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Hazardous substances

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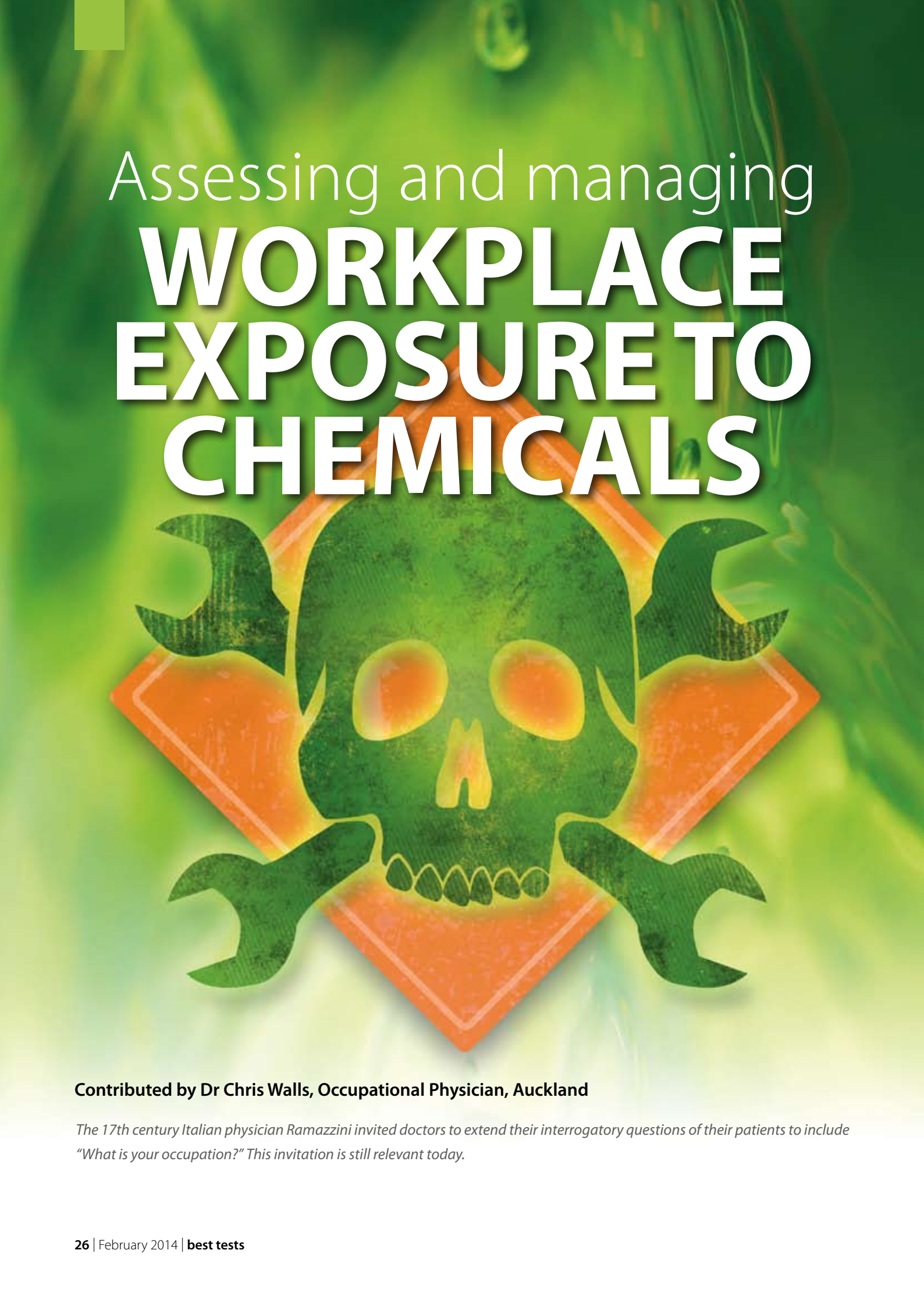
Assessing and managing WORKPLACE EXPOSURE TO CHEMICALS



Contributed by Dr Chris Walls, Occupational Physician, Auckland

The 17th century Italian physician Ramazzini invited doctors to extend their interrogatory questions of their patients to include "What is your occupation?" This invitation is still relevant today.

Many people present to their General Practitioner concerned about the possible health consequences of chemical exposures. Despite this, the health effects of the commonest workplace chemical exposures are often overlooked.

Assessment of patients with such exposures, and their clinical outcomes, is complex and difficult, with many cases presenting as more conventional illnesses. Failure to recognise a problematic exposure, coupled with on-going exposure, can lead to medical conditions that are difficult to manage.

Effective evaluation of possible chemical exposures contributing to a health concern requires consideration of a person's occupation as part of their clinical history, as well as some knowledge about the effects of specific chemical exposures.

The potential medical consequences of workplace chemical exposures

Workplace chemical exposures can result in the development of a new medical condition, however, the more common consequence is a worsening of a pre-existing condition. For example, a welder who has asthma may develop more brittle asthma as a result of exposure to welding fume.

The impact of adverse workplace exposures on a person with compromised health is often under-recognised. In such situations, the workplace exposure initiates the "illness cascade". For example, a worker who is obese, with poorly controlled diabetes, who smokes, and who works in an enclosed environment with petrol/ diesel powered equipment without adequate ventilation (carbon monoxide exposure), is suddenly required to undertake some excess physical activity (emergency response); this can lead to angina or collapse.

Assessing a patient's concerns

In order to detect health consequences from any exposure of concern, it is necessary to identify which substances are involved, and understand the likely effects of these chemicals. Patients who present with concerns about hazardous substance exposure without any particular exposure history (or specific substance of concern) are particularly challenging to assess.

Once the substance of concern has been identified, a clinical history should identify the patient's occupation and in particular what the tasks and likely exposures are. The health

Notification of disease and injury from hazardous substances exposure

Cases of injury or disease relating to hazardous substances, and wider poisonings arising from chemical contamination of the environment, require notification to the Medical Officer of Health under the Hazardous Substances and New Organisms Act 1996 and the Health Act 1956.

A short electronic notification form is available on the *bestpractice* dashboard (log in at www.bestpractice.org.nz or go directly through MedTech) – look for "Hazardous Substances & Lead Notifications". Primary care practices that do not use *bestpractice*, should still inform their Public Health Unit of any notifications. Access to the notification form for non-MedTech Patient Management Systems will be available later in 2014.

The employer is expected to notify the Ministry of Business Innovation and Employment when an illness arises from workplace exposures, but this is not a requirement of the General Practitioner.

ACC carries out its own determinations according to its Act, and it is possible for a worker to suffer a work related illness but not meet ACC's criteria for assistance.

Common illness presentations resulting from chemical exposures

Common workplace illnesses that are often misdiagnosed include:

- Metal fume fever or chemical pneumonitis ("welder's flu")
  - Sudden onset of fever, shortness of breath, cough and wheeze within 24 hours of exposure to metal or plastic fume from the welding process
  - Rarely diagnosed on the history; lung function tests are useful to confirm the diagnosis and recovery. A chest x-ray excludes other issues.
- Carbon monoxide exposure
  - Common, and occurs in unusual circumstances, e.g. prolonged chainsaw use in dense undergrowth
  - Often overlooked in the illness cascade leading to collapse
- Organic solvent exposures
  - Acute (intoxication) and chronic (encephalopathy) illness patterns from printing, painting/finishing and plastic industries
- Occupational asthma
  - From many industries, including pine wood processors, MDF manufacturing, cedar wood processing and car painting
- Pesticide/biocide exposures
  - Patients may present with chronic malaise, the cause of which can be difficult to confirm
  - Many of the more toxic biocides are no longer in use

consequences of chemical exposures depend not only on the material that people are exposed to but the route of exposure, metabolism and excretion. An important concept is "dose" – how much for how long?

The timing of symptoms is important. Symptoms that persist during an absence from work tend not to be related to the workplace. Ask about chronic effects of the exposure, but also try to identify episodes of acute toxicity around the exposure time. For example, pesticide spray exposures are often blamed for low grade chronic "unwellness" but a history of symptoms, such as acute malaise, skin rashes or shortness of breath, around peak exposure times (e.g. mixing concentrate, unexpected soakings) would suggest a more significant exposure.

Identifying health consequences of chemical exposure is only occasionally aided by specific testing of the patient or the workplace. These measurements are either of:

1. Exposure assessment, e.g. static sampling in a workplace or personal sampling of the worker (e.g. dust/fume measurements in the breathing zone of a welder). There are specific "acceptable" concentration limits for known hazardous chemicals (in New Zealand called Workplace Exposure Standards). However, measurement against these Standards is usually only done by concerned companies.
2. Effect assessment, e.g. peak flow measurements at work or away from work
3. Specific biological monitoring (very occasionally), e.g. blood lead levels

If physiological or laboratory measurements are possible, they might be taken both during and away from exposures.

In reality, there are few exposure assessment services available to General Practitioners, and physiological measurements ("effect assessments") are usually the only accessible tests in primary care.

There are currently few New Zealand governmental resources to assist General Practitioners with advice on assessment of workplace exposure to chemicals and illness this may cause. Potential sources of information/contacts include:

- The University of Otago Department of Preventive and Social Medicine

- The National Poisons Centre
- Occupational medical specialists
- Local occupational health services
- Medical literature

Other considerations

Many people at work fear that they place their job security at risk if they report their concerns about workplace conditions.

General Practitioners may be involved in a patient's dispute with their employer or the workplace insurer, e.g. providing a medical certificate. Such circumstances often complicate determining whether a workplace chemical exposure may be affecting their patient's health, confirming the suspected relationship, and advising on appropriate treatment or protection.

Management of occupational exposure

General Practitioners have two main roles in management of workplace exposure related illness: treating the symptoms, and providing the patient with appropriate information about preventing further exposures. An overall goal is to help the patient to maintain their work.

Many conditions are either self-limiting (the symptoms resolve when exposure ceases or shortly after) or can be attributed to historical exposures. Controlling the exposure at the source (e.g. ventilation, substitution with less toxic products) is optimal because it controls the symptoms and benefits employer and employee. In general, recommending "safety gear" (Personal Protective Equipment) is not a useful way to provide protection.

Where workplace chemical exposures cannot be reduced, or the health consequences of these are significant, advice about seeking suitable alternative work may be necessary. For example, when people develop allergies to workplace chemical exposures, they usually have to abandon that work.

The important message is that control of symptoms caused or worsened by workplace exposures becomes very difficult where the linkage between those symptoms and that exposure remains undetected. Enquire about a patient's occupation, and consider if workplace exposure to chemicals is causing or contributing to their symptoms.

Hazardous Substances

Hazardous Substances Disease & Injury Notification

GPs in all regions of New Zealand are now able to use e-notification to inform your Medical Officer of Health about hazardous substances, diseases and injuries.

By law, injuries from hazardous substances, lead absorption and poisoning arising from chemical contamination of the environment (including from agrichemical spraydrift) are required to be notified.

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For more information on these notifications see the article on page 34 of the April **Best Practice** journal http://www.bpac.org.nz/BPJ/2013/April/docs/BPJ52.pdf.

If you have any questions regarding a patient or notification, please contact your local public health unit.

bestpractice
DECISION SUPPORT FOR HEALTH PROFESSIONALS

Pyrethroid toxicity and its management

Contributed by Dr Michael Beasley and Dr Wayne Temple, National Poisons Centre

Pyrethroids are insecticides that are synthetic modifications of natural pyrethrins, which are extracts from the flowers of some *Chrysanthemum* species.

Pyrethroids have been developed for the control of household and agricultural insects, and human lice. Pyrethroids have a very high "selective toxicity" for insects compared to mammals, which is due to higher insect nerve sensitivity, lower mammalian skin absorption and more efficient mammalian hepatic metabolism. Traditionally, pyrethroids have been considered as having relatively low toxicity, particularly when compared to organophosphate insecticides. However ingestion of concentrated pyrethroid-containing products can cause severe, and occasionally fatal, effects.

Pyrethroid formulations include aerosol sprays, smoke coils, electric mats, oil formulations, emulsifiable concentrates and wettable and dustable powders. A shampoo and lotion formulation is also available for the control of human lice. The formulated products often combine the synthetic pyrethroids with a synergist, such as piperonyl butoxide (which inhibits their metabolism), and they may also contain other insecticides.

Physiologic effects of pyrethroids

Pyrethroids are ion channel toxins that interfere with the function of the nervous system. They modify the "gating" characteristics of neuronal voltage-sensitive sodium channels to delay their closure,[1] thereby prolonging neuronal excitation.

The toxic effects of pyrethroids result from this neuronal excitation and include a wide spectrum of signs and symptoms from paraesthesia and increased salivation, through to seizures and potentially death (Table 1). Allergic reactions, including contact dermatitis or asthma, are only rarely reported with synthetic pyrethroids.

Table 1: Toxic effects of pyrethroids

Mild pyrethroid toxicity	Moderate pyrethroid toxicity	Severe pyrethroid toxicity
Paresthaesia	CNS depression	Seizures
Nausea	Increased salivation	Coma
Headache	Fasciculations	Pulmonary oedema
Vomiting	Fever	Respiratory failure
Dizziness	Diaphoresis	
Fatigue	Blurred vision	
Anorexia		

Enquiries to the National Poisons Centre about pyrethroids

In the five year period between 2008 and 2012, the New Zealand National Poisons Centre (NZNPC) received 1544 enquiries about synthetic pyrethroids; 106 of these were from medical centres. Medical centres enquired about a range of pyrethroid products including agricultural insecticides, household aerosol fly sprays, household bug bombs and household liquid insecticides. Typical calls included:

- A patient who developed immediate nausea and rhinorrhoea, and a delayed skin rash, when treating livestock with a cypermethrin (synthetic pyrethroid) product without using protective measures
- A patient who developed a burning and tingling sensation on his face and neck after spraying his house with a pyrethroid insecticide
- An asymptomatic child who briefly activated an aerosol spray into her mouth.

Typical clinical presentation of patients with pyrethroid exposure

The largest risk of pyrethroid toxicity is from the ingestion of undiluted formulations. The presentation of patients with exposure to pyrethroids depends somewhat on the setting of exposure.

Occupational exposure to pyrethroids

Most reports on the adverse effects of pyrethroid exposure have arisen from occupational settings, particularly where insufficient precautions are taken during pyrethroid preparation and application.[2] People using pyrethroids in this setting may develop cutaneous paraesthesia as well as ocular and upper respiratory tract irritation. The cutaneous sensation, typically described as stinging or burning, may not develop until several hours post-exposure, and can be associated with erythema but not usually other skin lesions. Acute systemic symptoms have also been reported in cases of careless use of pyrethroids. There are few studies which have investigated the possibility of long-term adverse effects in people exposed to pyrethroids occupationally.

Household/indoor exposure to pyrethroids

The risk of pyrethroid toxicity is low when pyrethroids are sprayed indoors, e.g. in the home or office, by professional applicators. However, anecdotally it is not uncommon for some people to complain of a range of symptoms from such exposures. There is general agreement that a period of several hours (ideally at least 24 hours) should be observed between pyrethroid application and re-occupation of the building. Spray droplets can settle on furnishings, causing potential ongoing skin exposures, but it appears that re-entrainment of particles into air is minimal. If measured, floor or other surface levels can be an unreliable guide to air levels of pyrethroids.

The use of permethrin as a topical treatment or shampoo for head lice or scabies is associated with relatively low risk of toxicity, if used according to directions. However, the NZNPC is aware of some caregivers using pyrethroid-containing fly sprays to treat children's head lice. There is some risk with this; adverse effects can include scalp and face burning and itching, and ocular discomfort if sprayed into the eyes.

Management of pyrethroid exposure

If a patient presents with signs and symptoms of toxicity and a history of exposure to a pyrethroid, it is recommended to phone the National Poisons Centre for advice on management.

Patients with significant pyrethroid ingestion can present with severe symptoms and signs (Table 1) which would constitute a medical emergency, and should be immediately referred to hospital for life support measures and ongoing monitoring. General practitioners may occasionally need to commence standard emergency care. Seizures can be resistant to benzodiazepines and other pharmacotherapy; thiopental may be used in a hospital setting.[3]

Patients with an occupational exposure to pyrethroids may require symptomatic treatment for cutaneous paresthesia or upper respiratory tract irritation. While controversial, the use of creams containing vitamin E has been claimed to be useful for paresthesia,[4] although this treatment is more likely to be helpful if applied prior to exposure. Relief may be obtained by the use of lipophilic agents, such as cooking oil or white soft paraffin. A cool cloth or ice may also be helpful.

Persistent symptoms following indoor pyrethroid exposure may be reported, even when a period of time away from the environment has been observed. Complex psychosocial factors can play a role in this, similar to that seen with "sick building syndrome". The patient can be reassured that the presence of paraesthesia does not correlate with a high level of exposure, and that chronic neurotoxicity is unlikely from such exposures.[5]

Notification of pyrethroid toxicity

Cases of pyrethroid toxicity must be notified to the Medical Officer of Health, under the Hazardous Substances and New Organisms Act 1996.

A short electronic notification form is located on the bestpractice dashboard (log in at **www.bestpractice.org.nz** or go directly through MedTech) – look for "Hazardous Substances & Lead Notifications". Primary care practices that do not use *bestpractice* Decision Support software, should still inform their Public Health Unit of any notifications. Access to the notification form for non-MedTech Patient Management Systems will be available in early 2014.

Further information

For advice on toxic exposures to pyrethroids, phone the National Poisons Centre on 0800 POISON (0800 764 766).

For information on the treatment of head lice see: "Treating head lice", BPJ 14 (Jun, 2008).

For information on the treatment of scabies see: "Scabies – diagnosis and management", BPJ 19 (Feb, 2009).

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Laboratory investigation of

Exposure to metals or other hazardous substances in the environment

On occasion, General Practitioners will encounter a patient with a concern relating to possible exposure to a hazardous substance. These presentations can be very challenging – the symptoms may be non-specific, there may be no objective evidence of exposure, and the number of potential hazardous substances that the patient has been exposed to may be large. In this situation, laboratory investigation requires careful consideration. Testing is usually only useful if there is evidence of systemic toxicity, and a specific treatment option is available.

Contributed by Dr Stephen du Toit

If a patient presents with a possible exposure to a hazardous substance, what do you do?

Ask the patient if they have a suspicion as to the identity of the hazardous substance, the time and date of suspected exposure and any relevant occupational details if the exposure occurred during work.

Take a history and examine the patient. Assess blood pressure, pulse rate, respiratory rate, temperature, neurological status and presence of gastrointestinal disturbance, such as diarrhoea or vomiting.

As a subset of hazardous substances, diagnosing environmental metal toxicity can be difficult since symptoms and signs are usually non-specific. Diagnosis of metal toxicity generally requires three features to be present:

- A realistic source of exposure
- Symptoms and signs typical of exposure to the metal
- "Abnormal" levels of the metal in an appropriate biological sample

Metal toxicity should be considered in patients with:

- History of exposure
- Unexplained renal disease
- Symmetrical peripheral neuropathy
- Unexplained acute changes in mental/neurological function
- Acute inflammation of nasal or laryngeal epithelium

Examples of conditions that may be caused by metal toxicity include bilateral pain radiating from the feet to the leg with arsenic exposure, renal disease in spray painters with cadmium exposure and early onset of Parkinsonism (age < 50 years) with manganese exposure.

Who can you call?

If the patient has signs of acute toxicity or their history suggests significant and recent exposure, it is recommended to seek advice on management.

Options to consider include the National Poisons Centre (0800 POISON), the TOXINZ database (**www.toxinz.com** – requires a subscription), a Chemical Pathologist or the local district health board's Toxicologist.

Advice from these experts should include treatment options (if any) and collection of samples such as urine or blood to be stored for possible analysis.

What laboratory investigations are appropriate?

Testing for possible chemical exposure requires careful consideration. In general, testing is only useful if there is evidence of systemic toxicity, and a specific treatment option is available. In some situations baseline levels may be helpful and serial tests may also be required. Expert advice is strongly recommended prior to undertaking any testing. It is also recommended to contact the local laboratory to discuss collection of appropriate samples.

There is no single analytical technique that can identify all hazardous substances. Targeted testing (if available) can be used when attempting to identify a specific chemical, e.g. investigating lead toxicity (see: "Lead exposure").

Interpretation of blood and urine tests for chemicals can be complex. Laboratories use inductively coupled plasma mass

Lead exposure

Investigating lead level in a patient with exposure to lead, (e.g. lead-based paint) is an example of an appropriate targeted test.

Guidelines for managing exposure to lead are available from the Ministry of Health. The Medical Officer of Health should be notified of patients with blood lead levels ≥ 0.48 micromol/L. Children with a blood lead level ≥ 0.96 micromol/L and adults with a blood lead level ≥ 3.4 micromol/L should be referred to an appropriate specialist.[2] Patients with elevated lead levels should reduce (or eliminate if possible) exposure to lead and then be re-tested after six weeks and six months.

For further information see: "The environmental case management of lead-exposed persons", available from: **www.health.govt.nz**

spectrometry (ICP-MS) to determine levels of elements in blood or urine, but the analytic process involves "standardising" all ionic states to a single catatonic charge, which can mask toxicity.[1] For some metals, toxicity varies depending on the ionic state. For example, Hg (elemental mercury) is non-toxic, Hg^{2+} (mercury ions) is toxic and CH_3Hg (methyl mercury) is very toxic. Similarly, Cr^{6+} (chromium) is toxic but when it enters cells it is converted to Cr^{3+} which is non-toxic. Biological monitoring using ICP-MS cannot distinguish between toxic or non-toxic forms of chromium, so measuring the source of the possible exposure is more reliable.

What about other types of "toxicity testing"?

Performing wide-ranging screening tests (e.g. hair analysis – see sidebar) for any form of hazardous substance is seldom appropriate. The implications of a positive result need to be considered before a test is requested. All people are exposed to hazardous substances in the environment, and may have detectable levels without being "poisoned". In a normal reference interval, 5% of healthy patients will have results falling outside this range. An "abnormal" result may occur purely by chance, but may cause unnecessary concern. In addition, using population-based reference intervals established overseas may not be appropriate for people in New Zealand.

Tests requested (usually by the patient themselves) from overseas laboratories are particularly difficult to interpret and may result in over-diagnosis and unjustified concern, as well as incurring significant cost to the patient.

Hair analysis is not recommended

Hair analysis is valuable in forensic medicine when assessing acute toxicity, and in drug testing. Hair grows at a rate of 1.06 cm/month, therefore providing a timeline of exposure. While it seems reasonable to expect that hair analysis, using sophisticated modern analysers such as ICP-MS will be useful in assessing long-term exposure to toxic metals, this is not the case.[3]

There are several reasons for this:

- There are no international hair standards available to calibrate the analysers
- Analysis of the same sample by different laboratories yields different results
- Reference intervals are often calculated by using data obtained from testing the samples received. Ideally, reference intervals should be established using samples from healthy individuals. Since reference intervals are not well defined, more (or less) than the arbitrary 5% of healthy, non-exposed patients will have results that fall outside reference intervals.
- The probability of having at least one "abnormal" result increases with the number of tests performed. A large number of analytes (e.g. 20 – 40) are usually tested; the probably of at least one "abnormal" result is 65% for 20 tests and 87% for 40 tests, assuming the reference intervals include 95% of results obtained from healthy individuals[4]
- Patients are constantly being exposed to hazardous substances and hair will always contain some toxic elements
- Hair is exposed to the environment, and in general it is not possible to remove only external contaminants from hair. For example, arsenic deposits on the outside of the hair shaft with exposure to the environment (e.g. washing hair with arsenic-containing water). Arsenic is also deposited on the outside of the hair shaft when arsenic-containing water is ingested.

More research is required to define the correlation between the clinical state, hair analysis and blood test results.[4]

Do exposures to hazardous substances need to be reported?

By law, medical practitioners must inform the local Medical Officer of Health of patients with the following conditions:

- Lead absorption ≥ 0.48 micromol/L (Health Act 1956)
- Poisoning arising from chemical contamination of the environment (Health Act 1956)
- Hazardous substances disease and injury (Hazardous Substances and New Organisms Act 1996).

A hazardous substance is officially defined as anything that can explode, catch fire, oxidise, corrode, or be toxic to humans.

Electronic notifications of hazardous substance exposures (including lead exposures) may now be made through the *bestpractice* Decision Support module, introduced nationwide in 2013. These notifications are assessed by the Medical Officer of Health and Public health unit staff to determine if further follow-up with the patient is required.

Where the diagnosis of poisoning is unclear, discussion with the Medical Officer of Health may assist in deciding if notification is appropriate, what action might be taken, and what if any public health investigation is required.

ACKNOWLEDGEMENT Thank you to **Dr Stephen du Toit**, Chemical Pathologist, Hamilton for contributing this article.

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Hazardous Substances

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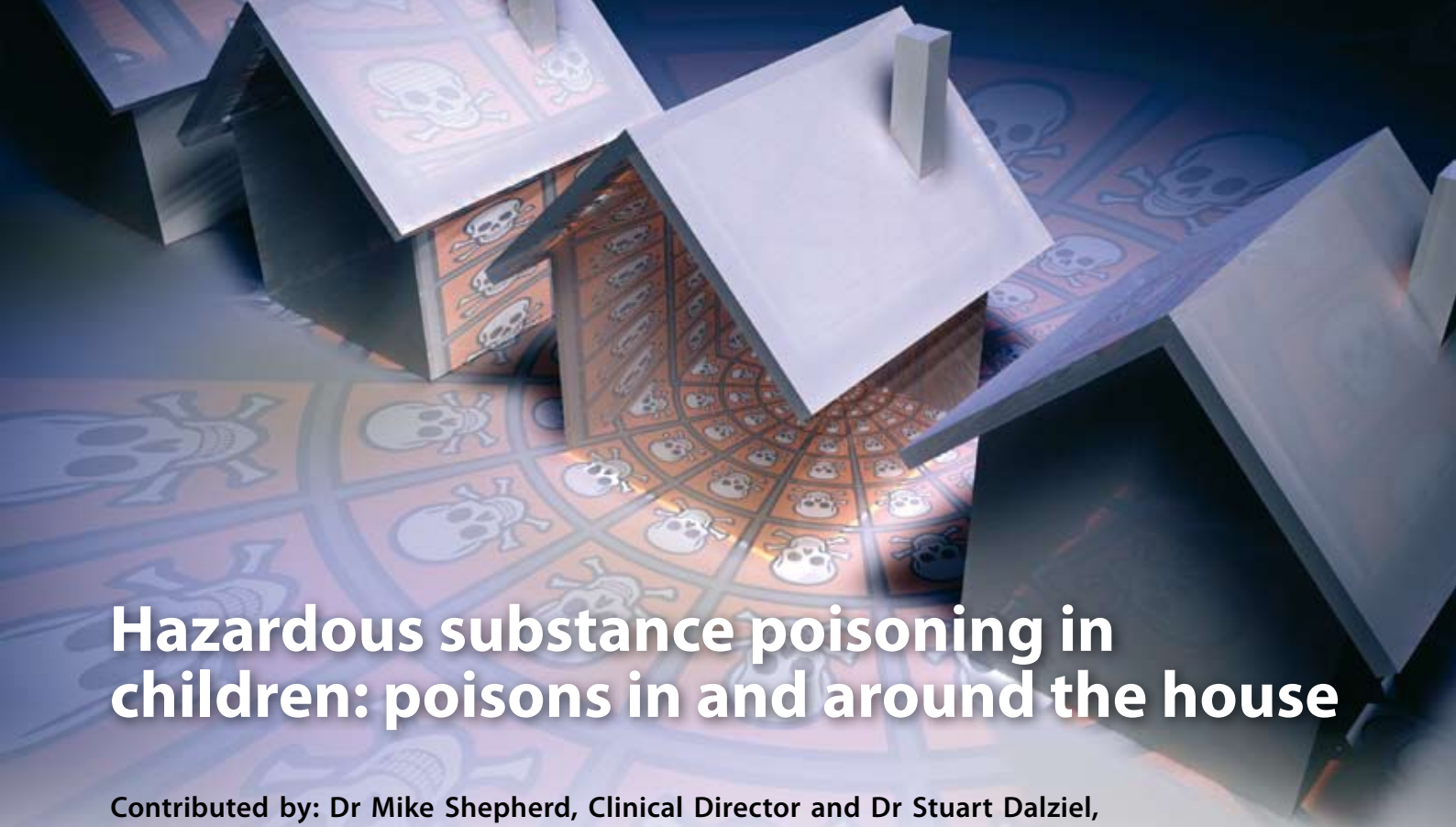
DECISION SUPPORT FOR HEALTH PROFESSIONALS



Hazardous substance poisoning in children: poisons in and around the house

Contributed by: Dr Mike Shepherd, Clinical Director and Dr Stuart Dalziel, Paediatric Emergency Specialist, Starship Children's Health, Auckland

Children are great explorers, and preschool children spend much of their time exploring at home. This can lead to children unintentionally being exposed to a number of hazardous substances. This article describes some of the common household poisonings, outlines their management and discusses their prevention.

The family home: welcome to the danger zone

Cleaning products

Exposure to cleaning products in the home is the cause of many unintentional poisonings in children. The most frequently involved toxins are bleach, low-molecular weight hydrocarbons (e.g. some house-hold solvents), acids/alkalis, detergents and ammonia products. These products have highly variable toxicity and highly variable packaging in terms of safety. There is an emerging issue with pre-packaged cleaning products, laundry detergents and particularly dishwasher tablets, as these appear attractive to small children.

Bleach is generally of low toxicity, with household solutions commonly containing less than 10% sodium hypochlorite (the active component of bleach). Children rarely ingest significant quantities as bleach is extremely unpalatable. Less than 100 mL of household bleach is unlikely to cause serious adverse effects. However, if children develop symptoms, they should be referred to hospital. Common effects include nausea, vomiting, and diarrhoea. Occasionally exposure to more concentrated bleach solutions may occur (industrial bleach may contain up to 50% sodium hypochlorite), presenting a risk of oesophageal injury (see below).

Acid/alkali ingestion such as dishwasher powder, drain cleaner and oven cleaner can cause severe corrosive injury. Oesophageal injury can occur without obvious lip or oral burns. Any stridor, dyspnoea, dysphonia, drooling or vomiting suggests serious injury to the airway or gastro-oesophageal tract and the child should be urgently referred to hospital. The child should be kept nil by mouth.

Ammonia solutions in household cleaners are at a concentration that does not cause corrosive injury, however occasionally exposure to more concentrated ammonia solutions occurs. These should be managed as for acid/alkali exposure.

Ammonia gas is highly irritant to mucosal surfaces and may be released when an ammonia-containing cleaning solution is mixed with a strong alkali, such as sodium hydroxide in drain cleaner. The child's eyes should be irrigated and they should be urgently referred to hospital if they have signs of respiratory irritation (cough, wheeze, stridor or respiratory distress).

Superglue (cyanoacrylate)

Cyanoacrylate adhesives have become a common household product. While exposure will not be lethal it can be both painful and distressing. Exposure may occur during exploration by child or if the glue is mistaken for an ear or eye drop due to similar packaging.

The general principles of managing superglue related injury are to:

- Immerse the bonded surfaces in warm soapy water
- Attempt to peel or roll the surfaces apart with the aid of a blunt edge, e.g. a teaspoon handle. Do not try and pull surfaces apart with a direct opposing action.
- Attempt to remove the glue with acetone, however, acetone should not be used in the mouth or on the eye

If lips are accidentally stuck together, irrigate with warm water and encourage maximum wetting from saliva and pressure from the tongue inside the mouth. Peel or roll lips apart.

If the eyelids are glued together, irrigate with warm water. Eyelids may then be able to be separated by rolling the lids. Otherwise trimming the eyelashes may be effective. If the eyelids still cannot be separated the recommended approach is overnight application of a wet eye patch, followed by ophthalmology review. Once the eyelids are separated, the eye should be carefully examined to ensure any fragments of glue are removed and corneal abrasion is excluded. Treat any corneal abrasion with chloramphenicol 0.5% eye drops, one drop, four times daily, for seven days, to prevent secondary infection. Ideally, children with corneal abrasions should be reassessed in 24 – 48 hours, and referred for review if the abrasion is not healing.

Nail-polish remover

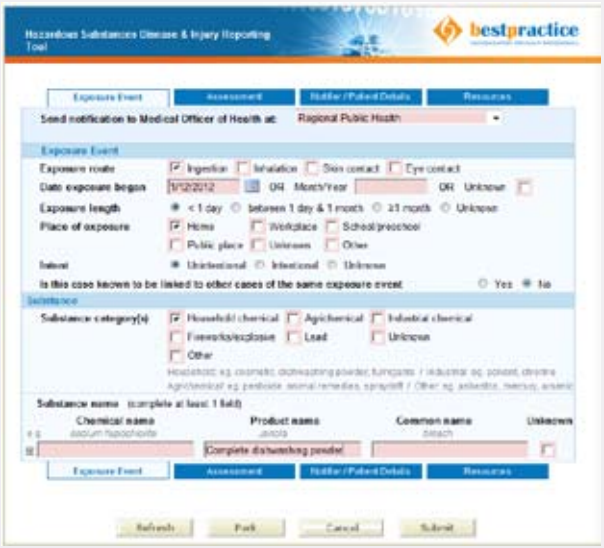
Nail-polish removers can be composed of a number of different products, including ethyl acetate, isopropanol and acetone (now less commonly used). The management of nail polish remover exposure is supportive. Charcoal is not recommended. If children are asymptomatic two hours after ingestion then no further treatment or follow up is required. Children with CNS symptoms should be referred to hospital.

Ethyl acetate has a local irritant effect to the skin, eyes, and mucous membranes that develops rapidly. If no symptoms occur over the first few minutes then exposure is likely to have been minimal. Only large ingestions result in systemic symptoms (gastrointestinal and CNS), and these symptoms are also likely to occur rapidly.

Isopropanol toxicity can cause CNS effects. Ingestion is best managed by observing the child for altered mental status. An

Notification of hazardous substances injuries

Any injury or disease caused by hazardous substances must be notified to the Medical Officer of Health, under the Hazardous Substances and New Organisms Act 1996. However, some medical practitioners may be unaware of this requirement. An electronic notification form is located on the bestpractice dashboard (log in at **www.bestpractice.org.nz** or go directly through MedTech) and look for "Hazardous Substances & Lead Notifications". Primary care practices that do not use bestpractice Decision Support software, should inform their Medical Officer of Health of any notifications manually.



observation period of two hours post-ingestion can be used to rule out clinical toxicity in paediatric patients.

Ingestion of small volumes of acetone can cause central nervous system (CNS) symptoms. The onset of symptoms is likely to occur rapidly but recovery may be slow. CNS symptoms may be followed by metabolic acidosis, cardiovascular compromise and coma.

Hazards outside the house

Although exposure to hazardous substances outside of the home is not as frequently implicated in unintentional child poisonings, a number of products used in the garage and garden present a risk.

Anti-freeze (ethylene glycol)

Ethylene glycol is rapidly absorbed and signs and symptoms similar to ethanol intoxication develop within four hours of ingestion (nystagmus, drowsiness, nausea and vomiting). Cardiorespiratory features may develop, leading to shock, seizures, coma and renal failure within several hours. All symptomatic patients, as well as those patients in whom exposure level is unknown, should be referred urgently to hospital. Patients with significant ingestion will develop metabolic acidosis. Patients presenting with unknown exposure level who have a normal bicarbonate level and a normal examination at four hours can be safely discharged.

Children with minor ingestions of ethylene glycol, e.g. a witnessed small taste, sip or a lick, do not require hospital evaluation and can be observed in the community unless symptoms develop.

Brief skin and inhalation exposure does not result in ethylene glycol intoxication. Skin exposure can be managed with soap and water. Ocular exposure should be managed with removal of contact lenses and irrigation with tap water at room temperature. This is usually sufficient; children with persistent ocular symptoms should have a formal ophthalmology examination.

Petrol

Ingestion of a small amount of petrol usually results in mild transient nausea and vomiting which can be managed in the community with observation. Administration of fluid "to dilute" or induce emesis, is not recommended due to the potential to further increase the risk of pneumonitis. Pneumonitis can be associated with ingestion and evolves over a few hours. Persistent coughing, gagging and respiratory signs may indicate aspiration and these patients should be observed in hospital.

Systemic CNS toxicity with onset of CNS depression, seizures and possible death within one to two hours can occur with larger ingestions/inhalations (usually >1-2 mL/kg). These patients require emergency transport to hospital. Fortunately such ingestions/inhalations are uncommon in unintentional poisonings in children. However, intentional "huffing" of petrol has resulted in deaths in New Zealand, and parents, caregivers and young people should be aware of the risks associated with this practice, and access appropriate support if needed, such as mental health or youth counselling services.

Dermal exposure to petrol should be decontaminated with soap and water. Ocular exposure should be managed with removal of contact lenses and irrigation with tap water at room temperature. This is usually sufficient; children with persistent ocular symptoms should have a formal ophthalmology examination.

Rodenticides (long-acting coumarin anticoagulants)

Common domestic rodenticides use long-acting anticoagulants or "superwarfarins" such as coumatetralyl, bromadiolone and brodifacoum. A child who has unintentionally ingested a single pellet does not require INR testing or medical review. Parents should be advised to seek medical attention if the child develops mucosal bleeding or bruising. Children who have ingested larger amounts of rodenticides should be evaluated for coagulopathy; it is estimated that a child needs to ingest > 30 g of a 0.005% (a standard concentration) preparation as a single dose to cause significant anticoagulation.

Cholecalciferol (vitamin D3) is also commonly used in domestic rodenticides and medical assessment is not required for single unintentional ingestions in children. Evaluation should occur if symptoms of hypercalcaemia occur.

Glyphosate

Glyphosate is present in common domestic herbicides, such as some Roundup, Zero Weedkiller and Weed Out products.

Ingestion of diluted preparations causes little concern other than mild gastrointestinal symptoms. Ingestion of concentrated preparations can lead to gastrointestinal symptoms (nausea, vomiting, diarrhoea and abdominal pain) as well as oropharyngeal/oesophageal erosions, aspiration pneumonia and hypotension.

Risk stratification in adults is based on volume of concentrate ingested:

- <50 mL – asymptomatic or minor gastrointestinal symptoms
- 50-120 mL – gastrointestinal symptoms
- 150-300 mL – severe gastrointestinal symptoms, risk of upper airways oedema and multi-organ failure
- >300 mL – potentially fatal.

In children risk stratification based on dose is less specific. However, children with minor ingestions do not require hospital assessment unless symptomatic.

Dermal exposure causes local irritation but not usually systemic toxicity. The skin should be decontaminated with soap and water; medical review is required only if the child is symptomatic. Ocular exposure should be managed with removal of contact lenses and irrigation with tap water at room temperature. This is usually sufficient; those with persistent ocular symptoms should have a formal ophthalmology examination.

Herbicides containing substances other than glyphosate are also available and care should be taken to read the label of the product ingested, and if necessary, information sought from sources such as the National Poisons Centre or the TOXINZ database (www.toxinz.com). Not all products from the same manufacture contain the same ingredients, further emphasising the need to read the label of the product ingested carefully and to confirm its exact name. N.B. glyphosate should not be confused with organophosphate poisoning, which is a separate toxidrome.

Prevention of unintentional exposure to potential toxins

Ideally the prevention of poisoning-related injury should form part of well child checks and primary care discussions. Specific recommendations include:

- All cleaning products and other potential poisons should be stored away from children; this includes using out of reach cupboards, locking cupboard doors and using child resistant catches on doors
- When getting products out to use, place immediately back into high storage, with closures correctly fastened
- Products should be supplied and purchased with child resistant packaging
- Products should always be stored in their original packaging and should be disposed of carefully
- Dishwasher detergent should be put into the machine last and the door closed immediately, children should be kept away when detergent is added
- When emptying dishwashers check for, and remove, leftover powder or liquid
- Choose a dishwasher with a child resistant lock or purchase an adhesive lock to prevent access to the dishwasher by toddlers
- Store petrol in a child resistant container
- If possible, purchase diluted herbicides

As new products are manufactured, packaged and purchased, further hazards in the home will emerge. Identification and prevention of injury to others requires notification of these events to the New Zealand National Poisons Centre and the Ministry of Consumer Affairs, as well as a Medical Officer of Health.

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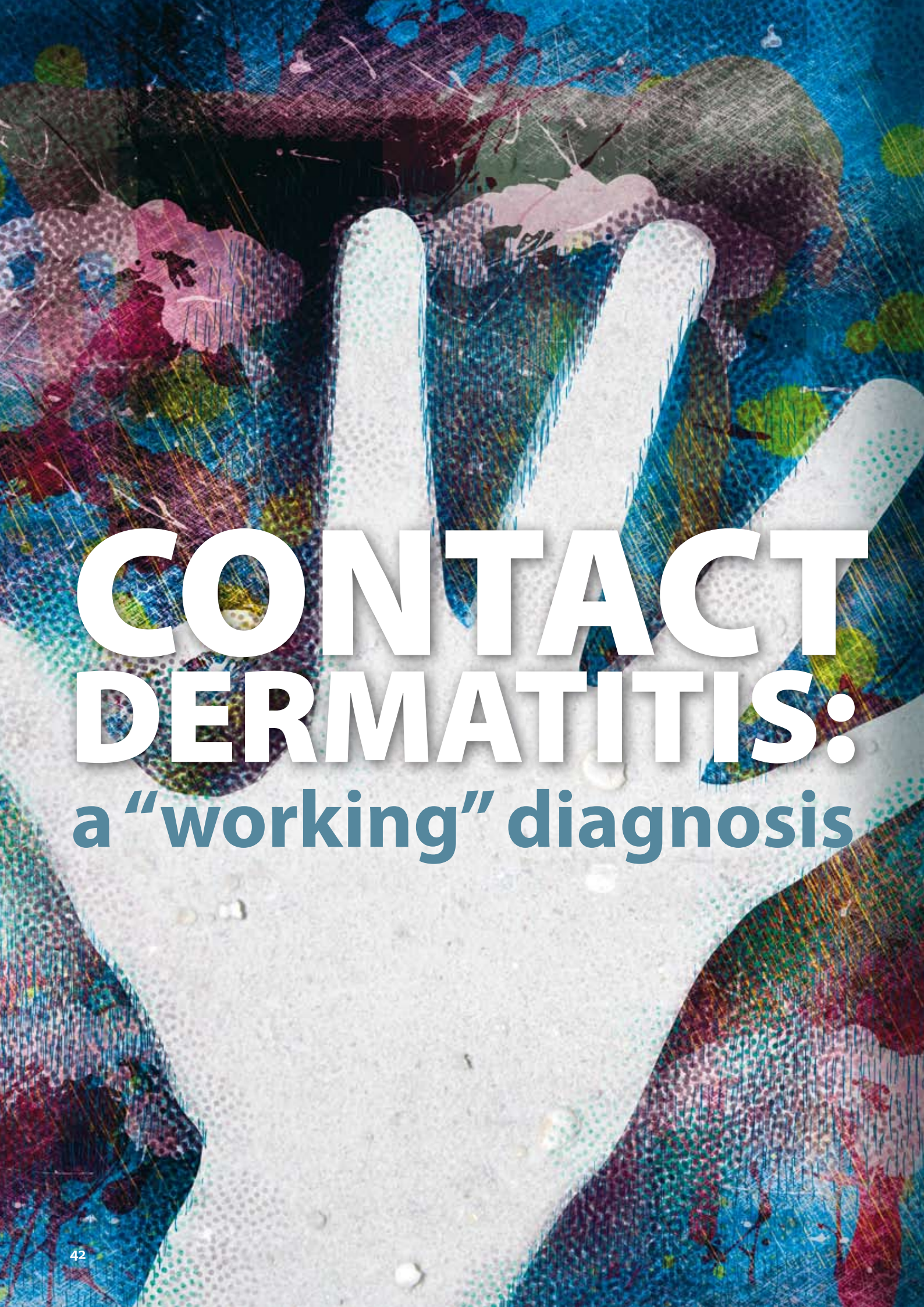

CONTACT DERMATITIS:
a "working" diagnosis

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Up to 20% of the general population suffer from contact allergy,[1] and it is estimated that there are 5 – 19 cases of occupational contact dermatitis per 10 000 full time workers per year.[2]

People working in the following industries are most affected by occupational dermatitis:[3]

- Food handler/chef
- Hairdresser/beautician
- Medical/dental/nurse/vet
- Agriculture/florist/gardener
- Cleaning/laundry
- Painting
- Mechanical/engineer
- Printing/lithography
- Construction

Clinical features of contact dermatitis

Contact dermatitis encompasses:

- Contact irritant dermatitis
- Contact allergic dermatitis
- Contact urticaria
- Photocontact dermatitis
- Systemic contact dermatitis.

Different forms of dermatitis may co-exist, e.g. an individual may have atopic dermatitis, contact irritant dermatitis and contact urticaria. In general, morphology does not differentiate contact from endogenous dermatitis; the diagnosis is suggested by the distribution, severity, temporal association with certain activities and allergy testing as appropriate.

Contact irritant dermatitis can be subdivided into subjective irritancy (stinging within minutes of contact, without objective findings), acute contact irritant dermatitis (a chemical burn) and chronic contact irritant dermatitis (when physical or chemical damage overwhelms the skin's repair mechanisms). Irritants include over- and under-hydration, soaps and detergents, solvents, abrasives, acids and alkalis. The likelihood that contact irritant dermatitis will develop depends on the potency of the irritant(s), occlusion, temperature, anatomical site and innate susceptibility; anything which impairs the skin's barrier function will potentiate the damaging effects of exposure to irritants. Contact irritant dermatitis is normally the cumulative effect of multiple irritants, and most commonly it affects the hands.

Contact allergic dermatitis affects only a small percentage of individuals exposed to an allergen. Many years of uneventful exposure may precede sensitisation, but once sensitised even tiny exposures can induce dermatitis. A cell-mediated immune reaction results in dermatitis one to four days after contact with the allergen. Contact allergic dermatitis most commonly affects the hands and face, but may also involve sites of secondary contact where small amounts of allergen have been transferred accidentally by contaminated fingers. Although there are thousands of potential allergens, a relatively small number account for the majority of cases of contact allergic dermatitis. Common allergens include rubber additives, chromate, epoxies, nickel, hair dyes, fragrances, biocides and plant derivatives including colophony (resin).

Contact urticaria may be IgE-mediated, or (more commonly) may occur through non-immunological mechanisms. It results in immediate itching, welts or aggravation of eczema at the site of exposure, and occasionally generalised urticaria (in the case of immune-mediated contact urticaria). It is most commonly caused by raw meat, fish or vegetables in food handlers, fish processors and abattoir workers; it can also be caused by rubber latex.

Photocontact dermatitis affects sun-exposed sites when a chemical in contact with the skin is altered by ultraviolet to produce either a photoallergen (causing dermatitis through immunologic mechanisms) or a phototoxin (causing dermatitis through non immunologic mechanisms). In New Zealand most photoallergic contact dermatitis is due to sunscreen chemicals, and most phototoxic reactions are due to furocoumarins in plants such as parsnip and celery.

Systemic contact dermatitis occurs when a person with a contact allergy to a substance (usually a medicine) is exposed to that substance systemically.

Investigation of contact dermatitis

Contact irritant dermatitis is diagnosed based on the patients history: the affected sites are exposed to irritants with sufficient frequency, duration or concentration to be a plausible cause of the dermatitis; the dermatitis improves or resolves following reduction or cessation of the irritant exposure; and there are no alternative explanations that might better account for the signs and symptoms.

Contact allergic dermatitis is diagnosed by patch testing: haptens are applied under occlusion to intact skin for up to 48 hours, and then the sites are checked for signs of reaction (erythema, papules, and vesicles). The sites are checked again on day four, and ideally again on day six or seven. The tests include a standard series of haptens (which is designed to pick up approximately 80% of the relevant positive reactions in that country), and any additional haptens as determined by the patient's history of exposure. Photopatch testing for the diagnosis of allergic photocontact dermatitis is the same, except the haptens are photoexposed on day two.

Contact urticaria is diagnosed by scratch-patch testing (test substances are applied over a superficial scratch, occluded, and left for 20 minutes), or occasionally prick tests or RAST tests.

A recent editorial in Archives of Dermatology commented that "most dermatologists use patch testing infrequently, and a significant minority of dermatologists do not patch test at all."[4] Of those that do patch testing, many limit their test to a routine screen, which adequately evaluates only 15.7% of patients with contact allergy.[4] Any patient with persistent dermatitis, which requires aggressive treatment for its control, should be considered for patch testing. The 2008 guidelines prepared for the British Association of Dermatologists suggest that the rate of patch testing should be around 143 patients per 100 000 population per year.[5] This would be equivalent to testing 600 – 700 individuals in the Wellington region per year, however, the actual amount of patch testing carried out is far lower than this. The scarcity of facilities for patch testing, photopatch testing and scratch patch testing is a major impediment to the adequate investigation (and therefore management) of contact dermatitis.

Management of contact dermatitis

Anti-inflammatory creams or systemic agents (the choice of which depends on the anatomical site, extent and severity of the dermatitis) form the basis of treatment for contact dermatitis, however, there are specific recommendations for irritant and allergic forms of contact dermatitis.

Contact irritant dermatitis can be prevented and managed by reduced exposure to irritants and the use of moisturising creams. While this sounds simple enough, in practice this is a complex area. Wearing gloves for prolonged periods may prove to be more irritating than the exposure the person was trying to avoid by wearing gloves. There is a paucity of data on barrier creams and moisturisers, particularly in respect of their benefit in the management or prevention of dermatitis in specific occupations.

Contact allergic dermatitis management usually requires complete avoidance of the relevant allergen(s), since even tiny exposures may cause a flare. Determining the relevance of positive reactions on the allergy test, and counselling the patient, are not always straightforward tasks. The patient needs to be educated regarding the substances which need to be avoided in a way which is comprehensive enough to avoid accidental exposure to the allergen(s) in future, but simple and concise enough that the patient is not confused and overwhelmed. The difficulty is that some chemicals have multiple names. For example, the sunscreen filter 2-hydroxy-4-methoxy benzophenone is also called Oxybenzone, Benzophenone 3, Eusolex 4360 and Escalol 567. A patient with an allergy to amine hair dyes might unwittingly use a "natural" hair dye, or they may think that black henna is safe, without reading the small print to discover that the product contains small amounts of p-phenylenediamine to boost the colour. The person who reacted to colophony used as a soldering flux needs to know that they may react to pine wood, the waterproofing agent on cardboard boxes, some adhesives, and so on.

Implications for work

While short periods away from work may be necessary for people with occupational contact dermatitis, recommendations to change career should not be given lightly. Most workers with contact dermatitis can continue in their jobs with appropriate treatment and work modifications; people who are atopic may still have symptoms, whether they stay or leave their jobs.

Notifying the Medical Officer of Health

Many medical practitioners are unaware that disease and injury caused by exposure to hazardous substances requires notification to the local Medical Officer of Health. This includes skin disease. A hazardous substance is defined as anything that can explode, catch fire, oxidise, corrode or be toxic to humans (Hazardous Substances and New Organisms Act 1996). To notify a case, a short electronic notification form is located on the *bestpractice* dashboard (log in at **www.bestpractice.org.nz** or go directly through MedTech) – look for "Hazardous Substances & Lead Notifications". Primary care practices that do not use *bestpractice* Decision Support software should still inform their Public Health Unit of any notifications.

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Case study: a surgeon with contact dermatitis

A 55-year-old surgeon, with a history of atopic eczema since childhood, had suffered from severe hand dermatitis for the last six months – it was seriously impairing his ability to work, despite treatment with potent steroid creams and systemic steroids (which only controlled it briefly). In his occupation he is at risk of contact irritant dermatitis on account of frequent hand washing and prolonged glove wearing, however, patch testing demonstrated that he was also allergic to six of the nine brands of glove available in his workplace (four of which produced very vigorous reactions), and two of the three surgical scrubs that were tested. Following patch testing we were able to give advice on appropriate gloves and scrubs which allowed him to continue his normal work.