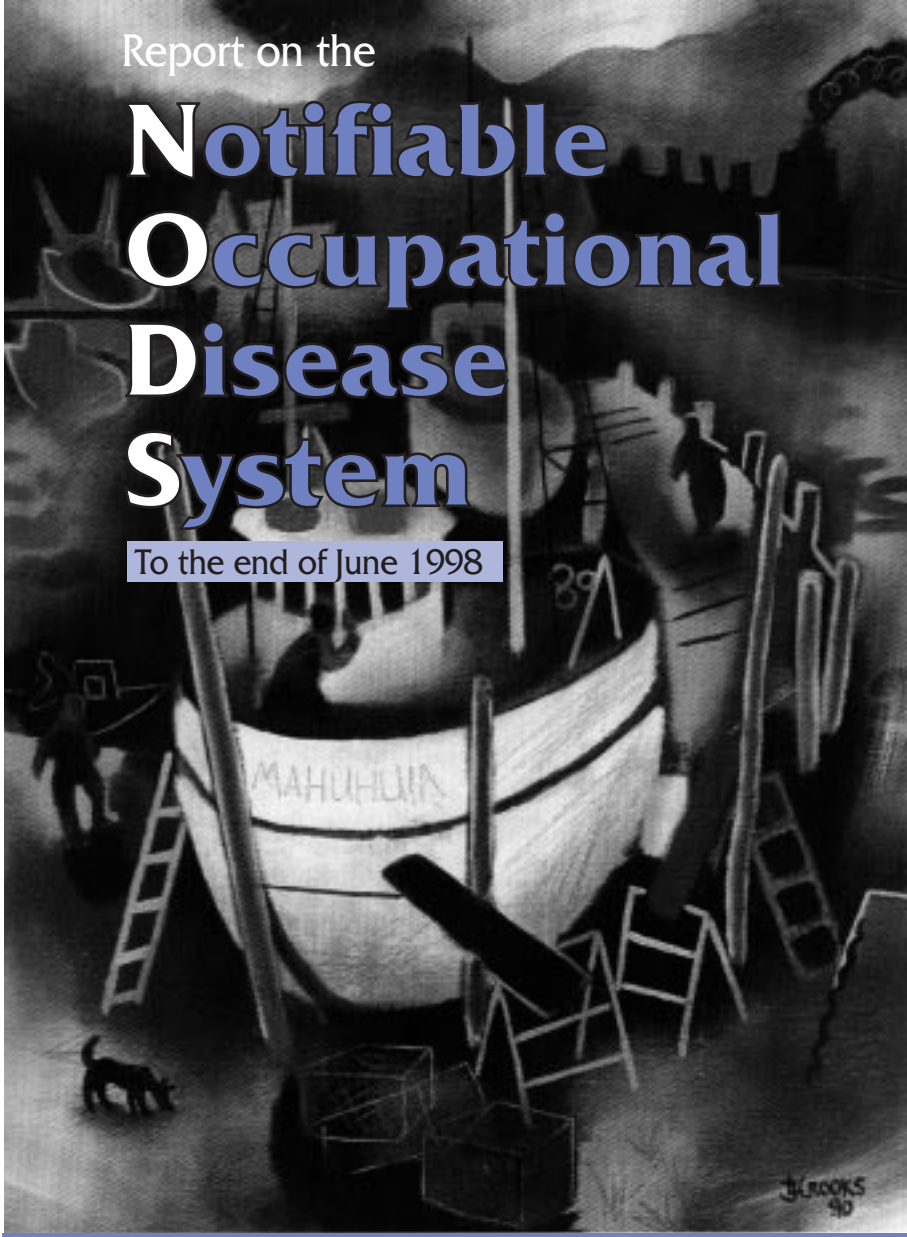
Report on the

Notifiable Occupational Disease System

To the end of June 1998



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OCCUPATIONAL SAFETY & HEALTH SERVICE

DEPARTMENT OF LABOUR
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OSH 3792

Contents

What is NODS?

NODS is an acronym for the **Notifiable Occupational Disease System,** which was established in March 1992. It is a voluntary system, whereby occupational physicians, specialists, general practitioners, occupational health nurses, other health professionals, and individuals can notify a health-related condition which is suspected to arise from work.

Who administers NODS?

NODS is administered by the Occupational Safety and Health Service of the Department of Labour (known as OSH). OSH is responsible for the operation and enforcement of the Health and Safety in Employment Act 1992. This is the principal legislation for ensuring workplaces are safe and healthy. It requires employers to:

- Provide and maintain a safe work environment;
- Provide and maintain facilities for the safety and health of employees at work;
- Ensure that machinery and equipment in the place of work is designed, made, set up, and maintained to be safe for employees;
- Ensure that employees are not exposed to hazards in the course of their work; and
- Develop procedures for dealing with emergencies that may arise while employees are at work.

The Health and Safety in Employment Act requires employers to notify OSH about workers who suffer serious harm as a result of their work. These notifications supplement voluntary notifications made to NODS.

The Act also requires employees to accept responsibility for their health and safety while at work and make sure that their actions do not harm other workers.

Why have a disease notification system?

The Notifiable Occupational Disease System has several objectives:

- It enables OSH to become aware of work-related health problems. In this way, the sick worker acts as an indicator of a sick workplace.
- At the national level, it assists OSH to plan and implement health promotional and intervention strategies.
- Also at the national level, it allows OSH to monitor trends in occupational disease patterns. In time, this will permit an evaluation of the effectiveness of promotional and intervention strategies.
- It raises awareness among health professionals about an individual's occupation being an important determinant of disease.
- It assists workers and managers to recognise more clearly harmful or dangerous work situations or work practices.
- It provides a database for the development of applied research.

How does NODS work?

The NODS process involves the following four stages:

Notification of a possible work-related condition.

Assessment and/or investigation of the individual worker, their work and their workplace by the health and safety team of the local OSH branch.

Verification of the notification by departmental medical practitioners, who can request further assistance from specialist medical panels.

Entry of the confirmed cases on the NODS database.

Notification

A notification comes either to the NODS registrar, or direct to the OSH branch on a standard notification form. If it is received by the NODS registrar, it is forwarded to the branch. When the NODS notification form reaches the branch, an assessment of the case is made, and if appropriate an investigation is initiated.

OSH recognises that general practitioners are increasingly inundated with paper work. In order to minimise the workload, the notification card has been designed so that the practice nurse can enter the individual's personal information: the doctor simply records the diagnosis.

Investigation

A team approach to the investigation of occupational disease, and the consequent intervention in the workplace, is now practical because OSH is the primary authority responsible for health and safety at work. Investigating teams may include: a departmental medical practitioner, an occupational health nurse, an industrial hygienist, an accident prevention consultant, and health and safety inspectors.

The first stage in an investigation is usually carried out by an occupational health nurse, who could be assisted by the industrial hygienist or one of the inspectors. This first stage may involve the individual notifier filling out a standard questionnaire form with the help of the occupational health nurse, monitoring of the workplace, and visiting the workplace. Once the investigatory process has been completed (and on occasions this may require other medical specialist input), the details are referred to the departmental medical practitioner.

Verification

The departmental medical practitioner verifies the notification, and this is entered on a database. If, on completion of the investigation, the departmental medical practitioner still has some concerns about diagnosis or causation, specialist panels have been established to which the case can be referred. These are listed in Appendix 1.

Verification is carried out to ensure that, as far as possible, only cases with well-established causation are included in the database. This means that the database will contain good-quality material for research and statistical purposes. Where a case is not accepted for the register, it will usually mean that another cause has been established.

There will always be borderline cases where there is some doubt. Such cases, even if not accepted for the database, will still have the benefit of workplace inspections and workplace improvements where necessary.

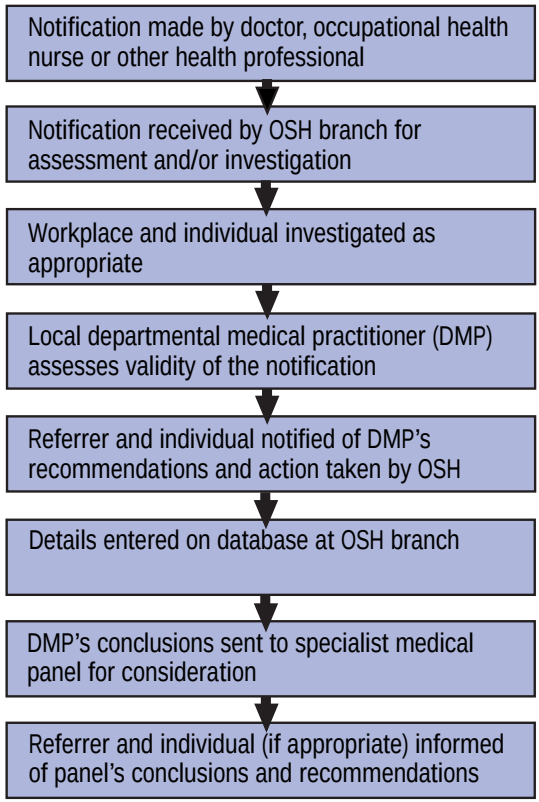
A decision not to accept a case for the register does not imply rejection of an individual's claim for accident compensation, which has different criteria for acceptance.

Information recorded on the register regarding an individual's case is stored under strict confidentiality. Personal information is not supplied to ACC or any other organisation without the individual's signed consent to do so.

Intervention

It is important to emphasise that notification is an indicator of a workplace hazard. A "sick" worker alerts OSH to the existence of a "sick" workplace. Intervention with effect must be the ultimate end point of the process of notification in order to prevent other workers from suffering

the same consequences resulting from these work circumstances. The complete NODS procedure is illustrated below:



Notifiable conditions

The most common diseases arising from work are listed in Table 1, together with the number of notifications received since the inception of NODS in March 1992. The 1992-93 year includes notifications made March 1992 through to 30 June 1993. Subsequent years cover 1 July to 30 June.

Table 2 (over page) classifies confirmed cases according to ISCO88 two-digit classification and notification type.

Table 1: Notifications received March 1992 to June 1998

Disease category	1992/93	1993/94	1994/95	1995/96	1996/97	1997/98	Total
Asbestos-related disease	383	103	132	67	71	58	814
Occupational asthma	39	60	97	81	67	61	405
Other occupational respiratory disease	7	11	34	31	24	12	119
Occupational disease due to chemical exposure	71	89	115	94	91	82	542
Chronic solvent-induced neurotoxicity	38	98	63	72	42	54	367
Occupational cancer	1	1	3	2	4	5	16
Occupational illness due to infection	28	41	49	61	45	58	282
Occupational noise-induced hearing loss	103	216	575	612	597	353	2456
Occupational overuse syndrome/ osteoarthritis	25	263	760	828	826	426	3128
Occupational skin disease	11	43	91	106	36	24	311
Total	**706**	**925**	**1919**	**1954**	**1803**	**1133**	**8440**

Table 2: Confirmed NODS cases for the period 1 March 1992 to 30 June 1998

Code	Description	Solvents	Chemical	NIHL	Respiratory	OOS	Dermatitis	Infectious	Total
12,11,13	**Managers, legislators and senior officials**	2		37	5	37	3		**84**
21	**Physics/math/engineering professionals**		2	41	25	16	3		**87**
22, 32	**Health professionals** *(doctors, nurses, vets, pharmacists, lab technicians, etc.)*		40	14	13	30	8	4	**109**
23, 33	**Teachers and teaching associate professionals**	1	7	3	21	3	3		**38**
24, 34	**Other professionals** *(lawyers, accountants, police, musicians, journalists)*			28	3	241	5	1	**268**
31	**Physical and engineering science associate professionals**	1	3	27	17	24	4	3	**79**
41	**Office clerks**	1	3	23	4	613	3	1	**648**
42	**Customer service clerks**		1	4	3	214	3	0	**225**
51	**Service workers** *(cooks, hairdressers, firefighters, home care workers, undertakers)*			28	1	21	19		**69**
52	**Salespeople, models and demonstrators**			4		13	1		**18**
61, 92	**Agriculture and fisheries workers**		14	65	11	34	8	52	**184**
71	**Building and extraction trade** *(carpenters, plumbers, painters, spraypainters)*	25	18	85	232	21	22	1	**404**
72	**Metal, machinery** *(welders, mechanics, tool makers, fitters, blacksmiths)*	1	26	128	85	16	10	1	**267**
73	**Precision handicraft workers** *(jewellers, potters, glass-makers, wood turners, printers)*	9	2	9		25	7		**52**
74	**Other craft and related trade workers** *(butchers, bakers, cabinet makers, tailors, upholsters, shoemakers)*	4	9	90	10	160	26	62	**361**
81	**Stationary plant and related operators** *(power plant operators, chemical plant operators, wood processing operators)*	2	7	59	30	11	7		**116**
82	**Machine operators and assemblers**	2	16	101	33	109	23	2	**286**
83	**Drivers and mobile plant operators**		1	32	16	8	2	2	**61**
91	**Sales and services elementary occupations**	4	2	15	2	29	9	1	**62**
93	**Mining and construction labourers**	2	9	81	80	47	20	5	**244**
	Retired	1		125	13				**139**
	Total	**54**	**154**	**1003**	**586**	**1690**	**186**	**138**	**3801**

Commentary

Asbestos-related disease

There have been 598 asbestos-related disease cases confirmed by the Asbestos Medical Panel since the register's inception in 1992.

The cases fall into the following categories:

- Mesothelioma: 113 cases
- Asbestosis: 139 cases
- Lung cancer: 52 cases
- Pleural abnormalities: 294 cases (this category includes: pleural plaques; pleural thickening; chronic fibrosing pleuritis; pleural effusions, but does not include pleural disease occurring together with mesothelioma, lung cancer or asbestosis).

The cases are predominantly male, making up 584 (98%) of the cases. Females do, however, account for 8% of the mesothelioma cases.

A gradual increase in mesothelioma cases has occurred throughout this decade. Lung cancer is under-notified because of the confounder cigarette smoking, and pleural disease is increasing.

Occupational asthma

There have been 405 suspected cases of occupational asthma notified since NODS was established in 1992. Of these cases, 116 have been confirmed as occupational asthma.

The causative agents and occupations involved in the 116 confirmed cases are shown in Table 3.

Table 3: Occupational asthma: causative agents and occupations

Causative agent	*Cases*	*Occupations*
Organic materials	**23**	
Animal proteins	8	Poultry worker, confectionery process worker, seafood processor
Cereal dusts	6	Bakers
Wood dusts	8	Boat builders, carpenters, joiners, manager
irritant dusts	1	Fertiliser manufacturer
Chemicals	**45**	
Aldehydes	6	Boat builder, radiographers, medical technician, medical manager.
Epoxy resins	5	Painter, metal finisher, process worker, boatbuilder.
Isocyanates	27	Spray painters, painters, process workers, boatbuilders, carpenter, foundry worker, technicians, floor sander, furniture manufacturer
Chlorine-based cleaners	3	Dairy process workers
Organophosphate	1	Cleaner
Ozone	2	Moulding room assistant, office manager
Colophony	1	Baker
Metals	**29**	
Aluminium	21	Aluminium smelter workers
Welding fumes	8	Engineers, fitter welders, mechanic, metal fabrication
Miscellaneous	**18**	
Acrylates	1	Lab technician
Colophony containing solders	4	Electrical process workers
Dibutyl phthalate	1	Printer
PVC, polypropylene, fume	3	Factory manager, baker
Detergent enzymes	1	Soap manufacturer
Polyurethane foam and fumes	2	Foam manufacturer, sheet metal worker.
Triglycidylisocyanurate	1	Powder coating process worker
Unrecognised	**7**	Dairy process technician, assembly process worker, spray painter, oven cleaners

Asthma notifications to NODS continue to come from doctors and nurses with an interest in occupational matters. The scheme continues to reveal cases of occupational asthma arising from causes that are recognised in the medical literature but are not well established in the public or medical profession's awareness. Doctors notifying to the scheme do, however, seem aware of the relationship between isocyanate exposure in the painting, foam and construction industries and the subsequent development of asthma.

Two cases of lung disease (occupational asthma and extrinsic allergic alveolitis) have been attributed to exposure to organic dusts (peat moss and bark dust). These conditions arose from bacterial and fungal contamination of the organic material. The extrinsic allergic alveolitis (EAA) patient was sufficiently ill to require a period in intensive care. The employer of the occupational asthma case failed to take the diagnosis provided by the employee's general practitioner seriously and made no investigations of the worksite or of the employee. The employer was prosecuted and convicted under section 10 of the Health and Safety in Employment Act 1992 for failing to monitor the employee's health status, a precedent-setting prosecution under this legislation.

The hazard was controlled in the EAA case by substituting sand for the peat moss and bark product and the employee stayed at work. The case of occupational asthma had to change jobs to avoid

exposure, elimination not being possible and isolation and minimisation being impracticable. Many cases have left their work by the time that notification occurs, a sensible course of action when suffering from such a debilitating disease but a reminder to employers, employees and medical providers of the significant impact on the individual of acquiring occupational asthma.

Because of its comprehensive respiratory surveillance programme, the Tiwai Point aluminium smelter identifies and notifies its cases of pot room asthma. The incidence of this condition seems comparable to overseas smelter experience but because of the paucity of notifications from other industries, "pot room asthma" achieves an undue prominence.

These case histories reinforced the need for good workplace hygiene, workplace monitoring and the need to choose broad-based control measures rather than relying on personal protection alone. Once the employee's symptoms are significant, resolution is often only achieved by leaving the workplace.

Other "classical" causes of occupational asthma (e.g. staff dealing with animals (veterinarian and laboratory), wood dust in carpenters, cabinet makers and joiners), are poorly reported. It is the view of the Occupational Asthma Panel that this represents a failure of notification rather than an absence of these causes.

The key to making an accurate diagnosis of occupational asthma remains based on:

- A thorough history including an occupational and exposure history. Questioning about symptoms should focus on the relationship of symptoms to work exposures.
- Carrying out peak flow measurements both at work and away from work. The Asthma Panel recommends recording the best of the three blows taken four times per 24 hours (before shift, in the middle of shift, after shift and on retiring) when the patient is off medication and at a time that they are both at work and away from work. Regular peak flow recordings (at work and away from work) remain the cornerstone on ensuring a person's symptoms are indeed related to workplace exposure.

OSH officers are available to help elucidate these problems if doctors wish.

OSH will shortly launch a complementary system aimed at gathering information from respiratory specialists. However, this system will not involve workplace assessments by OSH officers. Continued notification of occupational asthma by doctors is necessary.

For further information on occupational asthma, readers are referred to the OSH publication *A Guide to the Management of Occupational Asthma*, which is available from any OSH branch.

Occupational disease from chemical exposure

Chemical poisonings, both traditional and new, continue to be notified to OSH (Table 4), indicating that the New Zealand workplace remains a hazardous place in which to work.

Table 4: Main categories of confirmed chemical notifications

Chemicals in hospital x-ray and photographic departments	48
Chemicals in agriculture, horticulture and silviculture	14
Chemical fumes	23
Metal fumes	10
Lead	46
Mercury	4
Solvents	10
Cadmium	1
Arsenic	2
Others	30

Hospital x-ray and photographic fumes

Glutaraldehyde and other chemicals used in this process continue to be notified as a cause of symptoms (headaches, nausea, light-headedness, dermatitis) to workers engaged in these activities. It is recognised that glutaraldehyde can cause health problems among exposed workers even when the

degree of exposure is well below the recommended limits. Work circumstances still require attention and organisations engaged in these processes should be familiar with two publications:

- *The Safe Occupational Use of Glutaraldehyde in the Health Industries*. Department of Labour, 1992.
- *Radiographic Film Processing Procedures — Guidance Notes for the Provision of a Safe Work Environment and Safe Work Practice for Radiographers and Darkroom Technicians*. ACC, 1986.

Other chemical fumes

Poisonings have been notified to OSH following exposure to ammonia, phosgene, isocyanates, chlorine, hydrogen sulphide, methylene chloride, formaldehyde and styrene.

Metal fumes

Fumes from welding (zinc and lead) and foundry casting processes were reported. It is a common occurrence to welders, gascutters, metal sprayers, and occasionally when casting zinc metal. It presents as a 'flu-like illness several hours after the work is over. The worker recovers in 24-36 hours, and could contract the illness again later in the working week if re-exposed. Repeated exposure to mixed metal fumes (zinc, copper, manganese, aluminium) or intense exposure as the result of

overtime can lead to a pneumonitis and on occasions, fatigue becomes a feature of the illness, together with mood and memory impairment.

Lead and mercury

These two "ancient" metals, as they have been referred to, continue to cause illness. Lead poisoning is the most frequently occurring serious metal poisoning among workers in New Zealand. The two major occupations associated with lead poisoning were paint removal (sanding) from houses containing lead-based paints, and radiator repair and motor reconditioning activities.

A five-year review of NODS cases of lead poisoning notified to NODS indicated the following:

Work Activity	Numbers of Cases	Mean and Range Whole Blood Leads micromols per/litre
Paint stripping	22	2.5 (1.2 - 4.4)
Radiator repair	7	1.8 (1.3 - 3.1)
Foundry	5	2.4 (1.9 - 3.0)
Soldering	2	2.3 (1.8 - 2.8)
Pigment mixing	3	2.8 (2.4 - 3.2)

(Lead levels above 1.5 micromoles per/litre are considered excessive.)

Readers are referred to two useful publications on lead poisoning :

- The OSH/Public Health Commission publication *Guidelines for the Management of Lead-based Paint*, 1995.
- The OSH publication *Guidelines for the Medical Surveillance of Lead Workers* 1994.

Chronic solvent-induced neurotoxicity

Up to the end of June 1998, the panel had seen and considered a total of 223 notifications of possible chronic solvent-induced neurotoxicity and had verified 86. Of these, 15 have been classified, using the 1987 International Solvent Workshop classification, as Type 1, and 71 as Type 2.

Type 1 refers to cases with symptoms only and no objective measure of impairment. These symptoms are considered to be reversible on ceasing or reducing exposure to solvents. Type 2 cases, on the other hand, do have objective change in brain function and are considered on the basis of overseas research to be largely irreversible. The panel has now carried out its own research on the long-term outcome of 21 cases diagnosed as Type 2 neurotoxicity.

Each of the participants in the study had a repeat neuropsychological assessment and clinical interview 6 - 42 (mean 27) months after ceasing exposure. An exposure score was calculated for each

classified each as follows: Exposure score = A x B x C, where A = years of exposure to solvents, B = occupational group (boatbuilders, spraypainters and floorlayers = 3; drycleaners, coating applicators and printers = 2; lab technician, shoe repair, graffiti removal and paint sales = 1), and C = a weighting (1.3 for those where exposure was considered excessive for the occupational group and 1 for the remainder).

Improvement in neuropsychological function was seen in 9 participants (42.8%). Eleven (52.4%) showed no improvement and one (4.8%) showed a slight worsening of cognitive function (see Table 5). The improvement which did take place was, however, only partial, and only one returned completely to normal. The exposure score ranged from 7 to 109 and showed no association with severity of neurotoxic effect, improvement on follow-up neuropsychological assessment or time away from solvents.

The effect of solvents on any individual is therefore likely to be multifactorial, and not just related to the total exposure. Individual susceptibility probably plays a part. It is known, for example, that there are genetic differences between people in the ability of their body to metabolise solvents. Factors such as previous moderate alcohol intake or mild head injury may also increase susceptibility to solvent toxicity.

Table 5: Summary of chronic solvent-induced neurotoxicity cases

Case no.	Exposure score	Brain function impairment 1st test	2nd test	Change	Mood change 1st test	2nd test	Change	Still at work?
1	13	1	1	same	2	2	same	yes
2	18	1	2	worse	2	2	same	
3	84	2	2	same	3	2	better	yes
4	65	2	2	same	2	2	same	yes
5	53	2	2	same	4	4	same	no
6	26	2	2	same	3	3	same	yes
7	42	2	1	better	3	1	better	yes
8	52	2	2	same	1	1	same	yes
9	35	2	2	same	2	2	same	yes
10	40	2	2	same	2	2	same	yes
11	44	2	2	same	2	2	same	yes
12	20	3	2	better	3	3	same	no*
13	17	3	3	same	2	2	same	
14	94	3	2	better	5	3	better	no
15	78	3	2	better	3	3	same	yes
16	55	3	3	same	5	5	same	no
17	109	3	2	better	3	3	same	no*
18	8	3	2	better	3	2	better	yes
19	44	3	2	better	2	2	same	yes
20	94	4	4	same	5	5	same	no
21	7	4	4	same	5	5	same	no

Impairment: 1 = normal, 2 = mild, 3 = moderate, 4 = moderate-severe, 5 = severe
* not working for unrelated problems

The study confirmed previous research that the more severely affected are more likely to cease work, although this was not statistically significant in this small sample. The social consequences of job loss are, however, so profound, and the difficulties of

retraining so great in people impaired by solvents, that it is important whenever possible to conserve employment in this group of people.

Occupational cancer

Among the 15 cases of queried occupational cancer recorded in the NODS database, only one could be confirmed. This was a nasal cancer in a worker exposed to chemicals in the timber treatment industry. Clearly, notification of work-related cancers is sparse. Whether this is due to the non-occurrence of such cancers in the New Zealand workplace, or to the failure to determine causation, is not known. As a result, a proposal has been put forward to establish a Occupational Cancer Panel which aims to identify cases of previously unidentified occupational cancer, leading to identification of carcinogens in workplaces. The panel's overall objective is to reduce workplace deaths due to occupational cancer. The panel should be operational by late 1999.

Occupational infectious diseases

The most common condition notified was leptospirosis, for which there were 180 notifications. It occurred to workers in such occupations as farmers, meatworkers and meat inspectors. One case of interest occurred to a septic tank cleaner who visited a pig farm to clean the septic tank. Another case occurred to an individual who cared for a large number of stray dogs.

Although it is believed that New Zealand is free of brucellosis, seven cases have been notified.

Campylobacter and salmonellosis have occurred to farm workers, although one case was notified in a truck driver who collected some carpets which had been contaminated by sewage.

Orf continues to occur to meat workers and farmers, with notifications approaching 35.

Occupational noise-induced hearing loss (NIHL)

The OSH definition of NIHL is based on:

- A history of exposure to noise at work;
- A symmetrical hearing loss unless there is a history of unilateral noise exposure;
- A hearing loss at 4000 Hz of at least 30 dB which is at least 15 dB worse than the loss at 2000 Hz; and
- A recovery at frequencies above 6000 Hz (this is not a constant finding).

2411 cases have been validated, of which 95% were male. The average age at notification was 50 years, with little difference between males and females.

Occupational overuse syndrome

Known variously as OOS, RSI or cumulative trauma disorder (CTD), this condition continues to be widely notified among office workers, freezing workers and assembly workers. The number of

notifications until the end of June 1998 was 3128. The use of the term OOS is being discouraged as a diagnosis as it blurs the distinction between well-defined and well-recognised conditions and those conditions which are typified by general and widespread pain. Table 6 is a classification using ICD-10 codes, and the lists in the table are not exhaustive. Controversy surrounds local and general chronic pain disorders known as myofascial pain

Table 6: Occupational overuse syndrome

Localised conditions
Trigger finger M65.3 G56.0
de Quervains tenosynovitis M65.4
Bursitis M70.5
Palmar fascial fibromatosis M72.0
Plantar fasciitis M72.2
Rotator cuff syndrome M75.1
Medial & lateral epicondilitis M77.0 M77.1

Nerve compression syndromes
Carpal tunnel syndrome G56.0
Thoracic outlet syndrome G54.0
Ulnar nerve compression G56.2
Radial nerve compression G56.3

Regional or general pain syndromes
Fibromyalgia M79.0
Myofascial pain syndrome M79.0
Reflex sympathetic dystrophy M89.0
Regional pain syndrome
Chronic pain syndrome

syndrome (MPS), and fibromyalgia syndrome (FMS). Each case needs to be reviewed on its merits to determine the role of work as a causative agent.

Case studies

Acute solvent poisoning in a spray painter

A male aged 26, in good health, commenced a position as a spraypainter in the auto trade. Within two weeks, he felt on a high during the week. He felt thick in the head and sleepy, with sore eyes. Away from the workplace, he did improve slightly at the weekend.

During the following weeks, the symptoms progressed. He suffered extreme fatigue, loss of concentration and motivation in his work. At home he felt irritable. On one occasion, he spraypainted for six continuous hours and felt nauseated and dizzy, with a severe headache. This was still present the next morning. He visited his GP and was put off work for a week.

He recovered gradually and returned to work but not to spraypaint. The GP notified OSH and they visited to investigate the workplace. Their results revealed inadequate ventilation and solvent content in the work area.

This man suffered an acute solvent exposure. Some people persist in poor working conditions such as described and develop a low tolerance. After some years in the industry, these workers are at risk of developing a chronic condition known as solvent neurotoxocity, which cannot be completely cured.

Leptospirosis in a farmhand

Leptospirosis is an infectious disease caught from animals (e.g. livestock, cattle, pigs) as a result of contact with infected urine. A farmhand aged 30 milked 150 dairy cows twice daily. A month into his contract, he developed a flu-like illness with head aches, aching muscles and nausea which persisted. He visited his GP, who ordered a blood test to assist diagnosis. He had a further test to confirm the infection which proved positive. He was off work for 10 days.

OSH were notified by his GP and investigated under the NODS system. The dairy herd had not been immunised against leptospirosis. The employer had not informed the employee when he commenced employment of these issues or of preventative measures to avoid infection, i.e. scratches and cuts to the skin to be covered. As a result, the farmhand had little knowledge of leptospirosis and how to avoid contracting this disease. Preventative measures he could have taken include the washing of hands using a clean source of water following urine splashes. Aprons and boots should have also been made available by the employer.

Pentachlorophenol poisoning in a timber worker

A male aged 53 worked in the timber industry from age 24 until age 43. During his time, he worked on the "green table" for some 15 years. The "green

table" was where wet treated timber was sorted. At the end of the work shift, the man's clothing was soaked with chemicals over his thighs and chest. He suffered from a pertinent rash over these areas, as well as headaches and a blocked nose. He sweated a lot, soaking his bed clothes, and the sweat was malodorous. In time, he noticed developing fatigue, with an inability to do a double shift if asked.

At the time he was assessed, some 30 years after first exposure, he complained of memory, mood, and concentration difficulties. He also suffered a lot of muscle pain. He was reviewed by a clinical neuropsychologist experienced in assessing toxic exposures. The review indicated delayed memory recall, delayed recall of a Rey figure at a level significantly below average, and other tests confirmed impaired frontal lobe function. The review noted no history of concussion, drugs, and no alcohol for the last 15 years (possible confounding factors in diagnosis).

This case was one of a series being reviewed by a specialist ACC panel. Results to date indicate that of 28 fully assesses, 9 have been confirmed as suffering from long-term effects of PCP exposure. A preliminary paper on PCP exposure in industry was published in the *New Zealand Medical Journal* in 1998 (111: 362-4) entitled "Health Effects of Occupational Pentachlorophenol Exposure in Timber Sawmill Workers - A Preliminary Study".

Lead poisoning in a boat repairer

A man aged 56 began to strip and rebuild a 50ft boat. The paint was burned off with a hot air gun. The seams contained a red lead putty which was removed by a scraper and file. It was so hard, however, that a skill saw was used to cut through the seam between each plank. This created a lot of dust. The lead ballast was held in concrete and was separated with a jack hammer.

The lead ballast was later melted down in a shed, some of which (6 tonne) was subsequently used on the keel of the boat and was planed off by another worker using an electric sander. The boat was then repainted using a paint containing 10-30% red lead pigment. Numerous sandings of the paint occurred. The man swept and vacuumed the dust. Protective masks were worn intermittently.

Some nine months after beginning work, the man felt unwell, with loss of appetite, nausea, and a metallic taste in his mouth. He was extremely fatigued and suffered from gout of his elbow. His wife was concerned about his memory and concentration. She said he had unexplained mood swings and slept badly. Initially his symptoms improved when he stopped work on the boat but recurred when he recommenced.

Since stopping, his recovery has been delayed. Blood tests ranged from 1.75 micromols per/litre to 1.8 micromols per/litre — not high but when associated with his work history and his symptom pattern supported the diagnosis of lead poisoning.

Dermatitis in a young hairdresser

An 18-year-old apprentice hairdresser commenced work at a busy hair salon. Her tasks included washing hair at the basin, cleaning up, mixing and applying colours under supervision. After two months, her hands became itchy, red and the skin was broken in places.

She visited her GP, who diagnosed contact irritant dermatitis, and she was prescribed steroid cream for her hands. Following several return visits, the GP notified OSH and an occupational health nurse visited the salon to carry out an investigation

Her work practices were observed and the use of clean gloves were advised. It was also recommended that she alternate tasks, e.g. hair washing to be shared with all employees in order to avoid constantly having her hands in water. Careful handwashing and drying were advised. Continued monitoring by the employer of the affected hairdresser on a day-to-day basis was suggested in order to reduce further damage to the apprentice hairdresser's hands.

Unfortunately some people in this industry find they cannot continue working as hairdressers owing to the nature of the work that their hands are exposed to.

Trichloroethylene poisoning in a maintenance worker

A 44-year-old maintenance worker was asked to

repair a degreasing tank. The tank was sent to the maintenance department, and before commencing repairs, the worker drained the remaining trichloroethylene (methyl chloroform) from the tank. He then began the repair process using an arc welding machine. At the end of two or three hours, the worker noticed some chest tightness and developed shortness of breath. He was off work for two weeks as a result of exposure to phosgene gas, a breakdown product of trichloroethylene resulting from the high temperature of the arc on the trichloroethylene containment.

This case came to light following an investigation in which a number of workers complained of light-headedness when working around the degreasing tank. It was found that the tank had developed a leak, with the resulting loss of trichloroethylene. The heating coil was exposed and the remaining trichloroethylene was volatilised, producing a high concentration. The smell was detected some metres away (the odour threshold is 1000 mg/m^3). Although no air measurements were taken, it was estimated that the air levels would have been in excess of 1000 mg/m^3, well above the Workplace Exposure Standard of 680 mg/m^3 (1994). (In 1997, a NIOSH publication quoted a level of 270 mg/m^3).

The case emphasises that old problems continually recur and ongoing education and/or information is vital.

Occupational asthma caused by sensitisation to detergent enzymes

A 39-year-old maintenance engineer worked in both the production and packaging areas of a laundry detergent manufacturer. Regular sampling was carried out at the factory to ensure that levels of detergent enzymes did not exceed desirable levels. One such excursion was detected and was associated with an episode of plant failure and a spill of product. At this time, the maintenance engineer was working in the vicinity on ventilation equipment.

As a result of the spill, the maintenance engineer reported increasing respiratory symptoms of cough, production of phlegm, wheezing and shortness of breath. These symptoms improved while on holiday from the plant but were sufficiently severe to require several medications. Three other employees who were also exposed had acute irritant symptoms which settled without ongoing problems.

Lung function tests were normal (on treatment) but a provisional diagnosis of an occupational asthma secondary to detergent enzymes was made. The diagnosis was supported by the presence of serum antibodies to the detergent enzymes suggesting an allergic asthma. Removal from exposure (changing industries) resolved the symptoms and reduced the need for medication.

This was a case of an occupational asthma arising from exposure to a known provocative agent. The

workplace controls were good but small exposures over many years had caused sensitisation. Given the maintenance functions of his job, it is probable that the maintenance engineer had had some minor but ongoing exposures throughout his 13 years in this occupation. An incident affecting several employees acutely revealed this particular situation.

Appendix 1: Current NODS panels and membership

Asbestos and Mineral Dust Medical Panel

Professor Bill Glass (convenor)
Dr Robert Armstrong
Dr David Fishwick (overseas consultant)
Dr David Jones

Chronic Solvent Neurotoxicity Panel

Dr Evan Dryson (convenor)
Dr Neil Anderson
Dr Jenni Ogden

Asthma Panel

Dr Chris Walls (convenor)
Dr Margaret Wilshire
Dr Julian Crane
Dr Colin Wong
Dr John Gillies

Chemical Toxicity Panel

Professor Bill Glass (convenor)
Dr Andrew Macfie
Dr Michael Beasley
Mr. Errol Hodgkinson

Appendix 2: Other OSH publications

Below is a short list of occupational health publications available from OSH. A fuller list is also available from any branch office: also visit our web site: http://www.osh.dol.govt.nz

Asbestos Exposure and Disease: Notes for Medical Practitioners

Back in Care: Preventing Musculoskeletal Injuries in Staff in Hospitals and Residential Care Facilities

Chronic Organic Solvent Neurotoxicity: Diagnostic Criteria

Guidelines for the Medical Surveillance of Lead Workers

Manual Handling — Guidelines for the Workplace

Occupational Overuse Syndrome — Checklists for the Evaluation of Work

Occupational Overuse Syndrome — Guidelines for Prevention and Management

Occupational Overuse Syndrome — Treatment and Rehabilitation: A Practitioner's Guide

Practical Guidelines for the Safe Use of Organic Solvents

Safe Occupational Use of Glutaraldehyde in the Health Industries

The Pocket Ergonomist — Clerical/keyboard and retail/industry versions

Appendix 3: Departmental medical practitioners

The following departmental medical practitioners can be contacted through the OSH branch offices listed. Appendix 5 details branch addresses and phone numbers.

Practitioner	*OSH branch office*
Dr Evan Dryson	Penrose
Dr Chris Walls	Manukau
Dr Martin Robb	Hamilton
Dr Ian Bisset	Rotorua
Dr Paul Veitch	New Plymouth
Dr Lissa Judd	Lower Hutt
Professor Bill Glass	Nelson
Dr David McBride	Dunedin
Dr Gordon Hancock	Invercargill
Dr Greg Beacham	Napier
Dr John Gillies	Christchurch
(position vacant)	Tauranga
Dr Jonathon Morton	Palmerston North
Dr Bruce Gollop	Whangarei

Appendix 4: Departmental occupational hygienists and other specialist staff

The following departmental occupational hygienists and specialist staff can be contacted through the OSH branch offices listed. Appendix 5 details addresses and phone numbers.

Senior Scientists	*OSH Branch office*
Errol Hodgkinson	Christchurch South
Mel Tyson	Lower Hutt
Mark Fielder (Forestry)	Rotorua
Occupational Hygienists	
Garry Trotman	Penrose
David Appleby	Wellington
Nick Matsas	Hamilton
Jim Sutton	Palmerston North
Rod Dickson	Christchurch North