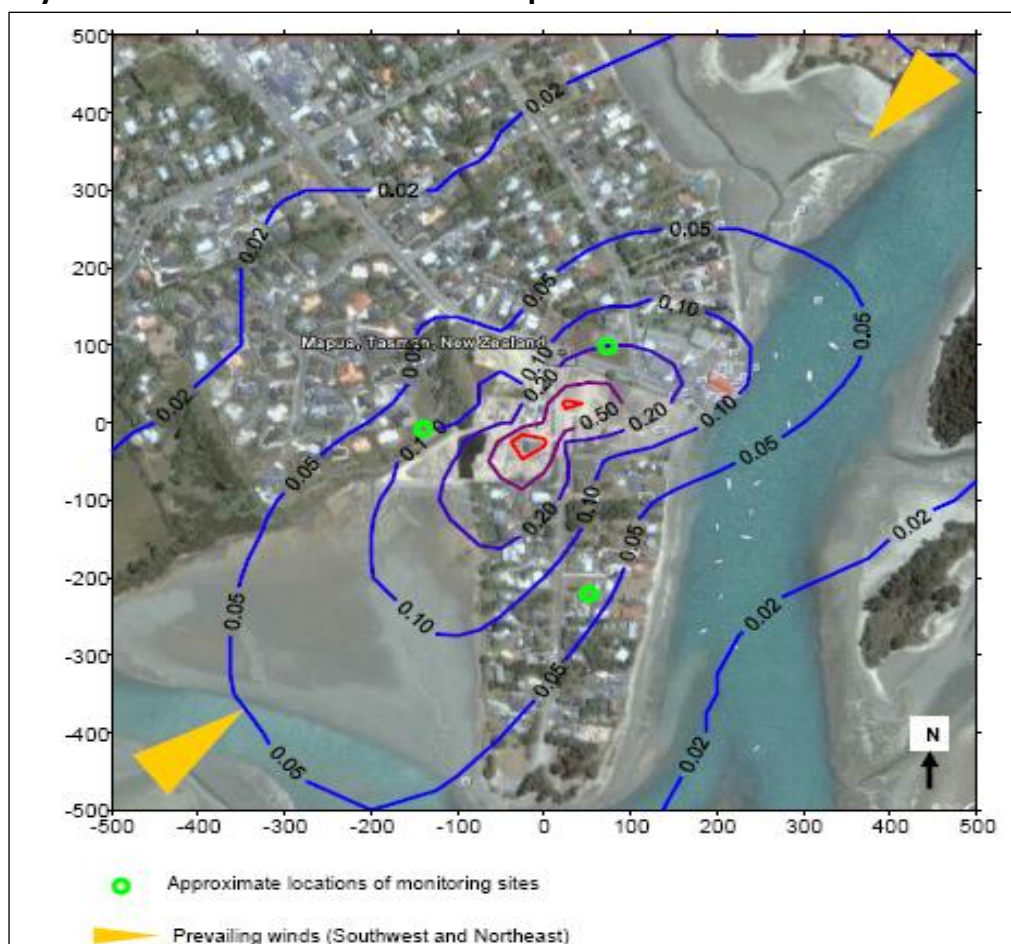
Source: Ministry of Health

There would therefore have been a more than negligible risk of exposure to contaminants, but whether these were of sufficient intensity to cause the effects described remains unknown due to the inadequacies of the monitoring. The dilution effects due to distance would have had some effect. That is not to say that there was no exposure and the situation was tolerable; it was not. Exposure

to dioxins is an emotive issue, and individuals have the right to be informed about such exposure.

The situation on the plant pad itself was different in that the problem would have been from fugitive emissions, which are likely to have led to higher concentrations of contaminant exposure. The exact nature of the exposures is questionable. The output from the MCD process is complex, but essentially is supposed to consist of chloride salts. Di-ammonium phosphate, urea copper sulphate and sand were added to the process. Although the fugitive emissions smelt ammoniacal, the exact composition of the MCD emissions was not identified and remains unknown. There were also fugitive emissions from the rotary dryer which would have been volatilized pesticides, and the decomposition and combustion products of the soil contaminants including dioxins, benzene and acidic emissions.

Having been closer to the source of the emissions, one might have expected the effects to be more marked in the latter two employees whereas three of the four employees interviewed have some degree of on-going health problems.

The laboratory exposures would have been those due to laboratory reagents (solvents), the soil samples being processed and the emissions from the plant. Solvents might possibly have caused some of the "neurological" health effects, but the quantities handled, even though ventilation was poor, seem unlikely to have been responsible for the ill health. Handling contaminated soil samples did pose a risk of exposure to "pure" or even unrecognised pesticides. Either would have had to be significant. Ingestion is also possible but unlikely. The exposures cannot however be accurately assessed due to the lack of monitoring.

The complex and transitory nature of the exposures of these four people is not readily explicable. Those emissions varied as soil contamination changed and the conditions at the plant changed, which could have been as often as hourly or daily. The potential exposures from the site are, depending on the dose of the agent received, plausible causes of the symptoms seen.

Two other features are worthy of discussion. The first is that only four individuals with symptoms were interviewed. Usually one would be looking for other individuals on the site with similar experiences. There are, however, different thresholds for reporting, which may explain this. The exposures of the respondents here was also, probably, somewhat different to the other workers on the site.

It would also help if there was more uniformity amongst the symptoms described, which have some, but not all, features in common and no readily demonstrable single causative agent. The precise nature of exposure is unknown and to complicate matters, the additive effects of mixed exposures are not well understood or documented. The workers were healthy at the start of the project and had no previous occupational organochlorine exposure. There is some similarity of symptoms amongst these workers with a temporal association

between exposures and development of symptoms. The precise cause is likely to remain evanescent, but the experience of illness real enough.

Summary

There are a number of facts that support the potential for chemical exposure;

- From the start of plant operation there were serious failures of the air emissions system, and other operational difficulties.
- All 4 workers' health deteriorated during the course of their work and those that worked there the longest seem to have had more frequent and significant health effects.
- They were all, according to the PCE report, working at the plant during the period of highest concern, which was prior to, and just after, November 2005. There is some contrary evidence in that the carbon filter failed in March 2005 , and had probably malfunctioned since November 2004. The new regime of monthly carbon changes was operating after September 2005. The worst time may have been November 2004 to September 2005.
- All 4 workers were exposed in the course of their work to toxic chemicals through fugitive emissions, inadequate protective equipment, dangerous work and stack emissions.
- The MOH report specifically acknowledges the likely emission of PM10, PCB, dioxin and benzene of unknown quantity, decomposition products of soil contaminants and products of incomplete combustion.

Recommendations for workers

There is a clear need to provide workers on site with appropriate medical assessment.

APPENDIX 3

DEPARTMENT OF LABOUR INVESTIGATIONS

DEPARTMENT OF LABOUR INVESTIGATIONS

Overview of department responsibilities

The responsibilities of the Workplace Safety and Health Group of the Department of Labour are (Department of Labour website):

> *"Working to reduce work-related death and injury rates, and support employers and employees in productive work. Providing information and guidance to workplaces on occupational safety and health issues and managing hazardous substances. Enforcing health and safety legislation; researching workplace health and safety matters, and providing policy advice to government."*

Information and guidance

Information and guidance is available through department publications, with an option of calling a contact centre or making email contact.

Enforcement

Department of Labour health and safety inspectors investigate the following events:
- complaints about health and safety
- notifications of serious harm, including fatalities
- notifications of occupational disease
- notifications of incidents (accidents that might have harmed someone).

Investigations are undertaken in order to determine:
- causes
- whether action has been taken or needs to be taken to prevent recurrence, and to secure compliance with the law
- lessons to be learnt, both in the workplace involved and in industry at large, and to influence the law and health and safety standards and guidance material
- if there has been any breach of the law, and the appropriate response.

The Department does not investigate every event reported to it. Most of the resources available for investigation are devoted to the more serious events. In selecting which events to investigate, and in deciding the level of resources to be used, the Department takes the following factors into account:
- severity and scale of potential or actual harm
- seriousness of any potential breach of the law
- knowledge of the workplace's past health and safety performance
- enforcement priorities
- practicality of achieving results
- wider relevance of the event, including serious public concern.

The Department uses its enforcement powers when it is unable to get voluntary compliance with the law or the matter is such that a duty-holder needs to be held

accountable for failure to meet minimum standards. Enforcement is used as a complement to other strategies such as engagement, education and enablement.

The Department's response to any observed breach of legislation is to choose the enforcement intervention that will best:
- see hazards eliminated, isolated or minimised quickly and effectively
- influence future compliance with the legislation.

On-site performance

Provision of information

There are a number of references to the Department's involvement with the project recorded in the material supplied. Many of these are extracts from email messages.

In January 2005 there is an email from the Wellington Office Departmental Medical Practitioner commenting on the occupational health monitoring policy, but stressing the need for an adequate hazard identification:

> *"Full hazard identification on site, quantifying ALL RISKS, not only chemical ones, before commencement of work. This includes actual time exposed, i.e. length of shift, length of work week, personal safety, food and water safety, safety during lunch and rest breaks, personal protective gear, decontamination procedures, washing and disposal of contaminated clothing and coveralls, washing and personal cleansing protocols and facilities. (Contaminated clothing can put family members at risk.)"*

An internal Department report states that health and safety systems had been assessed in 2005 by an occupational hygienist but there were no records of the outcome.

Another occupational hygienist subsequently seems to have visited the site in 2006 in response to a claim regarding dust at the site.

Investigation of complaints

Complaints in 2005
One complaint was received in January 2005 about an employee at the site who had strained a wrist at work while using a hammer to tap a silo. The worker was sacked after turning up at work with ACC forms. The worker also complained of a dirty smoko room and that the truck he drove had a loose wheel. After a visit by a workplace inspector, the smoko room complaint was upheld.

Complaints in 2006
A complaint was received from Tahi Street residents as follows:

> *Soil contaminated with pesticides and chemicals is being treated on site at Mapua. The project was due to finish in March, 2006, but could extend to December, 2006. The local authority responsible for the project is Tasman*

District Council, but Ministry for Environment has a governmental interest ([the] General Manager, based Wellington ....).

Part of consent conditions are that particle dusts of a certain size are not permitted across the boundary. There are several monitoring stations, including one on the [complainant's] property. [The complainant] has discovered that the levels have been exceeded (in March 2005) and this is a concern. He says that respirable dust is not measured. Also a concern is that he feels that no-one really knows what the hazardous levels are and what quantities are acceptable – there was computer modelling of dust movements. He has reported that his parents often have sore eyes and sneeze a lot. His children's clothes reek of chemical substances after they have played on their grandparent's lawn, and they "gag". He says that the smell and the dust can be intolerable, but it is not as bad at the moment due to the amount of rain.

There is also constant vibration. [The complainant] feels that the employees are possibly not at great risk – they have PPE, are trained and monitored, and work eight hours a day, five days per week. On the other hand, the residents are uninformed on the level of hazard, dust particle emissions have been exceeded, there is no monitoring of their health, and they suffer 13 hours per day, 6 days per week.

I said I would think about this problem. [   ] and I discussed this with [   ] and [   ] Public Health on 2 August, 2005. They had some input at conception. They see that the issue does raise real concerns and feel that a joint approach is appropriate, and the first step should be a meeting with TDC to assess the situation, particularly the dust monitoring regime.

The investigation log by the assigned inspector details the sequence of events following this complaint.

The MfE site manager was contacted on the 4 and 5 of May with no reply.

The assigned inspector contacted the Department of Labour occupational hygienist who had a copy of the health and safety plan, and arranged to receive a copy of this from him.

The inspector made contact with the company who gave "thorough details of how they are managing their H&S, see attached email outlining this". There does not, however, appear to be a copy of the email on file.

Investigations in 2006
In May 2006, the assigned inspector contacted the company again requesting further details. The letter on file is as follows:

Alleged raised blood test results

Thank you for your co-operation and information that you have provided me to help with my investigation into the claims of raised blood tests. I would like to reiterate that your health and safety plan and induction looks very thorough and I am impressed with the monitoring you are doing to ensure of the safety of your staff.

Further to our phone conversation, I was wondering if you could provide further information as below:

1. What are the blood tests results – are you able to provide an overview of this without identifying individuals?

3. How significant are these elevations (I realise this was discussed on the phone – but I would appreciate it if you could put this in writing).

5. What do you plan to do about this?

Please contact me if you wish to discuss this. I look forward to your response.

A reply was received on 9 May (an email from a worker to the assigned inspector):

Inspector: Just wanted to get back to you relative to the letter you sent to the site re: the blood levels of employees. We have forwarded this on to MfE for action, as they are overseeing the contracts with … [the occupational physician] and Ramazzini who handle the bio-monitoring programme. We are confident they will get back to you shortly.

This was followed up on 23 May 2006 (Email from the inspector to the site):
Hi [   ], just wondering where this is at and when a response will come this way.

From site to the inspector:

[   ] Hi there, thanks for your follow up. I have sent an email to [   ] and [   ] at the Ministry for the Environment. I will also call them shortly and see if I can chase his up.

On the 12 June 2006 the inspector sent several emails requesting a response to the above letter.

*Phone call from the worker confirming she would follow up on this request.
[   ] telephoned the inspector to say that a report would be sent in a
couple of days.*

On 23 June 2006 a letter was sent to the inspector from a department senior
advisor with an email copy to the Department's principal medical advisor.

Alleged raised blood test results

Thank you for positive comments regarding Health and Safety plan.

*Apology for delayed response. Had hoped to include ... [the occupational
physician's] report as felt he is substantially qualified to provide the
answers.*

*Have received an interim report from [the occupational physician] but
have not discussed this with him as he is now overseas.*

Did not wish to delay response any longer.

*Attached summary of results of ... [the occupational physician's] report.
Report focuses on three on-site groups, MfE, EDL and HSE. Background of
some individuals is mentioned. Would like further discussion with ... [the
occupational physician] before releasing full report.*

*Conclusion of the report is "Overall there is no evidence of an increase in
levels of organochlorines in serum with time which poses a concern."*

*... [the occupational physician] advises "In general, as far as Dieldrin and
pp-DDE were concerned, most results were within the range of the
Ministry's study and were of no concern. There was no pattern on increase
with time on site, and no pattern which related to the wearing or not
wearing of protective equipment. Pp-DDT on the other hand, showed a
tendency to respond to on-site exposure in some staff."*

*Also the report "The majority of the staff have results within the range for
non-exposed people and among these showing an increase in pp-DDT with
time, only two had a two or more times increase."*

> *In answer to questions "Have you identified any causes to the raised levels, such as work practices?" and "What do you plan to do about this?"... [the occupational physician] met on site with the workers, discussed exposure routes, skin and respiratory. Information pamphlet given and discussed occupational histories and work habits.*

> *Worker provided with all PPE required and monitoring is on going. Each on site contractor will ensure correct use of equipment. Visit has been educational. Blood monitoring has ensured a strong commitment to wearing the equipment provided.*

On the 28 June 2006 the inspector discussed the results with [   ], who was reported to be "not concerned".

> *On the 4 July 2006 an email from the inspector was sent to the Mapua site advising that [   ] and herself were of the opinion that all practicable steps were being taken and that they did not intend taking the matter further. There was a request to forward the email to [   ].*

August 2006 complaint

On 2 August 2006 an event notification form was received with a complaint from one of the laboratory workers, the details of the event being "concern re dioxins causing .... [the employee's] recently low thyroid". The employee was concerned that the "blood tests were inadequate". The inspector spoke to the MfE site manager, who gave assurances that dioxins were not produced at the site as the mill operated at 120 degrees, not the 260-300 degrees necessary for dioxin formation. He also explained the monitoring and improved work practices.

The inspector consulted [   ], who said that as dioxin was not being produced there was no need to monitor. The occupational physician had been to the lab, but the complainant was not working. The occupational physician also had an appointment to see the complainant in his OSH role, but the complainant did not turn up. There was some discussion with the complainant's doctor, but no time off work.

There followed a discussion of the PID testing carried out. The inspector met with [   ] and the occupational physician. The result was that ammonia was the only hazard identified.

Further monitoring by EDL was discussed and the fact that blood monitoring was negative. The inspector consulted the occupational physician and the occupational hygienist, then wrote back to the complainant, closing the complaint.

In November 2006 a memo (briefing paper) was sent from PCE. This detailed some of the concerns already noted, but contained an occupational health and safety section. OSH advice to MfE stated that (page 5 of memo):

> *"If there is to be significant exposure risk, you have a far greater responsibility under the HSE Act than to just monitor exposure levels and perform entrance and exit exams. You will need to monitor biological effect more closely. If you pick up a raised level at the end of the period, you have failed to monitor the effect of the toxic exposure, and can actually be prosecuted under the HSE Act."*

Department investigation into the Parliamentary Commissioner for the Environment report

As a result of the 2006 complaint a PCE enquiry into the allegations of poor environmental management at the site took place. This was referred to the Department, which undertook an internal investigation in April 2008. This related to three main areas:

- the lack of a site visit by the Department following a complaint made in August 2006 and a biased investigation of that complaint
- the adequacy of the biological and environmental monitoring during the clean-up
- poor communication by the Department to the original complainant.

Lack of site visit

The report noted that the site had in fact been visited on a number of occasions. These included visits in 2005 to assess the adequacy of health and safety systems; an earlier visit in response to a complaint regarding facilities; and a visit by a departmental occupational hygienist in response to a complaint by the public about dust coming from the site. The Department was of the opinion that none of these required enforcement action. Specifically:

> *"reviews of the site's health and safety plans confirmed that they included how risk from hazardous substances on site would be managed. The control measures the company indicated were in place were assessed as adequate to address the workplace hazards. It is worth noting that the maintenance of work practices to these standards, for example enforcing the wearing of personal protective equipment, is a duty of management."*

Adequacy of biological and environmental monitoring

The Department considered that environmental monitoring was outside of its activities.

As regards biological monitoring, the report made two points:

> 1. *The original complainant raised a number of concerns about the adequacy of the biological monitoring programme, including failure to test at the start of an employee's work at the site and irregular follow up. While these failings were evident, the principles of the programme were assessed as sound and the results which were obtained were seen as unlikely to be associated with harm to the employees. As a consequence, the results provide some reassurance that the control measures in place were adequately protecting employees on site.*
> 2. *It is useful to record that the deficiencies noted above are consistent with those seen when running a biological monitoring programme of this type, as in practice such programmes are recognised as being fraught*

with such difficulties. Overall then, the programme of biological monitoring is considered to fulfil the duty required of the employer under the HSE Act.

Poor communication
The response was that:

"This investigation found support for the concern that there were instances of poor communication between Department of Labour staff and the complainant, along with an acknowledgement from both parties of a shared contribution to creating this as an area of concern.

Gaps identified

Provision of information and guidance
There are a number of occasions on which Department staff assessed health and safety plans at the site. There were also site visits. The assessments were carried out by well-qualified staff (an occupational physician and occupational hygienist). The problem in assessing the adequacy of the advice is that there is little information as to what it was.

It is not certain whether this advice was given in order to influence policy (which is a departmental role) or whether the advice was given to help a government department fulfil an operational role. The Department does have occupational health and occupational hygiene experts. However, their role is normally to give specialist back-up to inspectors undertaking advisory or enforcement roles. They do not, in the normal course of events, carry out "consultancy" work for principals and contractors.

It appears that the Department assessed the health and safety systems at the site or at least the MfE plans, as being "adequate" and that EDL had taken "all practicable steps" to control the hazards of chemicals in the place of work. The MfE plans were, in the opinion of the writer, of an "adequate basic" standard at best, and "inadequate" at worst. The EDL plans were clearly inadequate.

There is also evidence that the Department may have considered that some of the procedures carried out that the site required much tighter management:

"If there is to be significant exposure risk, you have a far greater responsibility under the HSE Act than to just monitor exposure levels and perform entrance and exit exams. You will need to monitor biological effect more closely. If you pick up a raised level at the end of the period, you have failed to monitor the effect of the toxic exposure, and can actually be prosecuted under the HSE Act."

There is, to some degree, a confusion in roles between giving direct advice on what needs to be done as distinct to how to do it.

There does not seem to be any reference to the Department publication "Health and safety guidelines on the clean-up of contaminated sites". While not an Approved Code of Practice, "compliance with the provisions contained herein may be taken into account in deciding if all practicable steps have been taken to

ensure that employees at work, or others, are not harmed." It was therefore a guide to best practice and should have been recommended as such.

Investigation of complaints

As stated on the Department website, the role of the Department of Labour on this occasion was to:

> *"investigate complaints and ascertain whether action had been taken, or needed to be taken, to prevent recurrence of the conditions giving rise to the complaint and to secure compliance with the law. Enforcement action is usually taken only when the department is unable to get voluntary compliance with the law or the matter is such that a duty-holder needs to be held accountable for failure to meet minimum standards.*

> *The level of the investigation depends on the severity and scale of potential or actual harm, the seriousness of any potential breach of the law, knowledge of the workplace's past health and safety performance, enforcement priorities, the practicality of achieving results and the wider relevance of the event, including serious public concern."*

The local Department of Labour office had neither the resources nor the expertise to carry out an in-depth examination of events at the site, but this was not the role required at the time. The local managers did call on appropriate expertise. With the benefit of hindsight, a site visit would have been valuable.

The site had been visited on a number of occasions and the Department was of the view that the management plan was adequate and included how risks were being managed. Workplace controls were also assessed as being adequate, which they were. There is no indication of how adequate, as only an in-depth investigation would reveal the inadequacies of the EDL plan. To this extent there was no failure to meet minimum standards on the part of MfE. The role that the site visits played in developing the health and safety planning is not clear.

The Department of Labour report states that environmental monitoring was "outside of its activities", which would be true of the environmental monitoring designed to ensure resource consent compliance and assess the public health impact. The environmental monitoring programme also included some elements of occupational monitoring. The environmental monitoring was not specifically designed to protect workers and was the only routine monitoring being carried on. It is common for employees to perceive monitoring as being protective of health. This is an understandable misconception.

The finding was that the principles of the biological monitoring programme were indeed sound. But this was not really the question, it is how the results were used to inform control procedures and monitor their effectiveness. The report recognised that such monitoring was fraught with difficulties, but that it fulfilled the duty of the employer under the HSE Act.

Implications of the gaps

There were a number of well-qualified occupational health professionals giving advice, at various times, regarding health and safety at the Mapua site. It does not seem that there was any co-ordination of this effort, which is liable to lead to miscommunication or duplication of effort.

There well may have been some confusion regarding the roles when another government department was involved and opportunities for conflict of interest. While the provision of information is, quite clearly, a Department of Labour responsibility, an on-going advisory role may lead the parties to assume that the relationship has evolved into more of a consultancy role and that on-going compliance will be the result. The danger in this, especially where more than one person is giving the advice, will be a loss of continuity, a failure to appreciate the health and safety planning in the depth required of such a project, and fragmentation of the effort. MfE may have been considering that they were getting expert advice and guidance (which they were) but it would have been much better practice to refer MfE for independent advice on operational matters. A more serious situation would have arisen had enforcement action between two Crown entities been required.

As regards a detailed site visit, had this been carried out with full information about the process, with a team effort, the outcome may have been much better. An important opportunity was missed.

The Department's focus when carrying out investigations is usually upon whether a principal or contractor is failing to meet minimum standards, and the depth of the investigation will depend on a number of criteria, including the severity and scale of the harm. The investigation also takes place at one point in time, using limited information. The health and safety practices were adequate, but this may have misled the site management that they were good, which, bearing in mind the nature of the site, they were not.

The reasons for the environmental monitoring should have been made explicit to employees. It was not designed to protect employee health but was part of the resource consent process. This failure resulted in misconceptions by the staff. While the biological monitoring programme may have fulfilled the duties of the employer, it was not (as employed) an effective tool for managing the risk.

APPENDIX 4
TOXICOLOGY

TOXICOLOGY

Organochlorine compounds

The organochlorine group can be divided into several subgroups:

DDT and its analogues

These insecticides were widely used in agriculture and malarial control programmes for about 30 years. Since the 1960s they have fallen out of favour, largely because of their persistence in both the physical environment and biological organisms, with accumulation in the food chain.

In spite of some structural similarity and shared properties (such as high fat and low water solubility), there is considerable variation in toxicity and it is best to consider them on a one-by-one basis.

Although all of these compounds are fat soluble and absorbable through the skin, the efficiency of skin absorption varies considerably. For example, skin absorption of Dieldrin is about 50% that of gut absorption, whereas with DDT it is considerably lower, whether in the solid form or in solution. Respiratory tract absorption is minimal unless there are extremely minute particles, and as the vapour pressure of these compounds is low there is little vapour inhalation.

In general, the signs and symptoms of poisoning from the chlorinated hydrocarbon insecticides are similar, reflecting nerve hyperactivity. In many cases a convulsion may be the first sign of toxicity, although with DDT this is almost invariably preceded by characteristic muscle tremor. The safety record for DDT is quite good given its extensive use in the past. This is partly because skin absorption is not particularly high and indeed is very limited for the dry powder form. While the dust can cause eye irritation, it is not a very irritant chemical. Skin rashes from the liquid preparations are believed due to the organic solvents.

DDT

The IUPAC name for DDT is 4,4'-(2,2,2-trichloroethane-1,1-diyl)bis(chlorobenzene), the trivial name being dichloro-diphenyl-trichloroethane. It is almost insoluble in water, but dissolves well in solvents and oils.

Commercial grade DDT is a mixture of isomers. The major component, around 77%, is the p,p isomer. The o,p' isomer is also present in significant amounts at 15%.

Dichlorodiphenyldichloroethylene (DDE) and dichlorodiphenylduchloroethane (DDD) make up the remaining 8%, and are also the major metabolites and breakdown products of DDT in the environment. Total DDT refers to the sum of all DDT-related compounds in a sample.

DDT is an insecticide, and in New Zealand a major use was to destroy soil-dwelling pasture pests such as grass grub.

Figure 13. DDT from Boul {Boul, 1995 #1}

p,p'-DDT   p,p'-DDE   p,p'-DDD
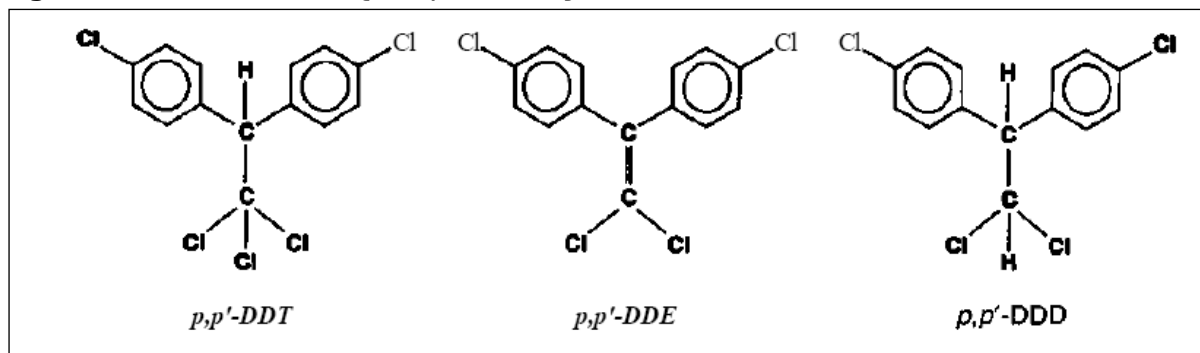
Exposure, absorption, distribution and excretion

People are not exposed to DDT or DDD individually but to the mixture of the compounds which technical DDT contained. The several routes of exposure of DDT include inhalation, oral and dermal. The primary route of occupational exposure is via the dermal route (due to mixing and application), with some ingestion due to mucociliary clearance. DDT is non-volatile, and though inhalation of spray drift might occur during application, this is less of a risk. Once absorbed, it is readily distributed to all body tissues and stored in relation to organ lipid content.

Detoxification is by "phase 1" microsomal enzyme actions involving oxidation, reduction and hydrolysis. In humans, ingested DDT undergoes reductive dechlorination to DDD, which is further degraded and readily excreted as DDA. The reduction to DDE takes place at a slower rate. Following detoxification, the metabolites are excreted in the conjugated form in urine and bile.

Mode of action

The mode of action of DDT is to cause hyperactivity in peripheral sensory organs (by opening sodium channels), which causes hyperactivity. DDT is also primarily suspected of influencing reproduction and development through its interaction with steroid hormones receptors for estrogens and androgens.

Acute toxicity

DDT is classified as "moderately toxic" by the US National Toxicology Programme, the oral LD_{50} being 113 mg/kg. With regard to oral exposure, there has been only one case of fatal poisoning recorded and that was an ounce of 5% DDT kerosene ingested by a one-year-old child. Doses as high as 285mg of DDT per kilogram of body weight have been ingested without fatal results. Occupationally, it has seldom been associated with deaths. The acute toxic effects are nausea, vomiting, parasthesiae, tremors and convulsions. If death does occur, it is due to respiratory failure.

The respiratory effects of inhalation seen have been limited to irritation of the nose, throat and eyes. The only specific study looking at long-term inhalation exposure was a case control study of lung cancer in a Uruguayan work force which showed a non-significant odds ratio of 1.6 (95% CI 0.9,4.6).

Chronic effects of DDT

Haematological: There have been a number of haematological studies of DDT, looking at various parameters. The majority of these included only small numbers of workers, and as a result the statistical power was low. In consequence, none of the haematological parameters seem to be correlated well with DDT or DDE blood levels. One extensive study did, however, measure pesticide concentration of 2600 individuals and matched these to 1000 controls with "minimal" exposure to pesticides. Many haematological parameters were measured, but none of them had a correlation co-efficient with DDT and/or DDE blood levels greater than 0.17, which is low.

Immunological: There have been a number of studies examining the immunological and lymphoreticular effects of DDT compounds.

In one study a group of 23 men exposed to DDT through fish from the Baltic Sea were compared with 26 men with almost no fish consumption. A full range of blood parameters were measured, including lymphocyte subsets. Of all the things that were looked at, the level of the natural killer (NK) cells was reduced in the fish eaters, but this was not statistically significant. Weekly intake of fatty fish correlated significantly with the reduction of NK cells.

One of the larger studies was of 302 individuals who lived near a waste site in North Carolina.{Vine, 2001 #33} The researchers tested for 20 organic chlorines but only DDE was detected. The concentration in the plasma of these volunteers was 2 parts per billion and the highest concentration was 32 parts per billion. The outcomes that they evaluated included white cell count, immunoglobulin measurements, and a test of immune secretion, mitogen-induced lymphoproliferative activity (assayed by concanavalina A). When the DDE levels were categorised, subjects at higher levels had lowered nitrogen-induced lymphoproliferative activity and slightly increased lymphocyte immunoglobulin levels.

There have also been numerous studies of DDT effects on animal immune function. Some of these have shown immune suppression of the humeral and cell mediated components.

Overall, the studies suggest that there is some evidence that the immuno-competence of animals can be affected by D.D.T, but the results in humans is inconclusive.

Endocrine effects: The endocrine effects of DDT and related compounds have been the subject of controversy. In animals it does cause an adverse effect in the reproductive system of both females and males, thought to be because some of the DDT compounds bind to receptors for oestrogens and androgens.

There have been no reports of thyroid effects in humans (but see a pregnancy study mentioned later), and laboratory animals seem to be similarly resistant.

Reproductive effects: There have been many studies of the reproductive effects of the DDT compounds. A study of 120 individuals who have had miscarriages and 120 controls showed mean DDT blood levels were similar.{Leoni, 1989 #34} Another study of 89 who had had at least two miscarriages found that 13% of them had DDE blood levels above the reference value of 2.5 parts per billion. The mean value was 1.2 parts per billion and the range was between 0.01 ppb and 8.6 ppb. {Gerhard, 1998 #35}

Another study looked at differences between women delivering at full term and premature infants. Twenty-three of the women who delivered early had infants with mean DDE blood levels of 19-22.1 ppb, but the 44 women who went to full term had infants with DDE blood levels of 4.9 – 6.1 ppb. {Oleary, 1970 #36} Other studies were similar and showed that total DDT was associated with pre-term birth and spontaneous abortion. Other studies have looked at endometriosis. One case control study looked at six women with endometriosis and six women in a control group without endometriosis, but no association was found in relation to plasma DDT and the condition.

The link between oestrogen and DDT is quite strong. This activity seems to be due to the o-isomer present as a 13% - 20% contaminant. The relationship has of course been shown most strongly in laboratory animals, both male and female.

There is also evidence the DDE is an androgen receptor antagonist. The effect is to reduce the weight of the seminal vesicle and the weight of the prostate in rats. In contrast, reproductive studies had not indicated reproductive toxicity.

Liver: There is conclusive evidence that the liver is considered a target for DDT in laboratory animals. Studies of DDT-exposed workers show increased activity of hepatic metabolic enzymes. These have included increase of xenobiotic metabolism through looking at the metabolism of antipyrine in which the half-life of antipyrine was shorter in workers exposed to insecticides than in controlled subjects.

Based on the data that I have seen, there is no conclusive evidence that DDT, DDD and DDE cause adverse liver affects in humans. Epidemiological studies of DDT-exposed workers do, however, show increased activity of hepatic microsomal enzymes.

Nervous system: The nervous system is one of the main target organs for DDT, this being the mechanism for its use as an insecticide.

Some of these early studies were actually carried out by exposing individuals to small amounts of DDT in pharmaceutical formulations. Some of the levels have been high. In one study, volunteers were exposed to levels of up to 1500 milligrams of DDT orally. Six hours after the exposure to the higher levels, people experienced a prickling of the tongue and areas around the mouth and nose, disturbance of equilibrium, dizziness, confusion, tremors, headaches, fatigue and severe vomiting. The volunteers did, however, recover within 24 hours. In 2001, chronic neurological studies on retired malaria control workers were reported. The

studies on 27 former workers and 27 non-exposed controls showed that the exposed group had overall poor neurological performance, verbal attention and visual motor speed. Visual motor speed and verbal attention differed mostly between groups.

As regard to musculoskeletal effects, the relationship between DDT and bone density has been studied, but no relationship was found.

Longer-term health effects have included morbidity and mortality in cohorts of male and female workers without any significant excess in mortality.

Looking at the overall evidence, human studies suggest that high DDT/DDE presence may be associated with hormonal end-points that include effects on pregnancy and fertility. There also seems to be an association with having pre-term infants and "small-for-dates" infants.

The International Agency for Research on Cancer (IARC) classifies DDT as a Group 2B, or "possible" human carcinogen.

Workplace exposure standard for DDT
A set of values, the Workplace Exposure Standards (WES), are published by the Department of Labour. These are levels of workplace exposure at which most individuals will not show significant effects. Most are developed internationally and evaluated by the department as regards applicability in New Zealand. For chemical substances they are measured in air, the airborne concentration being expressed in mg/m^3 for aerosols and parts per million (ppm) for vapours and gases.

Dichlorodiphenyltrichloroethane has a WES of 1 mg/m^3 with a skin notation (significant absorption may take place through the skin).

Biological monitoring
Biological monitoring is the measurement, in a biological sample, of the substance itself, or the metabolite of a substance, to which a person is occupationally exposed.

To fulfil its role, biological monitoring needs a set of values to act as a reference standard. These values are known as Biological Exposure Indices (BEIs), and, like WESs, they are published periodically by the Department of Labour.

BEIs are used as a guideline in evaluating potential health hazards. They represent the levels of determinants that are most likely to be found in biological specimens collected from a healthy worker who has been exposed to chemicals to the same extent as a worker with inhalation expose to the chemical substance WES. If the BEI has been exceeded, then the WES will have been exceeded. BEIs are useful in assessing exposure from all routes.

Fundamental to the use of both WESs and BEIs is the existence and knowledge of dose-effect and dose-response relationships. In humans, there is limited knowledge about the relationship between exposure and internal dose in

pesticides, so BEIs have not been set and biological markers cannot be used to assess quantitative exposure to this group of substances. Determination of OCPs in blood can, however, be used to assess exposure over short or long periods in comparison to baseline levels to assess whether these have risen, suggesting "excess" exposure, which is, strictly speaking, more of a qualitative index.

Biological monitoring of OC exposure can be carried out by measuring intact pesticides or their metabolites in blood or urine. They also cause induction of microsomal enzyme systems, and this has been used as a measure of a "health effect" in some exposed workers.

The biological half life (the time that it takes for the levels to decrease by one-half) of Dieldrin in blood is 267 days. For DDT in adipose tissue it is 3.4 years. Lindane and chlordane have a 10-20 day half life, but that of endrin is 24 hours. The isomers of benzene hexachloride and heptachlor have half lives in the order of Dieldrin.

The concentration of PCBs and its metabolites have been measured in the general population and healthy workers exposed to DDT.{Maroni, 2000 #20} Total DDT in the general population lies between 0.01-0.07 mg/l. In healthy exposed workers it is between 0.35-1.36 mg/l (a 20-fold increase over population values at the higher level).

Some biological limit values have been proposed. In particular, a blood Lindane concentration of 20µg/l and plasma serum concentrations of 25µg/l have been indicated as the upper "no-effect" level for neurological signs and symptoms (DFG, 1998). Absence of induction of liver microsomal enzymes was shown:
- for endrin at urinary anti-12-hydroxyendrin concentrations below 130 mg/g creatinine (Van Sittert and Tordoir, 1987a)
- following repeated exposures to DDT, at DDT and DDE serum concentrations below 250 µg/l (Kolmodin-Hedman, 1974)
- in repeated Aldrin and Dieldrin exposures, at Dieldrin blood concentrations below 100 µg/l (Van Sittert and Tordoir, 1987a).

Population levels of OCPs

Because of their environmental persistence, the OCPs are ubiquitous and found at detectable levels in those not occupationally exposed.

The Organochlorine Programme of the Ministry for the Environment carried out in 1996 and 1997 as part of the National Nutrition Survey gives some baseline levels for New Zealand. In this study, serum was pooled from people having similar ages and ethnicity for various regions (strata). {Buckland, 2001 #2}

The most frequently detected pesticides in serum from the New Zealand population were:

- pp'-DDE, which was detected in all strata: concentrations were in the range 413–2780 µg kg-1 lipid, with median and mean concentrations of 919 µg kg-1 and 1080 µg kg-1 lipid respectively

- Dieldrin, which was detected in 57 out of 60 strata: concentrations were in the range
- < 8–28.4 µg kg-1 lipid, with median and mean concentrations of 11.5 µg kg-1 and 14.2 µg kg-1 lipid respectively
- β-HCH, which was detected in 40 out of 60 strata: concentrations were in the range
- < 7–73.1 µg kg-1 lipid, with median and mean concentrations of 10.7 µg kg-1 and 19.7 µg kg-1 lipid respectively.

Of the remaining pesticides analysed, pp'-DDT was detected in 18 strata at a maximum concentration of 49.2 µg kg-1 lipid. All other pesticides were either not detected, or detected on less than 10% of occasions (i.e. detected in less than six strata). Of these, γ-HCH was detected in one stratum at a concentration of 91.1 µg kg-1 lipid, HCB was detected in four strata at a maximum concentration of 53.6 µg kg-1 lipid, and trans-nonachlor was detected in three strata at a maximum concentration of 8.4 µg kg-1 lipid.

Table 7. Concentration of organochlorine pesticides in the serum of New Zealanders

| Pesticide | Number of positives | Concentration, µg kg^{-1} lipid weight basis[1] | | | |
		Minimum	Maximum	Weighted median	Weighted mean (n = 60)[2]
β-HCH	40	< 7	73.1	10.7	19.7
γ-HCH	1	< 5	91.1	nc	nc
HCB	4	< 20	53.6	nc	nc
Aldrin	0	< 3	< 8	nc	nc
Dieldrin	57	< 8	28.4	11.5	14.2
Endrin	0	< 5	< 9	nc	nc
Heptachlor epoxide	0	< 3	< 8	nc	nc
Oxychlordane	0	< 3	< 8	nc	nc
t-Nonachlor	3	< 5	8.4	nc	nc
pp'-DDE	60	413	2780	919	1080
op'-DDT	0	< 10	< 20	nc	nc
pp'-DDT	18	< 20	49.2	nc	nc
Mirex	0	< 3	< 10	nc	nc

1. For any individual pesticide, calculation of the weighted median includes LOD values and calculation of the weighted mean excludes LOD values (see Appendix B, Section B1.9).
2. n = 59 for β-HCH, γ-HCH, aldrin, heptachlor epoxide, oxychlordane, t-nonachlor and op'-DDT
   n = 55 for endrin.
nc = Not calculated (median or mean concentration reported only if a congener was detected on more than 66% of occasions across all strata).

Source: Buckland, 2001 #2

The amount of these substances in serum depends on the lipid content: more lipid, more pesticide. These levels are "lipid corrected", and therefore numerically larger than the "crude" value of DDT reported in serum, the reference level being about 120 and total lipids being 600mg/dL or 6-7 g/L.
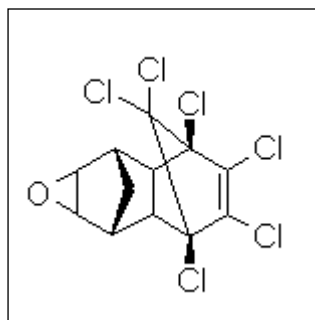
Aldrin and Dieldrin

Aldrin is a broad spectrum soil insecticide. After absorption it is metabolized to Dieldrin.

Dieldrin

This is another chlorinated hydrocarbon which has occasionally caused poisoning with exposures during manufacture or use. Symptoms may develop with blood levels of around 0.15-0.2mg/l and above. The serum half-life has been estimated as nearly one year, so that a worker with blood tests around the above values may remain for some time near this "threshold" even if exposure is a little sporadic. It only needs a sudden extra exposure to "tip over" into a toxic blood level. The average fat level in symptomatic workers was 6.12 ppm in one study while 0.14–0.21 ppm has been quoted as average levels in non-exposed subjects.

Symptoms occurring with Dieldrin poisoning can include headache, dizziness, blurred vision or diplopia, abnormal sweating, loss of appetite, nausea, insomnia and changes in personality. As with other organochlorines, tremors and convulsions can occur.

Figure 14. Formula for Dieldrin

Workplace exposure standard

The WES for Aldrin is 0.25 mg/m^3 with an Sk and A3 carcinogen notation. The WES for Dieldrin is the same, but without the A3 designation.

There have been some occupational studies of exposure to Aldrin and/or Dieldrin. In healthy male workers without clinical or laboratory changes, blood and serum concentrations of 1.8-100 and 1.5-182 μg/l were measured.

No cases of intoxication have been measured at levels below 200 μg/l, and a limit value of 100 μg/l has been proposed.

Lindane

Lindane is a broad spectrum insecticide containing > 90% of γ hexechlorocyclohexane (HCH). It is in WHO Class II, moderately hazardous. It has been used in a variety of applications, including public health (against ectoparasites, including the scabies mite) Technical grade HCH contains 60-70% α HCH with around 10-15% of the other isomers.

Lindane has about twice the toxicity of DDT, via the oral route. Skin absorption may approach 10% with prolonged exposure.

There have been few serious occupational poisonings with Lindane, but the symptoms when they do occur are similar to those from other chlorinated insecticides such as Dieldrin. These include headache, vomiting, depression, sweating, tremor, increased reflexes, and convulsions. Electroencephalographic changes can precede and therefore predict clinical epileptiform activity.

HCH concentrations have been measured in occupationally exposed workers. Sprayers of a 4% solution had mean concentrations γ HCH of 6.4–9.9. Industrial workers had higher levels, with γ-HCH between 16–57. Most workers had symptoms of peripheral neuropathy and EEG changes.{Maroni, 2000 #20}

Haematopoietic responses to pesticide exposure are rare, but there have indeed been reports of haemolytic anaemia and more severe responses such as haemolytic anaemia. These were summarised in a 1978 paper by Hamilton et al. The authors proposed that some individuals might be genetically susceptible, or predisposed through "prior sensitisation through enzyme systems". {Hamilton, 1978 #29}

Workplace exposure standard
The WES for Lindane is 0.1 mg/m^3 with Sk and A3 carcinogen notations.

Hexachlorobenzene

Hexachlorobenzene is a fungicide previously (up to about 1965) used as a seed treatment. It was also formed as a by-product of other pesticide manufacture and in the incineration of waste. It is a white crystalline solid insoluble in water.

Figure 15. Formula for hexachlorobenzene
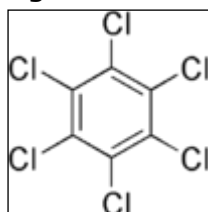
Toxicokinetics
Most data comes from ingestion, but limited data from a Spanish organochlorine plant shows HCB in the ambient air caused increased serum levels in the local population.{Department of Health and Human Services, 2002 #8}

> "Following complaints of odour, approximately 40 air samples were collected in July and November of 1989 and May and October of 1992 at diverse sites in the village [of Flix]. As a control, five air samples were collected in the city of Barcelona. Average air levels of hexachlorobenzene in Flix (35 ng/m3) were over 100-fold higher than in Barcelona (0.3 ng/m3), while other organochlorines were found at similar or lower concentrations in Flix than in Barcelona. Corresponding to the high air levels, it was found that residents of Flix had unusually high serum levels of hexachlorobenzene (mean of 39.8 ng/mL based on a total number of 604 tested) in comparison to populations in Barcelona (mean=4.13 ng/mL, n=100), the United States (mean=0.19 ng/mL, n=370), Croatia (mean=1.00, n=15), and Germany (mean=1.12, n=6). Serum levels of

other organochlorines in Flix residents were much lower than hexachlorobenzene levels and did not differ from other populations. Among Flix residents, serum hexachlorobenzene levels were several fold higher in factory workers (mean=93.4 ng/mL, n=185) than other residents (mean=16.9 ng/mL, n=419). Factory workers were presumably exposed to much higher air levels of hexachlorobenzene than other village residents, and some may have had dermal exposure as well."

HCB is slowly metabolised to pentachlorophenol, and 4–6% of HCB is excreted per day.

Toxicity

There is no information on the acute health effects in humans, but subacute effects were shown when 2–3000 people in Southern Turkey (Anatolia) developed porphyria cutanea tarda after eating bread made from seed grain which was treated with HCB and intended for planting, not consumption. Skin lesions, hepatomegaly and thyroid enlargement (without hyperthyroidism) were the principal clinical findings. {Cam, 1963 #6} Neurological findings included loss of appetite, tremors, convulsions, and "weakness that often made it impossible to eat with a knife and fork, rise from a squat, or climb stairs".

A 20-year follow-up on a group of these patients found that neurological symptoms persisted in adults who had been exposed as children, and included weakness (62–66%), paresthesiae (spontaneous tingling or burning sensations, 55%), sensory shading (graded sensory loss that diminishes upon testing more proximally and is indicative of polyneuropathy, 61–63%), myotonia (delayed muscle relaxation after an initial contraction, 38–50%), and cogwheeling (irregular jerkiness of movement due to increased muscle tone as seen in Parkinson's disease, 29–41%). {Cripps DJ, 1984 #7}

The thyroid is a target organ for HCB, with hypothyroidism shown in rats with high levels of intake. There have been no positive findings as regards the immune system,

It is an IARC group 2B carcinogen (possibly carcinogenic) to liver kidneys and thyroid.

The "No Observable Effect Level" for humans (40-year exposure) was 0.000035 mg/m^3.

Workplace exposure standard

There is no WES for HCB, but the American Conference of Governmental Industrial Hygienists (appendix 6 of the WES book) has set a Threshold Limit Value (TLV) of 0.002 mg/m^3.

Chlorophenoxyacetic acid herbicides, phenoxy herbicides

The chlorophenoxyacetic acid herbicides and the phenoxy acid herbicides (phenoxyacetate herbicides, PHE) are used as herbicides in agriculture and silviculture. The most commonly used are 2,4- dichlorophenoxyacetic acid (2,4-D), 2,4,5- trichlorophenoxyacetic acid (2,4,5-T) and 4-chloro, 2-

methylphenoxyacetic acid (MCPA). 2,4,5-T has been banned because of contamination with 2,3,7,8-tetrachlorodibenzodioxin (2,3,7,8 TCDD, or dioxin) formed during the manufacture of a feedstock precursor, trichlorophenol. Production runs in early years had higher levels of dioxin than latterly.

PHE herbicides are poorly metabolised, mostly being excreted unchanged in urine.

Toxicity
The PHE group have low acute toxicity, with symptoms at levels in excess of 400mg/l. It is in the WHO class II "moderately hazardous" category, with nausea and vomiting being the primary symptoms. There is little information on long-term health effects.

Monitoring
The intact compound can be measured in urine or blood.

Maroni et al report:{Maroni, 2000 #23} "In an extensive occupational monitoring programme undertaken in Australia during the years 1979–1982, about 3000 urine samples were analysed for herbicide residues (Simpson, 1982). The subjects included pesticide factory staff, pest control operators, farmers, park workers, and others potentially exposed to 2,4-D. Only 27 samples contained more than 1 mg 2,4-D/l (highest value 31 mg/l), with most subjects showing urinary concentrations between <0.001 mg/l (analytical detection limit) and 0.1 mg/l. On the basis of biological monitoring studies carried out on farmers and professional spraymen, Kolmodin-Hedman et al. (1979, 1983a) concluded that urinary 2,4-D concentrations up to 0.5 mg/l (after adjustment to a specific gravity of 1024) were indicative of good work practices."

Dioxins and related compounds

Dioxins are a group of environmentally persistent chemicals that share similar chemical structures and mechanism of toxicity. There are three closely related families, the polychlorinated dibenzo-p-dioxins, polychlorinated dibenzofurans, and polychlorinated biphenyls.

Occurrence

Dioxins are ubiquitous, exist in the environment as complex mixtures, and are largely the result of formation as by-products of combustion and industrial processes, occurring particularly when carbon-containing compounds and chlorine are both present in the reaction. Man-made sources outweigh the natural ones. As described previously, a major man-made source was trichlorophenol (TCP), a feedstock used to manufacture phenoxyacid herbicides including 2,4-dichlorophenoxyacetic acid and 2-4-5 trichlorophenoxyacetic acid (2,4D and 2,4,5T).

Acute toxicity

Seventeen of the dioxins are thought to be toxic, and this toxicity varies; 2,3,7,8-tetrachlorodibenzo-p-dioxin, abbreviated as 2,3,7,8-TCDD or TCDD and

commonly referred to as dioxin, is the most toxic, and is in fact the gold standard against which the others are measured, in toxic equivalents (TEQs).

The toxicity of dioxins varies tremendously, the LD_{50} in the dog (oral intake) being 1 ug/kg but that in the hamster being 1,157-5,051 ug/kg.

The mode of action is through binding to a protein on the cellular membrane, the Ah receptor. This is thought to account for the large differences between and even within species, so it is difficult to say how an individual will react – the hamster being a good example. In animals, there seem to be immune, reproductive and developmental effects, with enzyme and sometimes hormonal induction. Dioxins are also carcinogenic in animals, but seem to be promoters rather than initiators.

The effect of acute exposure has been seen following industrial accidents. The most famous of these was probably Seveso in Italy, when a TCP reactor at the Icmesa plant exploded, involving 187 workers. The largest – at Monsanto/Nitro, Virginia, in 1949 – involved TCP and exposed 228 employees.

The major effect of acute exposure is chloracne, a severe inflammatory skin condition with multiple closed comedones and pale yellow cysts that typically appear on the skin below and lateral to the eye and behind the ear. Hepatotoxicity can also be seen, with evidence of transient neuritis and hyperlipidaemia.

The former Ivon Watkins-Dow (IWD), now Dow AgroSciences (DAS), chemical plant at Paritutu, New Plymouth, manufactured 2,4,5-T from 1962 to 1987. The chemical was used extensively in New Zealand to control gorse.

Chronic toxicity

Based on animal studies and information on the mechanism of carcinogenesis, 2,3,7,8-TCDD is considered by the International Agency for Research on Cancer (IARC) to be a Group 1 carcinogen (i.e. known to be carcinogenic to humans). The human evidence is, however, "limited" because some but not all studies have been positive.

The main concerns, through study of Vietnam veterans and occupational cohorts, lie with the causation of Hodgkin's disease, non-Hodgkin's lymphoma, soft tissue sarcoma, and chronic lymphocytic leukaemia. There is some evidence that other cancers such as lung cancer and multiple myeloma may also occur. Some studies have found small excesses (with relative risks in the region of 1.2 and 1.3, some non-significant) of all cancers combined. Other studies have been negative. An analysis of the New Zealand component of the IARC study, which looked at herbicide production workers and sprayers in New Zealand, showed a 24% non-significant excess of all cancers combined for production workers, with a significant excess for multiple myeloma based on three cases, but reduced cancer mortality for sprayers.

Occupational levels

Serum dioxin levels at the DAS New Plymouth site were studied by inviting all current and former workers who worked at the plant during trichlorophenol (precursor to 2-4-5-T) manufacture still living within 75km of the site to participate in a serum dioxin analysis. Sixty eight percent of the eligible workers volunteered yielding 346 TCDD samples. This represented 22% (346/1599) of the total study population.{McBride, 2009 #24}

Seventy percent (241/346) of the serum sample participants were exposed workers. In the total study population, 71% (1134/1599) of the workers had potential exposure. The 2007 serum lipid-adjusted TCDD levels for workers with exposure to TCP or 2,4,5-T averaged 9.9 ppt. The highest levels were found in the TCP operation (23.4 ppt), particularly those involved in an accidental release in 1986 (37.9 ppt). The unexposed workers averaged 4.9 ppt, which is very close to what would be considered the New Zealand background dioxin level of 3.9 ppt for persons of similar age

Exposure standard

There is no WES for dioxins.

Triazines and other related nitrogen-containing pesticides

Atrazine is the most typical compound in this group, and is used as a herbicide.

Toxicity

Atrazine is of "slight" toxicity. Long-term consumption of high levels of atrazine has caused adverse health effects in animals, including tremors, changes in organ weights, and damage to the liver and heart.

Workplace exposure standard

The WES for Atrazine is 5 mg/m^3

Monitoring

Both the intact substance and metabolites can be detected in blood and urine. There is little information on the relationship between internal and external dose.

Metals

Lead

Occurrence and uses
Lead is a bluish-grey metal which is resistant to corrosion, malleable and heavy. It occurs in a large number of minerals, most notably galena (PbS), from which ore the lead is extracted by roasting and reduction. Lead is the most widely used non-ferrous metal, with 60% used in car batteries, approximately 13% in pigments and the remainder in alloys, particularly solder type products.

Exposures

Occupational exposure to lead occurs during lead smelting and refining, where exposures may be quite high. Environmental exposures occur from disposal of lead acid batteries and lead-based paint residues.

Lead absorption

The main route of lead entry is through the respiratory tract, where a particle size of less than 5 microns in size and a high respiratory rate increase the risk of absorption. Although the respiratory tract is the primary route of absorption, gastrointestinal absorption through such poor hygiene practice as smoking during work and eating meals without prior hand washing can substantially add to exposure.

About 40% of inhaled lead is absorbed, but the absorption from the gastrointestinal tract varies, being typically 10 to 15% of the dose in adults but up to 50% in children. Following absorption, lead is 90% bound to haemoglobin, and less than 10% is carried in the plasma where it is available for transport to the tissues. This lead is then distributed primarily amongst the blood, the soft tissues including kidney, bone marrow, liver and brain, and mineralising tissues such as bones and teeth. In adults, bones and teeth contain about 95% of the total body burden. This body burden contains a labile component which readily exchanges lead within the blood and an inert pool.

This "inert" pool does at times pose a special risk because it can be mobilised at times of physiological stress and may increase the blood lead level to the extent that toxicity ensues. The half-life differs in these varying tissues, in blood being 25 days and in soft tissues 40 days. In bone, it may be very stable with a half-life of more than 25 years. This is why even though an individual's blood-lead level starts to decrease after exposure the total body burden may still remain high, depending on the duration of exposure.

Lead toxicity

The major biochemical effects of lead can be classified into three groups. Firstly, it has a high affinity for sulfhydryl groups. There are several enzymes concerned with haem synthesis which this affects. Secondly, lead affects nucleic acids, both DNA and RNA, by mechanisms that are not yet clear. Lastly, lead has interactions with cell membranes, including interference with the sodium potassium pump. This has been suggested as the biochemical basis for a variety of lead-related toxic effects.

Lead toxicity can be manifested in both chronic and acute forms, and the clinical picture varies from subclinical biochemical abnormalities to severe clinical emergencies. One of the most important target organs is the nervous system. In severe poisoning (which hopefully is only rarely found these days) there is a global and profound disturbance of the central nervous system. The symptoms include fits and may result in coma. A more moderate exposure causes subtle changes that may not be immediately obvious. These include aches and pains, deficits in memory, sleep disturbance and changes in personality, with increased irritability and anxiety. These neurological effects are much more marked in children, which is why the ban on lead in petrol has come into place. In children,

because of developmental effects, childhood lead exposure may have permanent effects that result in lower IQ scores. Peripheral nervous system damage is rarer these days, but the resultant neuropathy is primarily of motor nerves, and wrist drop is the classical sign. All of these symptoms may be due to interference with neurotransmitters.

Because of the interference with sulfhydryl enzymes, lead toxicity is associated with anaemia due to inhibition of haem synthesis. These include interference with ALA-dehydratase and inhibition of the incorporation of Fe^{2+} into the protoporphyrin molecule (ferrochelatase). These changes must be fairly severe before overt anaemia is produced, but detection of these biochemical changes can be useful in the biological monitoring of lead exposure. Lead also has an effect on the kidneys, which is the main route of excretion. Lead nephropathy may give proximal tubular damage, and long-standing exposure to lead may result in diffuse interstitial fibrosis and renal failure.

The reproductive and developmental effects are very important in that lead readily crosses the placenta and places the foetus at risk. This was first noticed as an increased frequency of miscarriages and still births in women working in the lead trades at the end of the 19th century, but nowadays there is evidence that prenatal exposure to low lead levels may reduce birth weight and increase the rates of premature births. There is also some evidence that chronic exposure to lead may reduce sperm counts and fertility in males.

Clinically there is continuum of signs and symptoms associated with lead toxicity, as summarised in below.

Lead toxicity
Mild toxicity associated with lead exposure includes the following:
- myalgia or paresthesia
- mild fatigue
- irritability
- lethargy
- occasional abdominal discomfort.

The signs and symptoms associated with moderate toxicity include:
- arthralgia
- general fatigue
- difficulty concentrating
- muscular exhaustibility
- tremor
- headache
- diffuse abdominal pain
- vomiting
- weight loss
- constipation.

The signs and symptoms of severe toxicity include:
- paresis or paralysis

- encephalopathy, which may abruptly lead to seizures, changes in consciousness, coma and death
- lead line (blue-black) on gingival tissue
- colic (intermittent, severe abdominal cramps

Dose-response information
There is a well documented dose-response relationship for lead.

Impaired short-term memory, concentration, reaction time, mood, verbal concept formation and visuospatial functions may appear at ≥ 1.95–2.45 umol/L (≥ 40–50 ug/dL). Slowed nerve conduction velocities (e.g. small motor fibres of the ulnar nerve) can occur around ~1.45–3.4 umol/L (~30–70 ug/dL) but peripheral neuropathy (muscle weakness with minimal sensory loss) is rare below 2.90 umol/L (<60 ug/dL). Overt neurological signs are not usual until levels exceed 2.90–3.90 umol/L (60-80 ug/dL) for several months. Neurological and gastrointestinal effects are often less marked in chronic poisoning.

Severe encephalopathy is rare under 4.85 umol/L (<100 ug/dL) but has been described in children at ~3.4 umol/L (~70 ug/dL). Mild to moderate anaemia has been found in 5% of adults with levels of 1.95–2.85 umol/L (40–59 ug/dL).

However, frank anaemia does not usually develop until levels exceed 3.85 umol/L (>80 ug/dL) for a prolonged period.

Renal changes are not uncommon. Exposure for years, especially at levels ≥ 3.85 umol/L (≥ 80 ug/dL) increases the risk of chronic insufficiency but rarely progresses to renal failure. Decreased uric acid clearance occurs with the risk of "saturnine" gout. There is some evidence to suggest that levels of 0.95–1.95 umol/L (20–40 ug/dL) may be associated with a rise in systolic blood pressure (0.5-3.0 mmHg).

Decreased sperm counts have been observed at 1.95 umol/L (40 ug/dL) and abnormal morphology and motility at mean levels of~2.55 2.95 umol/L (~53 or 61 ug/dL). Decreased female fertility has been described mainly in the context of high exposure.

Workplace exposure standard
The WES for lead (inorganic dusts and fumes) is 0.1 mg/m^3, an A3 carcinogen notation.

Biomonitoring for lead
A blood lead level of 1.5μmol/litre whole blood is consistent with an average lead in air exposure over a 40-hour week of approximately 0.05 mg/m^3. Biomonitoring for lead is therefore useful. Employers are required to notify all blood lead results 2.6 umol/litre whole blood or above to the Department of Labour.

Mercury

Mercury is the only heavy metal that is a liquid at room temperature, having a melting point of -38° C and a boiling point of 357° C. As a consequence it exerts a considerable vapour pressure at room temperature.

Organic mercury compounds were also important, but because of their toxicity they are now little used. Former uses included seed dressing and as an ingredient in anti-fungal paints, the form most likely to be found at FCC. Because of its high vapour pressure, vapour inhalation is the main route for the entry of mercury into the body, and 80% of inhaled vapour is absorbed. Vapour absorption is taken up by the red cells dissolved in the plasma, where there is rapid transport to all parts of the body. It is bound to sulphydryl groups and can enter all body tissues. Acute mercury poisoning is rare, but has been described following attempts to extract gold from ore using a gold mercury amalgam.

The patients will usually present with an acute febrile illness, with the prominent symptom being respiratory, including cough, dyspnoea, tachypnoea and the feeling of tightness in the chest. A fever, nausea and vomiting is also common. Mild cases may recover, but in more severe cases a picture of acute diffuse interstitial fibrosis may develop.

Chronic poisoning gives renal symptoms. These may be caused by both organic and inorganic compounds, but psychiatric symptoms tend to predominate over the neurological symptoms in inorganic poisoning and the converse is true in organic poisoning. The early symptoms are vague, but they include, headaches, indigestion and the development of a peculiar timidity (erethism).

The most important pathological lesion is atrophy of the cerebellar cortex. The characteristic disturbance produced is a tremor that begins as an intention tremor in the hands but may spread to the face and tongue. This gives a characteristic disturbance to hand writing, which becomes progressively more unintelligible. Allied to this intention tremor are various speech disorders such as hesitancy in beginning sentences and difficulty in pronunciation.

Motor and sensory nerve dysfunction is also part of the neurological syndrome. A spastic gait may be found and also hyperactive tendon reflexes. The sensory disturbances include loss of taste and smell, disturbed proprioception in the fingers and toes, and sensory disturbances. The neural syndromes range between the acute organic psychosis known erethism and subtle neural physiological dysfunction. Erethism was once common in the hat industry, where mercury compound was used in felting. This is of course the origin of the expression "mad as a hatter". Initially it was described as an abnormal state of timidity, and can present as anxiety. Later on the symptoms become more obviously organic, and include headaches, irritability and apathy.

Environmental exposures
There have also been environmental disasters associated with mercury, for example, when mercury wastes were discharged from the Chisso chemical plant in Minimata Bay (Japan), and were concentrated in shell and other fish. As a consequence, several hundred deaths ensued in the following decades due to consumption of fish contaminated by mercury. By the end of 1972, 292 proven cases of illness, 92 of which were fatal, had been observed and there were a total of several thousand injured victims.

Biomonitoring for mercury

The WES for mercury (both organic and inorganic) is 0.025mg/m^3. Biomonitoring is well established, the level of mercury in urine being 0.25 mol/L.

Cadmium

Cadmium occurs in nature principally in association with zinc, but also with lead. It is recovered as a by-product in the extraction of both. Cadmium chloride acts as a fungicide, which may account for its presence at Mapua.

Cadmium enters the body by inhalation and ingestion. Once transported from the site of absorption, cadmium is metabolised in the liver and kidneys. In the liver, cadmium induces the synthesis of metallothionein, and this cadmium-metallothionein complex is released from the liver and transported to the kidneys. This binding is useful in that the cadmium-metallothionein complex is less toxic than cadmium itself.

The principal hazards of cadmium arise from smelting of ores, the welding and melting of cadmium- plated metals and the manufacture of alkaline cadmium (NiCad) batteries. The risk from all these cases is inhalation of either cadmium or cadmium oxide fume or dust. Acute toxicity is usually due to inhalation of cadmium compounds, where the initial effect is usually upon the respiratory system. During the time that cadmium is actually being inhaled, the subject may experience symptoms similar to those of metal fume fever.

Chronic cadmium poisoning has been reported after prolonged occupational exposure to fumes and dust. These changes may be local and associated with the respiratory tract, but are also associated with damage to the renal system. The lung disease is in the form of emphysema, which is focal in nature. This is probably due to interference with α-1 antitrypsin in the plasma. The kidney may also be a critical organ, as cadmium accumulates in the renal cortex. This causes tubular dysfunction, manifested by the excretion of low molecular weight proteins in the urine, of which the major constituents are beta 2 microglobulins. These are sometimes used as a means of biological monitoring. In spite of these changes, renal failure is seldom a feature, although osteoporosis has been reported in cases of severe chronic poisoning.

There is also evidence that cadmium may cause increased mortality from lung cancer, although the interpretation is somewhat complicated by the confounding exposure to other metals, and the findings of some studies have been equivocal. While it was once thought that exposure to cadmium held an increased risk of prostatic cancer, continuing observations of cadmium-exposed workers have failed to support this hypothesis. Control of cadmium exposure should be focused on keeping concentration levels to a minimum. Where practicable, processes should be enclosed and fitted with exhaust ventilation. When adequate ventilation is not possible, for example during welding and cutting operations, respirators should be carried and the air should be sampled to determine the cadmium levels.

Table 8.   NZ WES for cadmium

Substance	CAS	TWA Ppm	TWA mg/m³	STEL ppm	STEL m³
Cadmium and compounds, as Cd (A2, bio, 1994)	[7440-43-9]	-	0.01 Inspirable dust 0.002 Respirable dust	-	-

Biological monitoring is advisable, and the WES book gives both blood and urine BEIs as follows: Blood-0.09 □□mol/ litre (10 □□g/ litre) Urine-10 □□mol/ mol creatinine (10 □g/g creatinine). Note that there is a proposed change to 5 □g/g creatinine.

Arsenic

Arsenic is a metalloid element. It is found principally in the ores of copper, lead and zinc from which arsenic is recovered as an impurity during smelting.

The earliest organic arsenical compounds were used as herbicides and defoliants. Arsenic compounds, particularly the organic compounds, are regarded as very potent poisons. Over 95% of arsenic in the blood is bound to the protein in haemoglobin, and excretion takes place predominantly through the kidney. It is also stored in the tissues and tends to accumulate in the muscles and liver and, peculiarly, in the hair and nails. Poisoning in industry is rare, and most arsenic poisonings result from the ingestion of contaminated food and drink. The clinical picture resembles that of cholera and may include difficulty in swallowing, crampy abdominal pain, projectile vomiting, rice water diarrhoea, dehydration, a weak irregular pulse and loss of blood pressure. These are followed by coma, convulsions and death. The fundamental effect appears to be dilation and increased permeability of the small blood vessels in the gut wall and elsewhere.

Long-term exposure to inorganic arsenic has been found to give rise to effects in a large number of organs. However, the details of human exposure (e.g., type of arsenic compound), have been inadequate for the establishment of dose-response relationships. Lesions of the upper respiratory tract including perforation of the nasal septum, laryngitis, pharyngitis, and bronchitis have frequently been encountered in workers in the smelting industry exposed to high levels of arsenic.

Arsenic in the trivalent state can give rise to skin lesions in humans, especially palmo-plantar hyperkeratosis, which has a characteristic appearance. It has been observed in patients under prolonged medication with Fowler's solution receiving daily doses of arsenic of up to 10mg. Palmo-plantar hyperkeratosis has also been reported following ingestion of arsenic in drinking water (oxidation state not determined) in some parts of the world, including Argentina, Taiwan and Mexico. Other dermatological symptoms, including hyperpigmentation, have also appeared in inhabitants of these areas. It should be noted that hyperkeratotic lesions of the palms and soles and hyperpigmentation are very rare among smelter workers exposed to inorganic arsenic, but have been reported in other occupational situations. The reason for this discrepancy is not clear but could be the result of differences in dose.

Inorganic arsenic can exert chronic effects on the peripheral nervous system in humans. The only information on these effects as far as occupational exposure is concerned comes from case reports, and exposure levels have not been given. It is obviously difficult to draw any conclusions from such reports. Disturbances of CNS function were reported in Japanese youths 15 years after they had been exposed as infants to inorganic arsenic in average daily doses of 3.5mg for about one month. The effects included severe hearing loss and electroencephalographic abnormalities.

Because inorganic trivalent arsenic has an effect on the haematopoietic system, it has been used for several decades as a therapeutic agent for various forms of leukaemia, often in doses of several milligrams daily.

There are both in-vivo and in-vitro studies indicating effects of inorganic arsenic on human chromosomes. An increased frequency of chromosomal aberrations has been found among persons exposed to arsenic, mainly in the trivalent form, through medication. Similar findings have been reported among workers exposed to arsenic. However, the exposure of these workers to other toxic substances may have been of importance. Several studies have indicated that inorganic arsenic affects DNA repair mechanisms.

There is substantial epidemiological evidence of respiratory carcinogenicity in association with exposure to mainly inorganic arsenic in the manufacture of arsenic-containing insecticides. However, conclusions cannot be drawn on the carcinogenic potential of trivalent versus pentavalent inorganic compounds since exposure to both forms occurred in these workplaces. A possible association between the use of pesticides containing arsenic – often in the form of arsenate in vineyards and orchards – and in an increased risk of lung cancer has been found, but the data are not conclusive.

The carcinogenic potential of inorganic arsenic in smelter environments is evident from many epidemiological studies. One report revealed a roughly linear relationship between cumulative arsenic exposure and lung cancer risk. Although exposure data are uncertain, it is estimated that exposure to airborne arsenic levels of about 50,ug/m3 (probably mostly arsenic (III) oxide) for more than 25 years could result in a nearly three-fold increase in the mortality rate of cancer in the respiratory tract after the age of 65 years.

Exposure to inorganic arsenic can cause skin cancer, mainly tumours of low malignancy. This has been observed following ingestion of arsenic in drinking water or drugs resulting in a total intake of several grams of arsenic over a number of decades. The form of arsenic in drinking water has yet to be elucidated, but in medication it has most often been inorganic trivalent arsenic.

Biomonitoring
The WES for Arsenic & soluble compounds, as As, is 0.05 mg/m^3, the BEI for arsenic being 100 µg/L.

Chromium

Elemental chromium (Cr) is not found free in nature, and the only ore of any importance is the spinel ore, chromite or chrome iron stone, which is ferrous chromite ($FeOCr_2O_3$).

The ILO encyclopaedia explains the chemistry of chromium compounds, which is complex.

> *"Chromium forms a number of compounds in various oxidation states. Those of II (chromous), III (chromic) and VI (chromate) states are most important; the II state is basic, the III state is amphoteric and the VI state is acidic. Commercial applications mainly concern compounds in the VI state, with some interest in III state chromium compounds.*

> *The chromous state (CrII) is unstable and is readily oxidised to the chromic state (CrIII). This instability limits the use of chromous compounds. The chromic compounds are very stable and form many compounds which have commercial use, the principal of which are chromic oxide and basic chromium sulphate.*

> *Chromium in the +6 oxidation state (CrVI) has its greatest industrial application as a consequence of its acidic and oxidant properties, as well as its ability to form strongly coloured and insoluble salts. The most important compounds containing chromium in the CrVI state are sodium dichromate, potassium dichromate and chromium trioxide. Most other chromate compounds are produced industrially using dichromate as the source of CrVI."*

There are also many other widespread uses of hexavalent chromium compounds, including the dyeing of textiles, and in printing inks. A common use in New Zealand is in copper chrome arsenic as a wood preservative. FCC may have produced potassium dichromate for this purpose.

The toxicology of chromium is complex. Trivalent chromium is significantly less hazardous than hexavalent chromium and is also less well absorbed. In contrast, hexavalent chromium is readily absorbed after ingestion as well as during inhalation. Inhalation is in fact the route of exposure most responsible for the associated risks from chromium exposure in humans. After absorption in the lung, pulmonary macrophages can reduce hexavalent to trivalent chromium, thus reducing the toxic impact. However, hexavalent chromium is carried in other red and white blood cells, so the distribution of chromium cannot be accurately predicted. It is cleared from the blood by the kidney and excreted principally in the urine.

The effects of chrome are primarily due to the hexavalent chromium species. Apart from being directly toxic to cells through an impact on cellular respiration and the production of ATP, hexavalent chromium also reacts with nuclear acids

and DNA. It has chromosomal effects, causing chromosome damage. All the hexavalent chromium compounds so far assessed have been found to induce chromosomal mutations.

The most commonly recorded adverse consequence of chromium exposure is that of chrome ulceration. This most frequently occurs during electroplating, when chromic acid is formed. During this process, the object to be plated is placed in a bath containing chromic acid and a current is passed through the solution. This results in evolution of hydrogen at the cathode, which produces bubbles at the surface of the bath and a chromic acid mist.

The direct effects are on the skin and the respiratory system. "Chrome holes" develop on abrasions on the skin and are most commonly found at the root of the fingernail, the knuckle or the dorsum of the foot. They are circular in shape, clear cut, usually 1 cm or less in diameter and they look as if they have been punched out, hence the name. They have a strong tendency to heal but may penetrate very deeply, even to the bone. Although painless they are said to itch intolerably at night. There does not seem to be a tendency towards malignant change. Perforation of the nasal septum is also found, but usually this causes no inconvenience and is discovered on routine medical examinations.

There is also increased incidence of gingivitis and periodontitis. Possibly because of the genotoxic effects, there is also an excess of lung cancer in workers producing and using chromium compounds. This exposure mostly concerns the chromate-producing industry, where there is extensive airborne exposure to chromium VI dust. Because of this carcinogenicity there is now a great deal of interest in less intense chrome exposure, including that found in stainless steel welding and metal spraying processes. There also have been concerns about chromium exposure and occupational asthma.

Enclosure and extract ventilation is the control method of choice in the chromate-producing industries and also in chromium plating, where minimisation is also practiced by breaking up the surface tension of the chromic acid bath by the addition of "cruffles". In welding and metal spraying operations, local extract ventilation is essential, as is the use of respiratory protective equipment.

Biomonitoring

Table 9.   WES for chromium

Substance	CAS	TWA ppm	mg/m³	STEL ppm	m³
Chromium (VI) compounds, as Cr. Insoluble Cr (VI) compounds (sen, A1)	[7440-47-3]	–	0.01	-	-

OSH has published a biological exposure index for chromium VI soluble salts in the WES book, and this is BEI: 0.6 μ □mol/ litre (30 μ □g/ litre)

Sulphur

Inorganic sulphur was used in many types of formulation as a fungicide and insecticide on pipfruit and other fruits. It is considered to be of low toxicity. There is no WES.

Organophosphorus compounds

These compounds were first studied just prior to and during World War II, but did not gain widespread usage as insecticides until the late 1950s, when they began replacing organochlorine compounds. The term "organophosphates", or OPs, is sometimes used to denote those organophosporous compounds which have a significant anticholinesterase effect. Virtually all of the acute toxic effects of OPs are due to this action.

There is considerable diversity in the chemical structure of OPs, although of course there are some features common to all. As a consequence there is also a considerable variation in physico-chemical properties and toxicity.

A good example is the large difference in the estimated oral LD_{50} values between one of the most toxic and least toxic OPs, i.e. parathion (about 3 mg/kg) and malathion (about 1300 mg/kg).

Figure 16. Formula for malathion
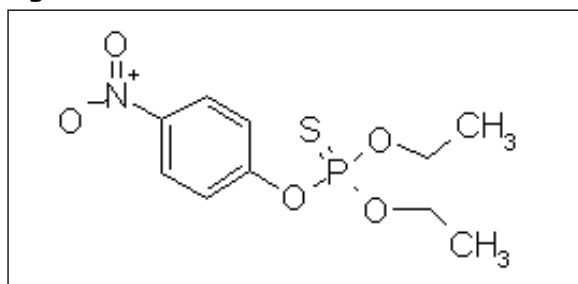
All compounds share a general structure characterised by the presence of a phosphorus atom linked with a double bond to a sulphur or oxygen atom. Two alkyl groups linked to the phosphorous atom with an oxygen bridge are either methyl, ethyl or isopropyl. The remaining bond of the pentavalent phosphorus is linked to a so called "leaving group" that varies greatly between chemicals.

It can be helpful for understanding to view the anticholinesterase effects of OPs as a "double negative". Thus they inhibit the enzyme (acetylcholinesterase) responsible for inhibiting or destroying the neurotransmitter acetylcholine, with the result that increased levels of acetylcholine occur at the nerve endings.

In the normal situation, acetylcholine, as a response to a nerve impulse, is released from the nerve terminal and so enables transmission onwards of that impulse, either to another nerve – as in the central nervous system – or to an 'effector' organ such as a muscle or a gland. It is normally then inactivated rapidly by the enzyme acetylcholinesterase, with consequent limitation of neurotransmission to a short sharp stimulus.

As OPs inhibit this inactivating enzyme, acetylcholine persists at the nerve terminal and gives continuing nerve stimulation. This causes an increased response although, in extreme cases, the response can be decreased by fatigue.

Acetylcholine is the major neurotransmitter in the parasympathetic nervous system (as well as part of the sympathetic nervous system, i.e. the ganglia) at the neuromuscular junctions of skeletal muscle, and is one of several neurotransmitters in the central nervous system. Thus by prolonging its action at all of these sites, OPs have the potential to cause a wide range of effects, which have been classified as muscarinic, nicotinic or central nervous system effects.

Muscarinic: Is the term given to effects resulting from para-sympathetic nervous system stimulation. The parasympathetic and sympathetic nervous systems are the two components of the autonomic nervous system, which largely regulates the continuous non-voluntary function of organs and exocrine glands. The dual effects of the two components are necessary to keep the system in balance.

Excess parasympathetic nervous system activity increases stimulation of non-voluntary smooth muscles in the alimentary, respiratory and urinary tracts, and increases the activity of various glands while decreasing heart stimulation.

Thus OPs may cause nausea, vomiting, diarrhoea, increased salivation, sweating, increased bronchial secretion, bronchospasm, constricted pupils (miosis), and bradycardia (slow heart rate).

Nicotinic: Largely refers to the results of excessive neuromuscular stimulation, such as skeletal muscle tremor and weakness. When this involves the intercostal and other "respiratory" muscles, impaired ventilatory function can become a critical factor in OP poisoning. (It also includes effects from some sympathetic nervous system stimulation, such as tachycardia.)

Central nervous system
Symptoms may vary from anxiety, fatigue, headache, drowsiness, and confusion to, in some extreme cases, convulsions, coma, and respiratory depression and ultimately arrest.

Workplace exposure standard
The WESs for these compounds varies, for example:
- chlorpyrifos 0.2 mg/m^3
- diazinon 0.1 mg/m^3
- dichlorvos 0.1 mg/m^3
- malathion 10 mg/m^3
- parathion 0.1 mg/m^3

All have Sk notation.

Monitoring
The effect of OPs on the body (due to inhibition of cholinesterase, which is only slowly reversible) outlasts the presence of most of the absorbed dose in the body, as most OPs are eliminated in one to three days. Thus the kinetic fate of the

individual OP is less relevant than the fate of the enzyme it inhibits. It has been stated that inhibited enzyme regenerates at about 1% per day. Thus uninhibited enzyme levels fallen to 50% of normal will take about two months to reach their usual levels. (In reality, while the enzyme inhibition by OPs is usually long-lasting, it varies somewhat with different OPs, and for some it is not completely irreversible.)

The above estimation relates to a worst-case scenario, where no reactivation of enzyme occurs, and recovery of blood levels depends almost entirely on production of new red blood cells, (which turn over at about 0.8% each day) with their quota of 'fresh' acetylcholinesterase). Both plasma or whole blood cholinesterase levels give a reasonably good guide to acetylcholinesterase levels in nervous tissue, which is the critical factor, although plasma cholinesterase levels are not completely specific indicators as they can be affected by chronic liver disease, malnutrition and other rare conditions.

The concern with occupational use is that exposure may be quite low, and the absorbed dose from a single episode may be insufficient to cause symptoms. Usually a 40–50% drop, or even more, in cholinesterase level is necessary before the development of definite symptoms. However, slight but repetitive exposures can cause gradual and progressive declines in uninhibited or "active" enzyme levels, due to its slow recovery rate between exposures. Thus the worker may remain asymptomatic in the face of declining cholinesterase levels until a final exposure is enough to "tip the balance" to a level which does produce symptoms. Hence the need for some system of monitoring acetylcholinesterase levels. New Zealand (DoL) Guidelines{Occupational Safety and Health Service Department of Labour, 2000 #28} on this issue are as follows:

Organophosphate monitoring

When monitoring a worker's exposure to organophosphate, both red cell and plasma cholinesterase levels should be determined.

Plasma enzyme (pChE) activity is generally a more sensitive test of exposure because it is more rapidly inactivated by most, though not all OPs. However, it is less specific in reflecting levels of enzyme depression in the nervous tissue, as it is a different compound from acetylcholinesterase. In some cases it may overestimate nerve tissue levels as it often recovers from inhibition and also regenerates more quickly than AChE. It is also a less specific test, in that there are a wider range of other conditions or personal characteristics that can affect the measurements. It also represents only about 8% of the total cholinesterase activity of whole blood, the majority (about 92%) being due to RBC AChE.

The RBC (erythrocyte) AChE test is more specific and reliable, and various authorities have recommended that this test should form the basis for decisions on management. It is the preferred test of the two. However, it can be a somewhat conservative indicator of nerve tissue levels, particularly in the later stages of recovery.

Ideally, both RBC and pChE should be done because interpretation can be aided by having the results of both tests available.

NOTE: It is recognised that in some cases complete recovery of RBC AChE may lag behind enzyme recovery in nervous tissue, because full return to normal RBC AChE depends on its re-synthesis, which in turn depends on the re-synthesis or turnover rate of RBCs themselves. This is limited to about 0.8% per day. Thus, recovery of RBC AChE, unlike that in nerve tissue, is artificially constrained. However, it is better to sometimes underestimate recovery rates of AChE in the nervous system with the use of RBC or whole blood AChE tests than to use pChE as the criterion, which can sometimes overestimate such recovery rates and lead to a false sense of security. The Ellman method is recommended for the tests. The tests may be performed on separated red blood cells and plasma, or on whole blood using a procedure that is specific for AChE and pChE activity. (Biological Monitoring of Chemical Exposure in the Workplace, WHO, Geneva, 1996.) The same laboratory using the same method should do cholinesterase tests for any individual. This is because there may be considerable variation in results even among different laboratories using the same method.

When to monitor

Monitoring takes two forms

Initial testing

Note: this section summarises best practice for other "baseline" or before exposure testing.

An initial test of the individual's "normal" cholinesterase activity levels takes place before they are exposed to OPs, and periodic testing is carried out thereafter. The test of the user's baseline level is very important, because of the large range of normal values observed between individuals. Thus, if a test is only done once OP exposures have recently commenced, it can be difficult to know, in the event of a low result, to what extent this

signifies OP-induced enzyme depression, on the one hand, versus a naturally low individual level on the other.

1. Baseline testing

These tests should be done only after at least 30 days freedom from exposure to OPs. They are best done, however, prior to employment or before first use of OPs. Such stringent timeframes are not required for carbamate insecticides, where inhibition of AChE is more short-lived. At least one pre-exposure test should be done (ideally two). Many authorities recommend averaging two such tests (as a minimum) for optimum baseline

estimation.

2. Periodic testing

Testing should be related to intensity an4d frequency of exposure, with the following recommendations:
  a) *Periodic testing should be carried near the probable peak of the application/exposure period.*
  b) *Retesting should be more frequent in cases of:*
  • *an inexperienced user, or if there is evidence for occurrence of "mishaps"*
  • *substandard protective equipment or work practices*
  • *a new formulation, where the method of absorption has not been thoroughly assessed*
  • *where the extent and frequency of use is increased.*

Testing of new workers may need to be on a weekly basis for the first two or three tests (provided they have been regularly exposed during this time), then monthly for two or three months. If no significant decrease is found, tests could thereafter be reduced to once per season, near to the probable peak of the application period, as recommended in 2a).

Interpretation of results and management of users

Monitoring using blood tests can give only an approximate idea of nervous tissue levels and hence risks. Furthermore, the risk depends on the rate of enzyme inhibition as well as its absolute level at a point in time. (Thus, in someone regularly exposed, a gradual 70%–80% depression to 20–30% of normal levels may not always be associated with symptoms, while in previously unexposed workers, a rapid 30% drop to 70% of normal may.)

AchE % of baseline	AChE fall from baseline	Significance percentage of baseline	Management baseline
20% to 39%	61% to 80%	Evidence of significant exposure	(i) Retest (ii) Check work practices
40% or greater	60% or less	Increased vulnerability from subsequent exposures	(i) Remove from work (ii) Notify to OSH

NOTE: The above criteria refer to AChE. The fall from baseline and the percentage of baseline are complementary. For example, a person with a baseline of 100 may subsequently be tested after exposure to OPs and have an AChE level of 75. This is a 25% fall from baseline, and it is also 75% of the original baseline.

Two fundamental points to remember are:

1. Decisions are best based on AChE levels.

2. Decisions are much easier if baseline values are available.

3. Criteria for return to work

Workers should be suspended from work when their fall from baseline is 40% or greater. They should then return to work only when the fall from baseline has partially recovered and is only 25% or less, i.e. is 75% or more of the baseline.

NOTE: The above criteria relate to RBC active AChE levels, rather than whole blood levels. The relationship between the two results depends on the type of test method used, in particular whether it measures AChE specifically. However, in any case there is a close correlation and little difference between the two test results. Therefore, the above criteria can be used for whole blood results as well.

4. Testing for diagnostic purposes

Sequential post-exposure testing in the absence of a baseline can also be used to help confirm or question the diagnosis of OP-induced illness. Recovery of enzyme levels is more rapid during the first few days of post-exposure (for some OPs at least) than subsequent rates of recovery towards the baseline. This is because for many OPs there is some degree

of spontaneous reversal of enzyme inhibition (termed "reactivation") which occurs more rapidly than the slower progress of regeneration or re-synthesis of new active enzyme to replace the permanently inhibited fraction, (i.e. recovery = reactivation + regeneration.)

Other issues with OPs

Chronic or delayed neurotoxicity

A different, largely neurological syndrome has been described with (as yet) a relatively small number of OP compounds. This syndrome may develop after two to four weeks, but almost without exception only in people very heavily exposed who had developed severe and typical 'cholinergic' symptoms initially. The toxic mechanism is quite different and is related to inhibition of another enzyme, neurotoxic esterase (NTE).

The OP combines with the enzyme NTE ("phosphorylates"). This complex then undergoes a change called "ageing", involving loss of part of the OP molecule, but leaving a negatively charged phosphoryl group still attached. The remaining combination with the enzyme somehow triggers a chain of processes leading ultimately to axon (or nerve) damage. This phenomenon is understandably of concern because it is unpredictable, with no specific treatment. It is very uncommon, however, certainly with the level of occupational exposures occurring in New Zealand.

Motor function is more affected than sensory – there is a distal symmetric polyneuropathy with weakness, wasting, and hypotonia of the limb muscles. Electrophysiological testing reveals partial denervation of affected muscles. OPs incriminated include mipafox, leptophos, trichlorphon, trichlornate, methamidophos, TOCP and chloropyrifos. Being distal, the weakness may be most marked in the muscles of the hands and feet.

To complicate matters, an "intermediate syndrome" has been described. However, this again is largely a complication of large life-threatening doses, usually from ingestion, although it has very occasionally arisen after heavy skin contamination.

Behavioural effects

This issue has been reviewed and there is some consensus that after a poisoning episode sufficient to cause cholinergic symptoms and therefore likely to involve depression of blood levels by at least 50–60%, there may be impaired concentration, reduced reaction times, impaired intelligence and memory, together with depression and/or anxiety.

Environmental aspects

OPs are not very persistent in the environment, their half lives being just a few days in water at neutral pHs, but longer, up to a few weeks, in acidic soils. They are degraded by hydrolysis, yielding water-soluble non-toxic products. They are, however, best inactivated by alkalis and this is recommended in a situation of environmental contamination.

For the above reasons the "withholding period" – i.e. the minimum recommended time between last spraying and consumption of OP-sprayed crops – is relatively short, three days to three weeks, but usually nearer the shorter end of the range. Because of skin absorption, pickers or harvesters of sprayed crops may also be at risk, and a period of at least three days is recommended before harvesting treated produce.

Summary of toxicology

DDT and analogues

A large number of pesticide residues were found at the Mapua site. The most extensive contamination occurred with OCPs, including DDT. There is coherent evidence that high environmental levels may be associated with hormonal effects, including effects on pregnancy and fertility. There is no evidence of clinically apparent haematological effects such as haemolysis, but some evidence that liver microsomal enzymes are induced. The acute effects are neurological, with disturbances of sensation, dizziness, headache and other non-specific effects such as headache. More subtle neuropsychological changes have been noted.

There are no BEIs for OCPs, but liver microsomal enzymes are not affected following repeated exposures to DDT, at DDT and DDE serum concentrations below 250 µg/l.

Aldrin, Dieldrin and Lindane

This group has occasionally caused poisoning during manufacture or use. The effects are headaches, dizziness, blurred vision, nausea, insomnia and personality changes. Tremors and convulsions can occur. The symptoms of Dieldrin intoxication are similar.

The WESs have been described in the relevant section, and a biological limit value of 100 µg/L has been proposed for Dieldrin. Most of the workers in Lindane manufacture had levels between 16 and 57 µg/L and had symptoms of peripheral neuropathy.

Chlorophenoxyacetic acid herbicides, phenoxy herbicides

The phenoxyacetate herbicides have low toxicity, with nausea and vomiting the primary symptoms. Most of the effects have been attributed to contamination of 2-4-5T with 2,3,7,8,TCDD.

Dioxins and related compounds

A summary of the possible associations between pesticides (with an emphasis on Agent Orange) is carried out periodically by the Institute of Medicine (IOM) of the National Academy of Sciences of America. The 2002 associations and their strengths are as in the table below.

Table 10. Strength of association of diseases and herbicide exposure

Hierarchy by Strength of Association	Disease
Sufficient evidence	Chronic lymphocytic leukemia (CLL)
	Soft tissue sarcoma
	Non-Hodgkin's lymphoma
	Hodgkin's disease
	Chloracne
Limited/Suggestive evidence	Respiratory cancers (lung, larynx, trachea)
	Prostate cancer
	Multiple myeloma
	Acute and subacute transient peripheral neuropathy
	Porphyria cutanea tarda
	Type 2 diabetes
	Spina bifida (in offspring)
Inadequate/Insufficient evidence	Hepatobiliary cancers
	Nasal/nasopharyngeal cancer
	Bone cancer
	Breast cancer
	Cancers of the female reproductive tract
	Renal cancer
	Bladder cancer
	Testicular cancer
	Leukaemia including acute myeloid leukaemia (in offspring)
	Skin cancers
	Spontaneous abortion
	Birth defects (other than spina bifida)
	Neonatal/infant death and stillbirths
	Low birthweight
	Childhood cancer in offspring including AML
	Abnormal sperm parameters and infertility
	Cognitive and neuropsychiatric disorders
	Motor/coordination dysfunction
	Chronic peripheral nervous system disorders
	Gastrointestinal, metabolic and digestive disorders
	Immune system disorders
	Circulatory disorders
	Respiratory disorders
	AL-type primary amyloidosis
Limited/suggestive evidence of NO association	Cancer of the gastrointestinal tract (colon, rectal, stomach and pancreatic tumours)
	Brain tumours

Triazines and other related nitrogen-containing pesticides

Atrazine is of "slight" toxicity. Long-term consumption of high levels of atrazine has caused adverse health effects in animals, including tremors, changes in organ weights, and damage to the liver and heart.

Lead

In severe poisoning (which hopefully is only rarely found these days) there is a global and profound disturbance of the central nervous system. The symptoms include fits and may result in coma. The chronic and subchronic effects include aches and pains, deficits in memory, sleep disturbance and changes in personality with increased irritability and anxiety. Lead causes anaemia, which is hypochromic, and may be micro or normocytic. It does not cause platelet effects.

The NZ WES for lead is 0.1 mg/m^3, and a blood lead level of 1.5☐mol/litre whole blood is consistent with an average lead in air exposure over a 40-hour week of approximately 0.05 mg/m^3.

Mercury

The patients will usually present with an acute febrile illness, with the prominent symptom being respiratory, including cough, dyspnoea, tachypnoea and the feeling of tightness in the chest. A fever, nausea and vomiting is also common. Mild cases may recover, but in more severe cases a picture of acute diffuse interstitial fibrosis may develop. Chronic poisoning gives renal symptoms. These may be caused by both organic and inorganic compounds, but psychiatric symptoms tend to predominate over the neurological symptoms in inorganic poisoning and the converse is true in organic poisoning. The early symptoms are vague, but they include, headaches, indigestion and the development of a peculiar timidity (erethism).

The WES for mercury (both organic and inorganic) is $0.025mg/m^3$ and biomonitoring is well established, the level of mercury in urine being 0.25 mol/L.

Cadmium

Chronic cadmium poisoning has been reported after prolonged occupational exposure to fumes and dust. These changes may be local and associated with the respiratory tract, but are also associated with damage to the renal system. The lung disease is in the form of emphysema, which is focal in nature.

The WES is 0.01 mg/m3 inspirable dust and 0.002 mg/m3 respirable dust.

Arsenic

Acute arsenic poisoning in industry is rare, and most arsenic poisoning falls in the ingestion of contaminated food and drink. The clinical picture resembles that of cholera and may include difficulty in swallowing, crampy abdominal pain, projectile vomiting, rice water diarrhoea, dehydration, a weak irregular pulse and loss of blood pressure. These are followed by coma, convulsions and death. Long-term exposure to inorganic arsenic has been found to give rise to effects in a large number of organs. However, the details of human exposure (e.g. type of arsenic compound), have been inadequate for the establishment of dose-response relationships. Lesions of the upper respiratory tract, including perforation of the nasal septum, laryngitis, pharyngitis, and bronchitis, have frequently been encountered in workers in the smelting industry exposed to high levels of arsenic. Arsenic in the trivalent state can give rise to skin lesions, especially palmo-plantar hyperkeratosis, which has a characteristic appearance.

Inorganic arsenic can exert chronic effects on the peripheral nervous system in humans. The only information on these effects as far as occupational exposure is concerned comes from case reports, and exposure levels have not been given. It is obviously difficult to draw any conclusions from such reports. Disturbances of CNS function were reported in Japanese youths 15 years after they had been exposed as infants to inorganic arsenic in average daily doses of 3.5 mg for about one month. The effects included severe hearing loss and electroencephalographic abnormalities.

Because inorganic trivalent arsenic has an effect on the haematopoietic system, it has been used for several decades as a therapeutic agent for various forms of leukaemia, often in doses of several milligrams daily.

Arsenic also causes skin and lung cancer.

The WES for arsenic and soluble compounds, as As, is 0.05 mg/m3, the BEI for arsenic being 100 µg/L.

Chromium

The direct effects of chronic chromium exposure are on the skin and the respiratory system, with skin and nasal ulceration.

The WES for chromium is 0.01 mg/m^3, with a BEI of 0.6 ☐☐mol/ litre (30 ☐☐g/ litre)

Sulphur

Inorganic sulphur is of low toxicity. There is no WES.

Organophosphorus compounds

The muscarinic effects may cause nausea, vomiting, diarrhoea, increased salivation, sweating, increased bronchial secretion, bronchospasm, constricted pupils (miosis) and bradycardia (slow heart rate).

The nicotinic effects are to cause excessive neuromuscular stimulation
The CNS effects are anxiety, fatigue, headache, drowsiness, and confusion. In some extreme cases the effects include convulsions, coma, and respiratory depression and ultimately arrest.

The WESs have been described above: apart from malathion they are in the order of 0.1-0.2 mg/m^3. A fall from baseline AChE of 60% or more is of concern in biological effect monitoring.

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