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This Issue in the Journal

Original articles

Fatal dog bites in New Zealand

David Healey

Dog attacks represent a significant health hazard in New Zealand. Dog bites, although common, rarely result in death however. There has been an increase in the reporting of bites in the last 10 years; males, children, and Māori are over-represented in the statistics. Dog bites occur mostly to the head and face (especially in young children) and to the arms (especially in adults) and legs. Most dogs responsible for severe bites against people are un-neutered males and from relatively few of the 160 known breeds. Five deaths due to dog bites are known in New Zealand, and one of these is documented in this article. An increased awareness among the general public of the factors behind dog attacks may reduce their incidence.

Murine typhus: a newly recognised problem in the Waikato region of New Zealand

Erana Gray, Polly Atatoa-Carr, Anita Bell, Sally Roberts, Daud Al Mudallal, Graham D Mills

Rickettsial illnesses are common in the rest of the world, but have only been seen in New Zealand over the past 20 years and only around Auckland and the Coromandel Peninsula. Now we know that the illness is also in rural Waikato, it may be in other areas around the country too, but we need to look harder to try to find out. It is strongly rural in location. It is caused by a small bacteria, via fleas carried on our rats (in much the same sort of cycle as the plague is!). Increased awareness of this illness will lead to earlier diagnosis and treatment—which is cheap, easy, and effective.

Routine 'Health of the Nation Outcome Scales for elderly people' (HoNOS65+) collection in an acute psychogeriatric inpatient unit in New Zealand

Gary Cheung, John Strachan

HoNOS (Health of the National Outcomes Scale) is an outcome measure used by mental health professionals aiming to give a snapshot of a person's psychiatric symptoms and psychosocial functioning. An improvement in the person's mental well-being (e.g. through treatment) can be reflected by a reduction in the HoNOS score. This paper describes how these routinely collected data can be used to inform a psychogeriatric inpatient service regarding service users' case-mix and service development.

Heracleum mantegazzianum and *Toxicodendron succedaneum:* plants of human health significance in New Zealand and the National Pest Plant Accord

José G B Derraik

New Zealand's National Pest Plant Accord (NPPA) is a voluntary and cooperative agreement between industry, regional councils, and central government departments with biosecurity responsibilities. Plant species included in the NPPA are declared unwanted organisms under the Biosecurity Act 1993, which prevents their sale, propagation, or distribution across the country. Several introduced plants species in New Zealand can cause adverse reactions in humans via skin contact.

Two introduced plant species, in particular, were evaluated for the NPPA as a result of their public health significance and the possible need to officially control them: *Heracleum mantegazzianum* (giant hogweed, cow parsnip, wild parsnip) and *Toxicodendron succedaneum* (rhus tree, wax tree, Japanese wax tree). Their adverse effects to human health have been widely documented, and in this article, the relevance of these two species to human health are discussed, including symptoms and treatment, as well as the final decisions of the NPPA process and some possible management measures.

Special Article

Rural hospital medicine in New Zealand: vocational registration and the recognition of a new scope of practice

Garry Nixon, Katharina Blattner, for the NZ Rural Hospital Doctors Working Party

This article outlines a proposal currently with the NZ Medical Council to recognise rural hospital medicine as a distinct scope of practice with its own vocational registration and training programme. This will bring generalist doctors working in NZ's rural hospitals into line with those working in other branches of medicine. These developments have the potential to greatly improve the professional environment for these doctors, helping ensure standards of practice, provide appropriate training, and resolve many of the workforce issues.

Review Article

New hospital consultant: surviving a difficult period

Geoffrey Robinson, Johan Morreau, Marion Leighton, Richard Beasley

The public, patients, employers, and the medical profession usually have scant regard for the functioning and health of doctors. We identify the beginning of specialist practice as a period of vulnerability with potential for stress and ill health. Only recently has attention been focussed on this issue and we have suggested guidelines for maintaining learning, enlisting support, and balancing clinical and professional practice with a sensible work/life balance. Problems with depression, drug abuse, and burn-out may well emerge if the junior specialist comes under major stress.

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Medical opportunity of special scale? The future of rural hospitals in New Zealand?

J Campbell Murdoch

The current issue of the *Journal* contains a report by Nixon et al[1] on the vocational registration and the recognition of a new scope of practice in rural hospital medicine in New Zealand. These initiatives are claimed to represent an opportunity to generate the skilled generalist medical workforce which New Zealand rural hospitals need to move into the future.

There are about 120 rural hospital doctors working in 36 rural hospitals in New Zealand. Defined as "facilities with no resident specialists where acutely ill patients are admitted and cared for solely by generalist doctors," in 1998, these contained 293 acute beds and served a population of about 340,000.

Apart from a small number of specialists, surgeons, physicians, and emergency physicians, the doctors are in two broad categories—50% are rural General Practitioners (GPs) with part-time appointments, and 50% are doctors with the intriguing title of Medical Officers of Special Scale (MOSSs) who work under general registration.

The MOSSs contribute 80% of the total number of medical hours worked in New Zealand's rural hospitals. The MOSSs appear to be "rolling stones"—a third of these positions are currently vacant and more than 50% leave their positions within 2 years.

While there is no doubt that recruitment and retention of the staffing of these hospitals would be greatly improved by these measures, perhaps a more urgent priority in New Zealand is to define the place of the rural hospital in rural medicine as a whole—and then provide solutions to the education, training, and credentialling of the medical workforce.

The document states that rural hospitals require "generalist doctors to work in relative isolation far from complex diagnostic and specialist backup and across a broad generalist scope of practice"—but this is also a good description of the rural general practitioner and would be an appropriate outcome for all those in rural medical training in New Zealand.

In Australia, rural and remote medicine is an academic discipline which is taught and researched at a university level. The Australian College of Rural and Remote Medicine (ACRRM) defines its discipline as

> …the body of scientific knowledge underpinning clinical practice and medical service delivery in the rural and remote context. Its aim is to achieve the best possible outcomes in health care. Cross-disciplinary investigation is implicit to the practice of Rural and Remote Medicine. It presumes an interdependent model of medical service that **combines high level competency in primary, secondary and sometimes even tertiary medical care** and forms a distinct scope and method of practice.[2]

The Australians therefore define rural medical generalism as the practice of those medical competencies which are required by rural populations and not what has been

ordained by specialties who establish their norms by what happens in areas of large populations. It was this difference of opinion on the scope of rural and remote medicine which led to the formation of a separate College.

The New Zealand document wishes to avoid this type of division and it is to the great credit of the Royal New Zealand College of General Practitioners (RNZCGP) that it is prepared to welcome the "two variations on a theme" within its ranks, but hopefully not at the cost of failing to address the question of the place of rural and remote medicine.

A recent report,[3] while drawing attention to the importance of the rural sector in New Zealand, has commented that "the defining difference between New Zealand and other countries with large rural regions and a largely publicly-funded health and disability sector is that New Zealand has not examined its local context nearly as thoroughly as other jurisdictions."

Rural hospitals certainly do not seem to be there as the result of careful planning. Another study [4] has shown that there are 167,295 people in New Zealand that have to travel more than 1 hour to visit a basic public hospital and many of these people are located in the southern and northern regions of New Zealand.

The authors suggested that these results "may lead to new hospitals being developed or the upgrading of medical centres to hospital status in areas where access is currently poor." Such a network would be invaluable in developing the discipline of rural and remote medicine, and it would appear that it could contribute to finding a new role for rural hospitals in three main ways.

The first is in a more structured delivery of complex medical care. A recent study of rural and remote medical practice in Australia[5] found that the proportion of GPs providing complex services increases with increasing rurality or remoteness.

For instance, isolated rural and remote GPs managed myocardial infarctions to a higher level than GPs in larger rural and regional centres—and were more likely to administer cytotoxic drugs, perform forensic examinations, stabilise injured patients pending retrieval, and coordinate discharge planning.

In almost every area of medical care such as cancer care[6] and heart failure[7] there is evidence that access to specialist care, investigation, and management is a problem for country people.

A network of rural hospitals or regional resource centres within comfortable reach of rural and remote populations would seem to be key in solving the problems of access to complex medical care.

Moreover a network of rural hospitals might assist the delivery of after-hours care in rural New Zealand. Often it has been noted that a major problem in attracting doctors to rural New Zealand is the fact that 79% of rural GPs participate in after-hours calls compared to 28% of rural primary health care nurses, and 35% of rural pharmacists.[8]

Another study has shown that the percentage of GPs undertaking after-hours calls was much lower in Auckland (40%) than in the cities (84%) and towns (87%), while it was highest in rural areas (97%).[9] A network of rural hospitals staffed round the clock might assist in solving this crisis.

Finally these hospitals might contribute to the promotion of a network of academic rural centres of excellence. In Canada, these have formed the basis of a distributed model of medical education, bringing students in the areas of greatest medical need.[10]

In Australia, the rural health strategy has influenced undergraduate medical education by various means, including the setting up of University Departments of Rural Health (UDRH) and Rural Clinical Schools. The contracts for the latter have been tied to parameters involving educating at least 25% of all medical students for a minimum of 1 year in rural and remote locations, spending resources in these areas, and employing academics who must "live in the bush."

Preliminary results have shown that these centres are capable of delivering the undergraduate curriculum to an equal, if not superior, standard.[11] This network would also be able to fill the research void noted in the recent report.

The problems of rural health are directly related to access, and good access relates to structure and planning. Indeed, rural areas seem always to lie at the end of a long and tortuous pipeline as far as health care delivery and recruitment of health care professionals is concerned.

A recent Scottish report[12] has supported the concept of an extended primary care role through the community hospital; it stated that this role should be well-suited to all parts of Scotland, not just rural areas where the existing hospitals have developed.

Perhaps this is an idea whose time has come in New Zealand.

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Fatal dog bites in New Zealand

David Healey

Abstract

Aims To examine fatalities from dog bites in New Zealand and review New Zealand's most recent fatalities, and the commonalities they show with dog attacks both in New Zealand and overseas.

Methods Information was collated from past studies, international reviews, and press coverage and then summarised. This information was then applied and added to a forensic review of the dog bite-related fatality recorded in 2004 in Dunedin.

Conclusions Dogs are no different the world over. There have been five confirmed dog bite-related fatalities in New Zealand. Dog attacks in New Zealand strongly resemble those from other developed countries. Indeed similar situations increase the odds of an attack. Awareness of these situations may make avoidance of dog bites easier.

Dog bites in New Zealand

Dog attacks represent a significant health hazard in New Zealand. Dog bites, although common, rarely result in death however. While there has been an increase in the reporting of bites in the last 10 years,[1,2] it is difficult to determine if this is real, or just an artefact of changed coding and reporting mechanisms.

New Zealanders like those in many other developed countries have a high frequency of animal ownership. It is difficult to get exact details on dog ownership, however it is estimated that there are over 600,000 dogs in New Zealand; 27% of dog owners have two or more dogs.[3]

Details for dog attacks in New Zealand have improved significantly with computerisation of hospital records. Prior to 1960, records are somewhat unreliable. There have been two major studies in New Zealand examining the incidence of dog bites. Unfortunately a lack of consistency with the labelling and coding of dog-related injuries meant that the records are only superficially comparable.

Langley (1992) gathered data from the Health Statistics Services injury mortality data files during the period 1979 to 1988.[4] Codes including the word 'dog' and 'bite' were used. These recorded all bites, but sometimes did not distinguish dog bites *per se*. Other injuries from dogs such as being 'struck' were included. Langley reported an incidence rate of 4.8 hospitalisations per 100,000 in 1988 and noted that the increased incidence rate if the trend remained would reach 9.6 per 100,000 by 2000.

Marsh et al (2004) sought to update the earlier work by using a similar method to examine bites during the period 1989 to 2001.[1] They similarly identified suitable cases through an examination of Electronic Mortality and Morbidity Files in the New Zealand Health Information Services (NZHIS) Database. They also catalogued an increasing incidence rate.

Between 1989 and 2001 there were 3119 hospitalisations and 1 fatality; 3025 hospitalisations were estimated to have resulted from dog bites—94 of these were estimated to have been from being 'struck' by a dog.

New Zealand Health Information Statistics recorded 309 overnight hospital visits after dog bite incidents in 2000, 293 in 2001, and 324 in 2002. Incidence rate figures peaked in 1996 at 7.5 per 100,000. Given a population of 3.9 million in 2002, the incidence rate for dog bite incidents was 8.3 per 100,000.

These figures appear to represent an escalating trend in admission statistics similar to that observed in other countries.[2]

Who gets bitten

In New Zealand, children aged 4 years and under accounted for nearly 24% of the cases of hospitalisation for dog bite injuries.[1] High rates among children can probably be explained by their lack of physical strength or motor skills to ward off an attacking dog.

Immaturity and lack of judgment may also sometimes lead children to act in ways that animals perceive as threatening or aggressive. Specifically, they maintain eye contact, and their eye level is often the same as that of a dog. Furthermore, it has been suggested that (prior to their injury) children under 5 years of age are significantly more likely to provoke animals than older children.

When the 0–4 and 5–9 year age groups were combined, children under the age of 10 years received 39% of dog bite injuries. Those aged between 25 and 39 received 18% (n=423) of dog bite injuries.[16]

When the incidence rates were broken down by ethnicity, it was found that New Zealand Europeans represented 52% of the total bite victims, Māori 28%, and others 20%.[1] For the period 1979 to 1988 the Māori inpatient rate was 1.8 times the non-Māori rate. Furthermore, from 1988 to 2002 the Māori outpatient rate was 2.6 times that of non-Māori.

The age-adjusted incidence rates for Māori and non-Māori were 10.6 per 100,000 population (9.4–11.7) and 5.9 (5.6–6.3) respectively. Māori were therefore over-represented within the bite statistics. These rates may be a real change or be an artefact of differences in ethnicity classification.

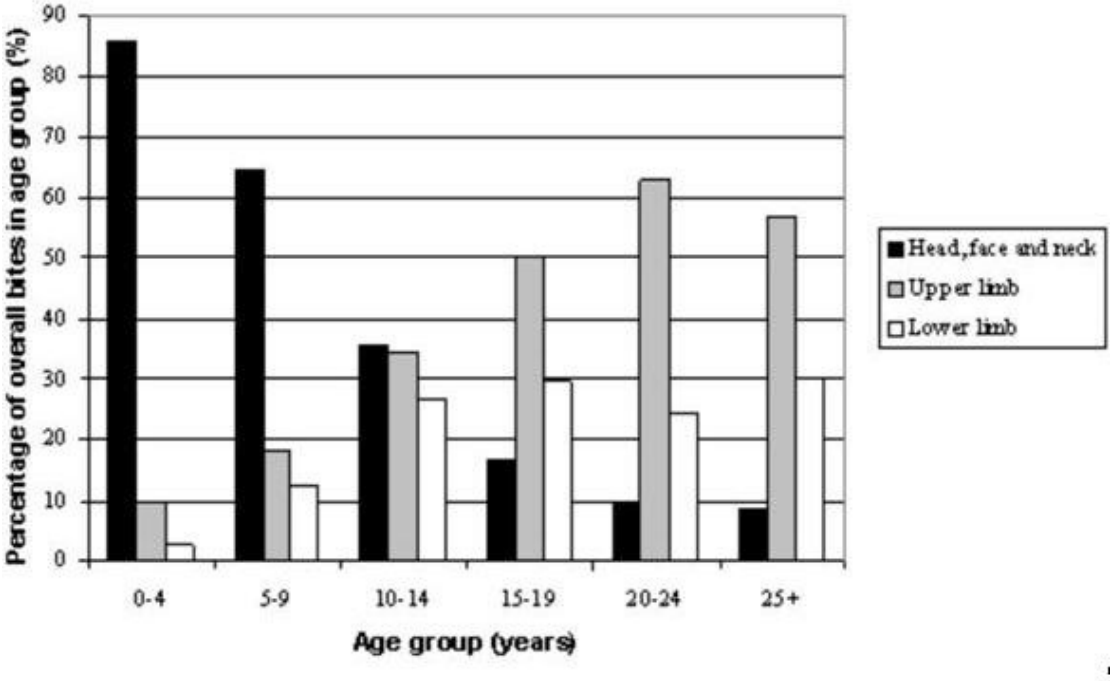
Thirty percent of victims were bitten at home with 6% occurring on the street and 1% on a farm; 60.5% of bites were to males.

Site of the bite

Upper limb, head, and lower limb were the most common regions to be injured—the most common site of injury is the face. There is strong evidence of a difference in the distribution of injury location by age group (Figure 1). Injuries to the head were significantly more common for the younger age groups.[8]

Injuries to the upper limb most commonly occurred in those aged over 15 years—these represent defensive wounds. Lower limb injuries were more consistently spread through the age groups. Males, and children less than 9 years of age (Figure 2), had the highest rates of injury.

Figure 1. Site of dog bites by age group (Reproduced from Marsh et al 2004 with permission)



In cases where a location was noted (42% of cases), 30% of the victims were bitten while at a home (not necessarily their own). For 6% of the victims, the bite occurred on the street or highway, and 1% of bites occurred on a farm. Chained dogs were 2.8 times more likely to bite than un-chained dogs.[1]

Breed-specific factors

Although often sited as a factor in attacks, breed-specific factors need to be treated with some caution. Not all dogs are purebred and identification in crossbreed cases can be problematic. In addition, the frequency of breed distribution is not even—more bites might be attributed to German shepherds than Alaskan malamutes, but the population of German shepherds is much higher, thus frequency representation is important to note.

Humane Society US statistics[5] reflect that dogs that have not been spayed or neutered are up to three times more likely to be involved in a biting incident than neutered or spayed dogs. Male dogs are involved in 80% of all bites.

In those cases where breed-specific information was available, some breeds appear to be disproportionately represented.[6]

In the United States, three-quarters of all hospital-treated dog attacks were caused by just 5 of the 160 or so known dog breeds:[9]

- Pit bull terrier.
- Rottweiler.
- German shepherd.
- Husky.
- Alaskan malamute.

Breed-specific factors contributed to the definitions in the Dog Control Amendment Act (2003, 2004, 2006) in New Zealand. Among other controls, the Act required all newly registered and dangerous dogs to be microchipped by July 2006, and banned the import of the American pit bull terrier, Brazilian fila, Japanese tosa, and dogo argentino.

Targeting specific breeds has not met with success in other countries—e.g. the UK control of pit bull terriers in 1991. Instead, a more effective approach may be to target chronically irresponsible dog owners.[7]

International comparisons

The comparison of international incidence rates is problematic for hospitalisations due to differing definitions. However New Zealand appears to compare favourably with other countries. Our incidence rate of around 8.3 per 100,000 population was similar to that observed in Australia of 7.7 for 1995 to 1996.[2]

For the period 1992 to 1994, the United States recorded a dog bite-related injury visit to hospital of 12.9 per 10,000 (over 10 times the New Zealand rate).[8] During the period (1979–1988) the United States recorded 157 fatalities from dog bites; the average number of deaths per year was 17.[9]

There were almost 4.7 million victims annually—about 1.8% of the entire population, but only 0.3% sought medical attention.[13] It has been estimated that, in the United States, dog bites cause 585,000 injuries necessitating medical attention yearly.[10]

Children aged 12 years and under made up approximately 51% of victims. Children aged 5–9 years were bitten most often, with 78% of the bites located on the extremities.[11] United States statistics suggest that almost half of all children will be bitten;[12] and most of these bites will remain unreported.

The United States has 73 million licensed dogs and 39% of United States households own at least one dog; 25% of owners own two dogs. More than 70% of owned dogs are spayed or neutered.[5]

The data indicate that rottweilers and pit bull-type dogs accounted for 67% of human dog bite-related fatalities in the United States between 1997 and 1998. It is unlikely that they accounted for anywhere near 60% of dogs in the United States during that same period and, thus, there appears to be a breed-specific problem with fatalities in that country.[6]

The social cost

Apart from the obvious psychological cost to individuals and families from dog bites, society also bears a social cost in lost productivity and use of hospital resources. The impact of these together is difficult to estimate. However, hospital records and Accident Compensation Commission (ACC) records can quantify some of these aspects.

In a 2003 report, New Zealand's 58 Councils reported a total of 3020 dog attacks on people in 2001/2. Extrapolating this figure gives a nationwide estimate of 3435 dog attacks in 2001/2; this is still well below ACC estimates.[14]

Males made up 60.5% of the hospitalisations from dog bites. In 2002/3, there were 257 new entitlement claims lodged with ACC and 55 ongoing claims, which amounted to NZ$1,041,099, and 7790 medical fee claims totalling $687,188—a substantial increase over the figures from 1999 (Figure 2).

Victims with injuries to the lower limb were more likely to stay in hospital the longest, with a mean number of 5.6 days.

Figure 2. ACC entitlement claims (in New Zealand dollars). In other words, these are moderate to serious claims that could have resulted in a week or more off work or required more support from ACC than medical treatment such as a visit or two to the doctor[14]

Dogbite Entitlement Claims		New		Ongoing	
		Number	Amount ($000)	Number	Amount ($000)
Total	1999-07/2000-06	105	174	9	79
	2000-07/2001-06	156	304	19	95
	2001-07/2002-06	185	451	24	173
	2002-07/2003-06	216	481	27	209
	2003-07/2004-06	230	687	35	333
	2004-07/2004-08	35	55	38	121

Dog bite-related fatalities in New Zealand

To date, New Zealand appears to have suffered five dog bite-related fatalities. Although records are not complete, it appears that there are no recordings of traumatic fatalities prior to 1969.

In 1969, a farmer was killed by a mixed-breed farm dog. There are few details available about this incident.

In April 1997, a 59-year-old Te Puke man was killed by two bull-terrier cross dogs. A veterinary postmortem of the killer dogs confirmed the breed and it found they were approximately 2 to 3 years old. The dogs had been trained to hunt pigs, but they turned on their owner when he tried to stop them from fighting.

In February 2003, a 73-year-old Northland woman died after being bitten on the foot by one of her three pure-breed Alaskan malamutes. It appears that the bite had hit an artery and she had exsanguinated without moving from her chair.

The fourth case, from 2004, is the subject of the second part of this article.

A fifth case occurred in April 2007 when a 56-year-old Murupara woman was attacked and killed by two dogs: a pit bull terrier and a Staffordshire cross. She suffered multiple bites and lacerations to both lower legs in the attack. When found, she had lost a considerable amount of blood. She died in the ambulance on the way to the hospital. The cause of death was shock and trauma. The attack took place early in the morning on a suburban street.

A dog bite victim

In 2004, a 39-year-old Dunedin woman was savaged to death by her pet bull mastiff dog: an un-neutered fully grown male, weighing 55 kg. The victim was a slightly built (46 kg). Her injuries included extensive bites to both right and left arms, originating above the elbow. The majority of these were canine puncture wounds, but on her left arm there was one incised and torn crescent shaped wound around 7 cm long.

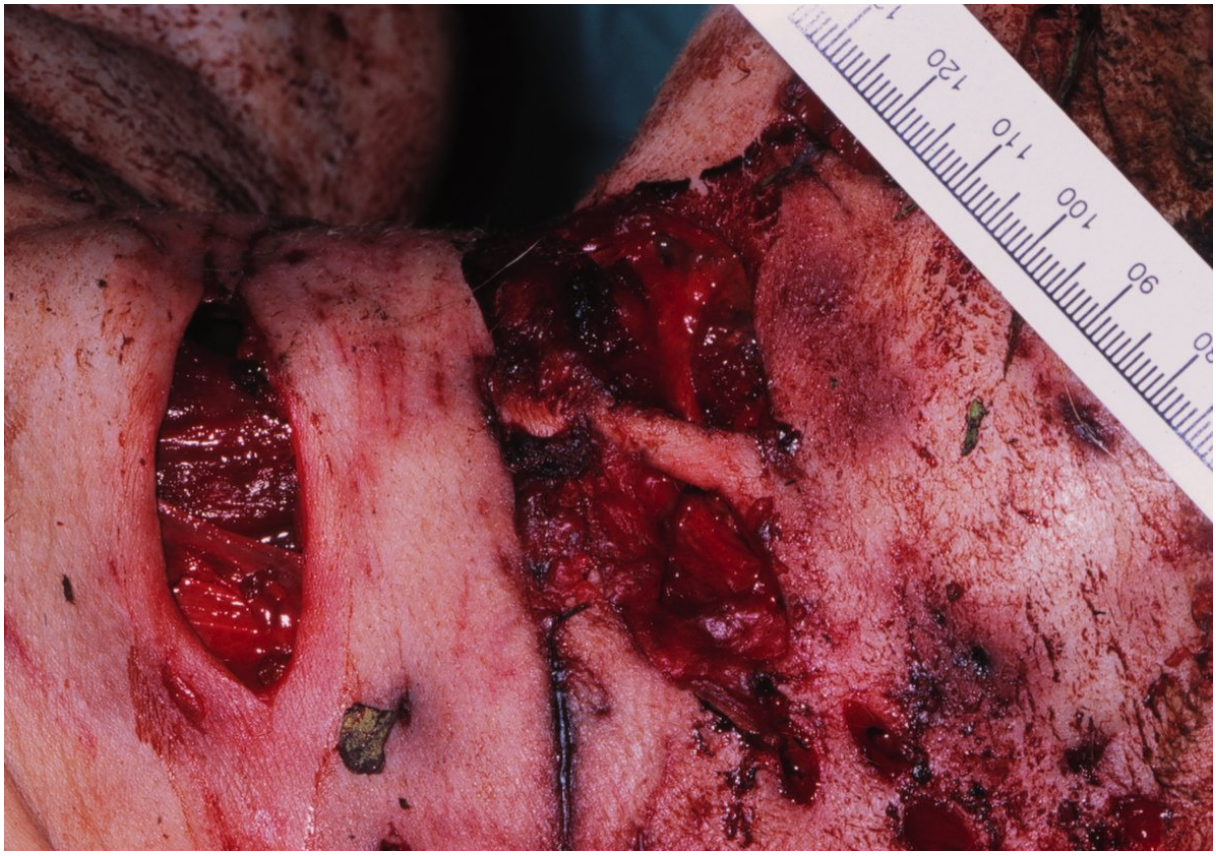
The majority of the serious wounds were found on the victim's face, neck, and skull. A large 8 mm-wide gash ran across bridge of her nose extending from 1 cm below the right inner canthus and terminating above her left infraorbital foramen. She had a 55 mm gash inferior to a 20 mm gash on her right neck. Her left ear and soft tissue covering the back of the skull were missing and had possibly been ingested by the dog.

A 25 mm tear ran medially over the right temporalis above the eyebrow, and a 35 mm gaping tear midway between the brow and the hairline extended from above the outer canthus of the eye to the midline. Her lower face was untouched.

The most serious wounds were to the back and left side of the neck. The back of the neck exhibited a large gaping wound 70 cm long just below the hairline, whilst the left side of the neck had a 55 mm-long tear running medially at the level of the thyroid cartilage. Superior to this were two ragged edged tears of 30 mm with a isthmus of tissue separating them. Each of these tears terminated over the region of the hyoid.

The cause of death was most likely exsanguination and asphyxia. A canine puncture wound was found in the trachea on her left side, coincident with the most caudal of the three incisions found in that region. Bleeding into the trachea had occurred. Numerous canine drag marks were visible over the skull, upper face, and arms.

Figure 3. The victim's neck injuries showing the tear at the level of the hyoid cartilage and the tear over the hyoid (with tissue isthmus)



There were what appeared to be healed bite marks and bruises on her lower legs; there appeared to be no fresh bite marks in these areas. An examination of the dog's bite radius and the healed marks showed a positive match.

The victim lived alone. An animal control team had been called to the property several times in the past, twice for barking, once for straying, and once for the dog's aggressive behaviour towards the victim, however she had withdrawn consent for it to be taken to the pound. The dog was destroyed by police to allow access to the scene.

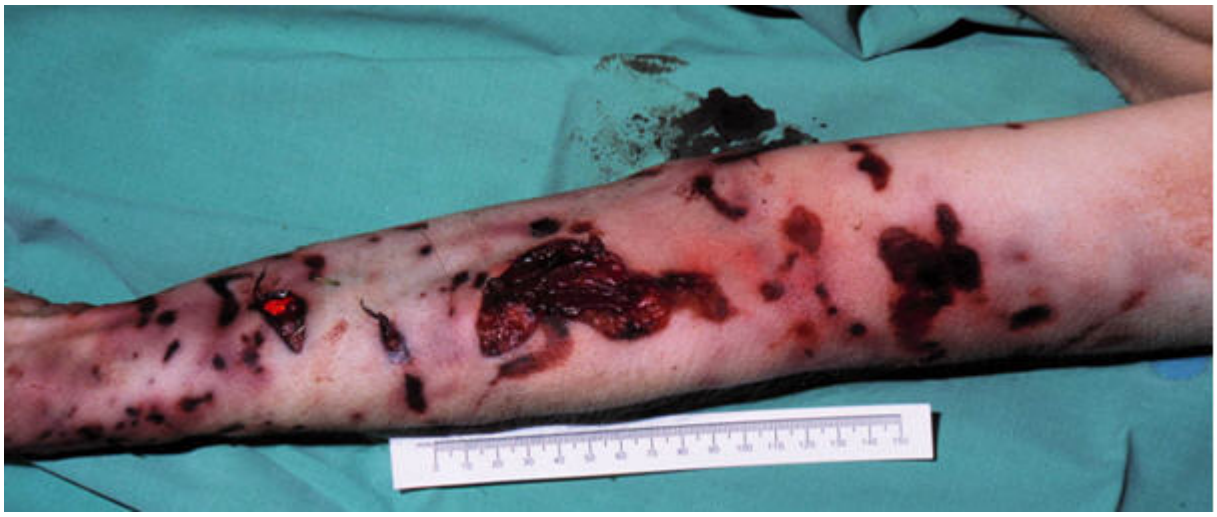
The victim's health status may have produced mitigating circumstances possibly provoking the dog to attack. She was suffering from Huntington disease (HD), also known as Huntington chorea (HC). This inherited (autosomal dominant with complete penetrance) disease is characterised by choreiform involuntary movements and slowly progressive dementia.

Between one-half and three-quarters of patients affected by HD present with primary complaints of rigidity or involuntary movements. In the rest of the cases, the presentation can be one of early mental status changes that appear as increased irritability, moodiness, or antisocial behaviour.

Patients can become argumentative, erratic, emotionally volatile, even physically aggressive. The classic choreiform movements begin as a piano-playing motion of the

fingers or as facial grimaces. As the disorder advances, a characteristic dancing gait evolves. The disease is slowly but inexorably progressive.[16]

Figure 4. The victim's left arm showing healing and fresh bite marks



Discussion

It has been suggested that the victim may have mistreated the dog as she was known to have exhibited some of the mental characteristics typical of HD. Indeed, lack of coordinated movements, erratic behaviour, or even aggression toward the dog may have seen changes in their relationship.

Coupled with a large, powerful un-neutered male dog in a small enclosed area, this factor may have tipped the balance. This history together with the presence of healed bites suggests that the victim suffered a recent spate of aggressive incidents with the dog and it may have been attempting to assert its dominance over her.

It has been acknowledged that dogs showing dominant aggression can respond to anxiety as well as to a perceived challenge to their rank.[17,18] Although entirely speculative, the victim's health may have contributed to, or even precipitated, the attack in this case.

Clearly the incidence of both fatal and nonfatal dog bite attacks has increased in New Zealand over the past 10 years. This trend is in-line with overseas findings, however, we still have a long way to go before experiencing the almost epidemic levels seen in the United States in recent years.

Many dog bites, both nationally and internationally, show common features so awareness of these may help individuals to avoid situations where they or their family members could be at risk of injury.

Conflict of interest: None.

Ethics statement: In 2004, this tragic case generated a large amount of press coverage. The woman's cause of death as well as her medical state were recorded in the coroner's report that is now available as a public record.

At the time of the incident, discussions were held with the family and Coroner, and out of consideration for the family this article was held back for 2 years (3 years have now passed).

My role in this case (as with other dog attacks) was as a forensic odontologist called by the police to make a positive identification of the individual and/or animal.

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Murine typhus: a newly recognised problem in the Waikato region of New Zealand

Erana Gray, Polly Atatoa-Carr, Anita Bell, Sally Roberts, Daud Al Mudallal, Graham D Mills

Abstract

Aim To characterise and investigate patients diagnosed with murine typhus in the Waikato District Health Board (DHB) region during 2006.

Method We reviewed the hospital and general practitioner records of all patients presenting with clinical and serological evidence of murine typhus. All patients were interviewed by telephone using a semi-structured questionnaire to identify environmental risk factors for infection. A limited, retrospective serosurvey was undertaken and surveillance was enhanced.

Results 12 patients were identified, all had either lived, or spent considerable time, in rural areas; 7 patients had seen rats on their properties 'regularly' and 3 remembered fleabites within the incubation period of the illness. The classic triad of symptoms is fever, headache, and rash—these symptoms were seen in 12, 11, and 8 patients respectively; lethargy, myalgias, nausea, and vomiting were also common. 11 patients had abnormal liver function tests at presentation, and 7 had low platelets. Treatment with doxycycline was associated with a shorter hospital stay.

Conclusion Murine typhus has now been confirmed in rural areas throughout the Waikato DHB region. Rats are likely associated with disease in rural communities and rat control is a complex issue. However, a greater awareness of the disease should lead to earlier diagnosis and treatment.

Murine typhus, also called endemic typhus, is caused by *Rickettsia typhi*—one of the rickettsial 'typhus' group. It is one of the many causes of undifferentiated fever in the adult.

R. typhi, an obligate intracellular bacteria, has worldwide distribution, including New Zealand.[1] The illness is characterised by fever, headache, and sometimes a rash. The first New Zealand-acquired murine typhus infection was reported in 1991.[1] Up until the end of 2005, there had been 22 patients with locally acquired disease occurring in the Northwest and South of Auckland and in the Coromandel Peninsula area (S Roberts, personal communication, 2006).

During 2006, the Population Health and Infectious Diseases Services at the Waikato District Health Board (DHB) became aware of an apparent increase in the number of patients being diagnosed with murine typhus. This not only included further patients from the Coromandel Peninsula area, but (for the first time) patients in other areas of Waikato DHB.

These patients were investigated—with a focus on patient demographics, clinical presentation, and laboratory findings. We sought to describe the environmental risk

factors associated with the disease and to identify other patients. Our aim was to find those features that would assist in the diagnosis of further patients.

Methods

All patients from the Waikato DHB (Figure 1) with serological evidence of *R. typhi* were identified from laboratory records. The case definition was a compatible clinical illness combined with serological evidence of infection. A case was 'confirmed' if there was a 4-fold rise in the titres of paired samples and a case was 'probable' if there was a single specimen with a high-titre IgM (1:512 or greater) or IgG (1:1024 or greater).

Patient serum was stored at 4°C for up to 5 days or frozen at -20°C, then tested using an indirect immunofluorescent antibody assay (Focus Diagnostic IFA IgG and IgM). Diluted serum was incubated on a *Rickettsia* substrate slide, using antigen spots for both the typhus and spotted fever groups, washed to remove unbound antibodies, and then re-incubated with fluorescein-bound anti-human IgG or IgM.

For the IgM assay, the diluent contained anti-human IgG in order to remove free and complexed IgG (such as rheumatoid factor) as a cause of false positives. Semi-quantitative endpoint titres were obtained by testing serial dilutions of positive specimens. Positive and negative controls were used.

We reviewed the hospital and general practitioner (GP) records of all the confirmed and probable cases. Data was collected on patient demographics, including rurality and area of residence—particularly if they lived in the Coromandel area or travelled there during the incubation period.

Also, clinical features and laboratory findings—signs and symptoms at presentation, duration of illness, time to considering rickettsial infection in the diagnosis, laboratory findings at presentation and during the illness, other investigations and treatments—were recorded.

Patients were interviewed by telephone using a semi-structured questionnaire to collect data on environmental risk factors—in particular bite recognition and identifying any contact with rats or fleas.

Enhanced surveillance was carried out both retrospectively and prospectively. Firstly, an update was placed in the DHB's Public Health Bulletin and all GPs were written to, asking for information regarding possible patients and for confirmatory *R. typhi* serology from any such patients.

Secondly, a limited sero-survey was undertaken. All previously stored sera from a patient group likely to yield 'missed' rickettsial diagnoses was tested for *R typhi* antibodies. We chose to test held sera from those who had had negative leptospirosis serology (a clinically similar illness) combined with abnormal liver function tests (seen in almost all murine typhus) between May and August 2006.

Results

Twelve adults (six males and six females) had consistent clinical illnesses and serological evidence of murine typhus. Six were defined as 'confirmed' and six as 'probable'. Patient #10 had undetectable antibodies on day 5 of symptoms, seroconverting with an IgM of 2048 by day 10. See Table 1.

Ten patients were identified through physician notifications and existing serology results. One further patient was diagnosed retrospectively by their GP following the Public Health Bulletin—and one patient (patient #11) was found during the limited sero-survey of 23 patients with negative leptospirosis serology and abnormal liver function tests. Patient #11's original sample was taken on Day 5 of his illness.

The median age (range) of the patients was 46 years (19 to 69 years) while the median duration (range) of symptoms before seeking medical attention was 5 days (3–14 days).

Of the nine patients admitted to hospital, the median time (range) taken—until a diagnosis of *R. typhi* was considered and serology ordered—was 4 days from admission (0–11 days), although this excludes one patient whose diagnosis was made more than 1 year after his illness (patient #5).

Figure 1. Map showing the northern part of the Waikato District Health Board region (shaded in yellow) of New Zealand, with patient locations marked in red

Note: Patient #9 was identified by his Coromandel bach (holiday house) location, rather than his main residence in Hamilton.

All 12 patients had abnormal liver function tests (LFTs), with these abnormalities seen at presentation in 11. They were a mixed picture with both transaminases and cholestatic enzymes elevated, with an initial ALT median (range) of 179 (25–419) U/L. Seven patients had platelets below 150×10^9 at admission; 21×10^9 was recorded as the lowest platelet count.

Appropriate antibiotics were only given to 7 patients as many of the others had fully recovered by the time the diagnosis was confirmed. For the majority of patients, murine typhus was not the working diagnosis and other diagnostic work-up was undertaken: 12 imaging studies (e.g. abdominal ultrasounds, CTs, ECHOs) were performed in 7 patients, along with 2 lumbar punctures.

All patients lived in rural areas, or had spent substantial time in rural areas. Three lived on lifestyle blocks, three lived and/or worked on farms, and five had houses in rural areas. The single patient that lived in an urban setting, spent every weekend at a bach (holiday house) in Coromandel, and had been underneath the dwelling (where rats were known to live) during the incubation period.

Table 1. Murine typhus patient demographics and serology results

Patient #	Age (years)	Gender	Hospital admission?	Located in rural area?	Main rural contact	Acute serology			Convalescent serology		
						Date	IgG	IgM	Date	IgG	IgM
1	36	M	Yes	Yes	Lifestyle block	25-Apr-06	2048	4096	30-May-06	>4096	4096
2	45	F	Yes	Yes	Farm	31-May-06	<64	256	16-Jun-06	2048	>2048
3	46	F	Yes	Yes	Rural area	7-Jun-06	64	128	16-Jun-06	2048	>2048
4	35	F	Yes	Yes	Rural area	28-Jun-06	>2048	256			
5	51	M	Yes	Yes	Rural area, gardener	12-Jul-06	2048	64			
6	69	M	Yes	Yes	Farm	17-Jul-06	2048	4096	24-Jul-06	>8192	8192
7	57	F	No	Yes	Lifestyle block	26-Jul-06	1024	1024	9-Aug-06	2048	2048
8	41	M	Yes	Yes	Lifestyle block	5-Aug-06	128	512	15-Aug-06	4096	4096
9	60	M	Yes	No*	Rural bach†	15-Aug-06	1024	4096			
10	39	F	Yes	Yes	Rural area	19-Aug-06	<64	<64	24-Aug-06	128	2048
11	50	M	No	Yes	Rural area	5-Aug-06	512	512	16-Oct-06	2048	512
12	34	F	No	Yes	Farm	3-Oct-06	1024	8192			

*However considerable time spent in rural areas, see text; †Holiday house.

Seven of the patients recalled regularly seeing rats on their properties and of these patients, five described close contact with rats within the incubation period. A further three patients described having rats on their properties in the past, or having several around. Only one rural patient had no knowledge of contact with rats or other animals in their living environment. Three patients could remember a fleabite within the incubation period.

Discussion

Murine typhus is present in rural areas of the Waikato DHB, over an area far more extensive than previously documented. All patients were adults, which is consistent with the very mild illness reported in children. There was no gender predominance.

The clinical presentations were consistent with the known features of murine typhus, but they are still relatively non-specific in relation to the many other causes of febrile illness. Laboratory findings may point to the diagnosis, but are likewise non-specific.

Seroconversion may not occur until the second week of illness and laboratory turnaround times may be prolonged for what will often be a 'send-away' test.

When murine typhus is suspected, doxycycline should be given based on clinical, laboratory, and epidemiological grounds, which will often be before a definitive serological diagnosis is made. Prompt response to treatment is a further clue to the diagnosis, especially defervesence within 48 hours.

Not surprisingly, the 'time taken until the diagnosis was considered' was quite short once patients were admitted to hospital, probably due to the heightened awareness after the first few confirmed cases.

All patients identified had rural contact, similar to the pattern reported from the Auckland area.[2] Many of these patients had obvious rat contact or known fleabites.

Murine typhus is maintained in nature in a rat to rat-flea cycle, with humans infected as incidental hosts only. The rickettsaemic rat is bitten by an "Oriental" rat flea, which ingests the bacteria along with its blood meal. The flea transmits the *Rickettsia* back to the rat either by regurgitation of rickettsiae as it bites[3] or by inoculating infected faeces into the bite wound.[4]

Unlike humans, the natural host and vector are not adversely affected by the infection,[4,5] thus surviving to continue the cycle. Fleas pass *R. typhi* onto their own progeny, by transovarial transmission, with infection seen in 11% of second generation fleas in laboratory conditions.

In the same study, fleas infected 18% of the rats they infested.[6] Humans are usually infected by contact with infected flea faeces, either by inhalation, ingestion, or by inoculation into pruritic fleabites—but humans can also be infected directly by biting.[4] The incubation period is 1–2 weeks, followed by fever that lasts up to 15 days untreated—even treated infections can be followed by prolonged lethargy.

A standard case definition requires that a patient have a consistent clinical illness, along with positive serology (see our 'case definition'), but to be robust, also requires that a patient be from an area of known endemnicity.

Thus, following the first probable case in 1991, the Auckland group attempted to confirm the presence of *Rickettsia* in their community using non-serological means.

Rickettsia is difficult to culture and molecular diagnosis is now routinely attempted instead.

The Auckland group has demonstrated *R. typhi* DNA using PCR, both in rats and in humans.[2] During the course of these investigations, the brushtail possum (*Trichosurus vulpecula*) was investigated as a possible source, given the role of opposum in parts of America, but no evidence was found to support it as possible reservoirs of infection here.[2]

Cat fleas in New Zealand have been found to carry *Rickettsia felis*, of the 'spotted fever' group, but New Zealand cats do not usually carry the Oriental rat flea (carrier of the *Rickettsia* that causes murine typhus) a finding confirmed in a New Zealand cat-flea survey.[7]

This new cluster may represent the first arrival of *R. typhi* in the wider Waikato rat population (known previously in the Coromandel area) or perhaps the new recognition of an illness present for a long time. Alternatively, there may be unknown environmental factors interacting, causing an enhanced transmission from the natural vectors to nearby humans—thus leading to a 'mini-epidemic'.

There is little to support the notion of a new infection in Waikato rats. Firstly, *Rickettsia* has been identified in the nearby Auckland area for over 15 years, and before 2006 there had been 4 documented cases in the Coromandel area.

Also the cases are widely distributed over the upper (northern) part of the Waikato DHB region (see Figure 1), whereas a new infection may be more likely to spread 'outward' from a starting point.

Diagnosis of rickettsial infection in rats is difficult, as it requires trapping and testing enough animals to find an animal that tests positive. The Auckland group has tested over 30 rats from the properties of locally acquired patients, recording only 2 PCR-positive results (S Roberts, personal communication, 2006) Thus the yield even from high-risk rats is low and it is unclear what real benefit there would be in mapping the rat-infected population in the Waikato region.

It is possible that the illness has occurred in the Waikato region for several years without being recognised. Most of the 12 patients were either discussed in consultation with, or diagnosed by, only a few doctors, thus suggesting that the clustering seen here may be due to increased vigilance.

In the past, diagnoses may have been missed because murine typhus is frequently a mild illness and because spontaneous resolution is usual if untreated (undiagnosed). Indeed, physicians unfamiliar with the disease would be unlikely to persevere in finding a diagnosis in such circumstances.

One method of investigating the duration of human infections with *Rickettsia* in the area would be to undertake a large serological survey, to see how many people showed evidence of past infection. However, antibodies wane with time, so only those with more recent illnesses would still be positive. Surveying only those adults that could remember having had a recent febrile illness could increase the detection rate, but logistically this would be challenging. Indeed, a small seroprevalence study, undertaken by the Auckland group, failed to find any positives amongst contacts of their patients.

Murine typhus is known to be seasonal. In the Northern Hemisphere, it is typically diagnosed more in summer, whereas in New Zealand, it is seen predominately over the colder months between April and October (S Roberts, personal communication, 2006). We have, however, subsequently had a 'confirmed' case in the Waikato region in February 2007.

Thus the environment clearly has an influence over transmission, but its mechanism is unknown. It may influence the proximity of rats to humans, or the efficiency of *Rickettsia* transmission. For example, effects on the aerosolisation of flea faeces, rat habitat changes, and the likelihood of rats moving closer to human dwellings—or people involved in more outdoor activities in sheds, garages, and under houses. It is unknown if any specific environmental changes were involved in the occurrence of the cluster seen in the Waikato DHB.

Our investigation confirmed that rurality is the most important demographic for murine typhus. Murine typhus should be considered in the differential diagnosis for those febrile adults from rural areas, especially within the Auckland and Waikato DHB regions.

We believe that it is possible that rickettsial illnesses are occurring in other parts of the country—especially in the North Island, but also potentially in the South Island. Routinely requesting rickettsial serology in those patients who fit the 'rickettsial picture' is likely to identify other areas of endemnicity.

Moreover, it may be particularly useful to consider murine typhus in the differential when the working diagnosis is leptospirosis, given the similarities of rural location, fever and abnormal liver function tests.

There is little hope of eradicating *Rickettsia*, as it would require rat *and* concomitant flea and flea egg eradication. A greater awareness of the disease will lead to earlier diagnoses and will result in improved patient care, fewer and shorter hospital stays, and will reduce unnecessary investigations.

Once a diagnosis of murine typhus is confirmed, treatment is simple and usually effective and can be given presumptively, whilst awaiting confirmation by serology.

Competing interests: None.

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Routine 'Health of the Nation Outcome Scales for elderly people' (HoNOS65+) collection in an acute psychogeriatric inpatient unit in New Zealand

Gary Cheung, John Strachan

Abstract

Introduction HoNOS (Health of the Nation Outcomes Scale) is an outcome measure used by mental health professionals aiming to give a snapshot of a person's psychiatric symptoms and psychosocial functioning.

Aim HoNOS65+ was introduced as a routine outcome measure in our service as part of the New Zealand Mental Health Standard Measures of Assessment and Recovery Outcomes Initiative. The aim of this paper is to report the findings from the use of this tool in our acute psychogeriatric inpatient unit.

Method This is a retrospective analysis of the HoNOS database from 2002 to 2005. Service users were classified into two groups (organic disorders and functional disorders) according to their ICD-10 clinical coding diagnoses. Statistical analysis was performed to compare the HoNOS65+ scores at admission and discharge within each group.

Results There were 431 start episodes and 452 end episodes completed. However, only 130 of these episodes were matched and were usable in this analysis. 29(21%) service users with diagnoses of organic disorders and 101(72%) with diagnoses of functional disorders. The mean total HoNOS65+ score reduced significantly for both groups from admission (organic: 16.45; functional: 14.00) to discharge (organic: 12.34; functional: 8.76). For the organic group, significant improvement was observed in 2 (behaviour and symptoms) subscales; and for the functional group, significant improvement was observed in 3 (behaviour, impairment, and symptoms) subscales.

Conclusion The findings provide objective evidence to support the clinical impression that psychogeriatric inpatient treatment is an effective intervention for both organic and functional disorders. We identified a lack of improvement in the social subscale for both groups. The social subscale scores on admission were relatively low and the lack of improvement might be because specific interventions were not necessary during the inpatient episode. Further exploration of this subscale would be useful in clarifying whether there is a need to plan service development to address these aspects of service users' care during the acute admission phase.

HoNOS (Health of the Nation Outcomes Scale) is an outcome measure used by mental health professionals aiming to give a snapshot of a person's psychiatric symptoms and psychosocial functioning.

The New Zealand Mental Health Standard Measures of Assessment and Recovery (MH-SMART) Outcomes Initiative is a national programme which aims to support recovery by promoting and facilitating the development of an outcomes-focused culture in the mental health sector.[1]

The Health of the Nation Outcomes Scale family of measures—HoNOS[2], HoNOSCA[3], and HoNOS65+[4]—was the first suite of measures introduced in 2000/2001. Other outcome measures currently under consideration include a service user self-assessment measure, a Hua Oranga measure for Māori, a Pacific people's measure, and a functioning measure.

The suitability of HoNOS65+ as an outcome measure in old age psychiatric service was previously reported by Turner who found it has satisfactory reliability, particularly when sufficient attention is paid to training and supervision of the raters.[5]

Spear et al reported it meets the criteria for an outcome indicator for community mental health services for older people.[6] In their study, HoNOS65+ was sensitive to change. In a recent systematic review, HoNOS65+ has good concurrent validity and inter-rater reliability.[7] However, there is currently insufficient evidence for its content validity, construct validity, predictive validity and test-retest reliability.

HoNOS65+ has also been incorporated as an assessment measure in clinical research.[8,9] However, reports of its use in routine clinical practice are lacking in the literature. MacDonald reported methods of analysing routine clinical outcomes in an old age psychiatry service, without specific report of the findings of HoNOS65+ in his group of service users.[10] There are reports of using HoNOS (but not HoNOS65+) to measure psychiatric symptomatology and to monitor clinical outcomes of older people managed in psychiatric services.[11,12]

McKay et al suggested the goal of outcome measures is to guide clinical practice, to improve consumer and carer outcome, and the development of services.[13] Andrews et al criticised the use of HoNOS as part of the Australian National Mental Health Strategy[14] where there are many mental health services in Australia collecting outcome measures, but very few are using them for clinical management or service development.[15]

The aim of this paper is to report the findings from a routine collection of HoNOS65+ in an acute psychogeriatric inpatient unit in New Zealand. The HoNOS database kept by our service is one of the longest since it was introduced in New Zealand.

Method

Service description—The Mental Health Services for Older People (MHSOP) at Health Waikato is based in Hamilton, New Zealand. It is a secondary specialist referral service with an estimated catchment population of 42,500 people aged 65 years and over. There are 20 full-time equivalent (FTE) staff (including 3 psychogeriatricians) in two community teams.

The inpatient unit is located within the mental health inpatient complex and has 11 beds. Both late-life organic and functional disorders are treated in the same unit.

HoNOS65+ collection—Routine HoNOS65+ collection at admission (start episode) and discharge (end episode) was introduced in the inpatient unit in February 2002. All the staff involved in completing the ratings had received training in a 4-hour workshop.

Other information collected at the end of an episode of care includes: focus of care, International Classification of Diseases (tenth revision; ICD-10) diagnosis,[16] legal status, cultural support services, and non-government organisation involvement.

The HoNOS65+ ratings, demographic details (date of birth, gender, ethnicity) and the other information are entered into a database held by the MH-SMART local site coordinator. This report is a retrospective analysis of the database for the period of February 2002 to December 2005.

Statistical analysis—For the purpose of statistical analysis and comparison, service users were classified into two groups according to their primary ICD-10 diagnosis.

The first group "Organic disorders" included those with ICD-10 diagnostic codes of F0; and the second group "Functional disorders" included those with ICD-10 diagnostic codes of F1 (Substance), F2 (Psychosis), F3 (Mood), F4 (Anxiety), and F6 (Personality).

The student paired *t*-test was used to compare the HoNOS scores between start episodes and end episodes within each group. The student unpaired *t*-tests was used to compare the HoNOS scores and other demographic figures between the organic and functional groups.

For each group, regression analysis was used to determine the relationship between the following variables: age and length of an episode; total HoNOS score and length of an episode; and age and total HoNOS score.

Results

There were 431 start episodes and 452 end episodes completed between February 2002 and December 2005. However, only 130 (about one-third) of these episodes are matched (i.e. a start episode and an end episode were both fully completed for a particular admission).

There were 29 (21%) service users with diagnoses of organic disorders (ICD-10 Category F0) and 101 (72%) with diagnoses of functional disorders (ICD-10 Categories F1-6). The most common diagnostic category was mood disorder with 72 (51%) service users. There were 13 (45%) male and 16 (55%) female in the organic group; while there were 39 (39%) male and 62 (61%) female in the functional group.

The mean ages of the service users in the organic and functional groups were 77.9 (SD=10.6) years and 75.1 (SD=6.7) years respectively (p=0.092). Majority of the service users were of European descents: 23 (73%) in the organic group and 93 (92%) in the functional group. The other ethnic groups were Māori: 3 (10%) in the organic group, 3 (3%) in the functional group; and non-specified: 3 (10%) in the organic group, 4 (4%) in the functional group. There was also one service user of Middle East descent in the functional group.

The mean scores for each of the 12 HoNOS65+ items from the start episodes are shown in Table 1. The items which were rated significantly higher in the organic group were item 1 (behaviour), item 4 (cognition) and item 10 (activities of daily living).

The total 12 items HoNOS65+ scores and the four subscales (behaviour, impairment, symptoms and social) scores for the organic and functional are shown in Table 2. The total HoNOS65+ scores reduced significantly from the start episode to the end episode in both groups. For the organic group, significant improvement was observed in 2 (behaviour and symptoms) subscales; and for the functional group, significant improvement was observed in 3 (behaviour, impairment and symptoms) subscales.

The mean episode length was significantly longer in the functional group: 37.6 (SD=40.2) days vs 24.3 (SD=19.8) days in the organic group (p=0.016). However, the length of an episode was not related to the age of the service user nor the total HoNOS65+ score from the start episode. Length of episode × age (Organic group: R^2=0.09, p=0.124; Functional group: R^2<0.00, p=0.717). Length of episode × HoNOS65+ (Organic group: R^2= 0.03, p=0.372; Functional group: R^2=0.01, p=0.257).

There was also no relationship between the age of the service user and the total HoNOS65+ score in either group (Organic group: R^2<0.00, p=0.818; Functional group: R^2<0.00, p=0.631).

Table 1. Mean HoNOS65+ item scores; organic disorders vs functional disorders

Start episodes		Organic disorders (N=29)	Functional disorders (N=101)	P value
Item no.		**mean**	**mean**	
1	Aggression, disruptive etc behaviour	1.69	1.00	**0.016**
2	Deliberate self-harm	0.59	0.70	0.635
3	Alcohol/drug problems	0.41	0.41	0.971
4	Cognitive problems	2.83	1.17	**<0.001**
5	Physical problems	1.72	1.50	0.420
6	Hallucinations/delusions	0.93	0.89	0.891
7	Depressed mood	1.52	2.08	0.070
8	Other psychological problems	1.79	2.09	0.283
9	Relationship problems	1.31	1.43	0.681
10	Activities of daily living	2.14	1.49	**0.02**
11	Accommodation problems	0.9	0.66	0.332
12	Occupational/leisure problems	0.62	0.59	0.906
Total (Items 1–12)		**16.45**	**14.00**	**0.067**

Table 2. Total HoNOS65+ scores and subscale scores; episode start vs end (Organic disorders and functional disorders)

Variable	Organic disorders		Functional disorders		Australian report (AMHOCN)[16]	
	Start episode (SD)	End episode (SD)	Start episode (SD)	End episode (SD)	Start episode (SD)	End episode (SD)
	N=29	N=29	N=101	N=101	N=4227	N=3264
Behaviour (Items 1-3)	2.69 (1.91)	1.31** (1.37)	2.11 (21.15)	0.52** (1.03)	2.5 (2.2)	1.1 (1.6)
Impairment (Items 4-5)	4.55 (1.97)	4.14 (2.31)	2.66 (2.03)	1.98** (1.73)	3.2 (2.1)	2.7 (2.1)
Symptoms (Items 6-8)	4.24 (2.52)	2.24** (2.37)	5.06 (2.23)	2.50** (1.99)	4.8 (2.7)	2.7 (2.4)
Social (Items 9-12)	4.97 (4.12)	4.66 (3.14)	4.17 (3.60)	3.75 (3.55)	4.9 (3.7)	3.3 (3.2)
Total (Items 1-12)	16.45 (6.82)	12.34* (7.28)	14.00 (6.15)	8.76** (6.44)		

*p<0.01; **p<0.001; AMHOCN=Australian Mental Health Outcomes and Classification Network.

Discussion

The total HoNOS65+ scores reduced significantly from start to end episode in both the organic and functional groups, thus suggesting inpatient intervention was effective for both groups in improving clinical outcomes.

Mean subscale scores from start and end episodes for psychiatric inpatient sourced from the Australian Older Person Outcome and Casemix Collection Standard Reports[17] were included in Table 2 for comparison with our findings.

The Australian report was chosen because such nationwide mandatory reporting will not be required in New Zealand until July 2008. The Australian report did not differentiate between organic and functional disorders and there was no matched episode analysis performed. However, the start episode HoNOS65+ subscale scores

for our organic and functional groups are similar to the Australian Report, thus suggesting service users in both countries are presenting with similar acuity as psychiatric inpatients.

We suggest separate analysis for organic and functional disorders should be adopted for old age psychiatry services so that a more direct comparison can be made for service users with these two groups of disorders.

We believe this routine collection of HoNOS65+, coupled with demographic and diagnostic data, is fulfilling some of the goals of outcome measures as suggested by McKay et al.[13] The distribution of diagnoses informs us as to resource allocation for the management of organic (about one-third of service users) and functional disorders (about two-thirds of service users). The impairment subscale did not improve from start to end episode in our organic group. This subscale captures cognitive problems, physical illness, and disability problems.

The majority (79%) of the service users in the organic group had a diagnosis of dementia. Dementia is a chronic progressive mental disorder that adversely affects higher cortical functions including memory, thinking and orientation. Its progression is characterised by deterioration in cognition and functional ability. Therefore, the lack of improvement in the impairment subscale is probably more a reflection of the irreversible nature of the disease, than ineffective treatment.

In contrast, the impairment subscale improved significantly from start to end episode in the functional group. Older people with functional disorders, such as late-life depression, often present with cognitive deficits, physical illnesses, and disability. These impairments can often be reversible with treatment of the underlying functional disorders and our findings certainly support this.

We identified the lack of improvement in the social subscale of HoNOS65+ for both organic and functional disorders. This subscale captures problems with relationships (Item 9), activities of daily living (Item 10), living conditions (Item 11), and occupation and activities (Item 12).

Orrell et al and Brooks found the lowest inter-rater reliabilities on all the items comprising the social subscale of HoNOS.[18,19] Similarly, Burns et al found that the inter-rater reliability of Items 10, 11, and 12 did not consistently perform well with HoNOS65+.[4] Therefore, the lack of improvement in the social subscale in our report will have to be interpreted cautiously.

Two of the main objectives of the MH-Smart programme are to use outcome measures: to assist clinicians to assess, plan and evaluate the services they deliver; and to assist service managers to understand and assess service delivery factors and dynamics.

If this lack of improvement in the social subscale is a true reflection of our clinical practice, future service development will be important to target these aspects of service users' care in order to further improve their outcomes. For example, more intensive occupational therapy focusing on rehabilitation may improve service user's role functioning and activities of daily living at time of discharge from the inpatient unit.

In contrast, the lack of improvement in the social subscales might be simply because interventions were not necessary to target these problems during the inpatient episode.

The social subscale scores on admission were relatively low. As shown in Table 1, the only item in the social subscale scored more than 2 was Item 10 in the organic group. An intervention is usually required when an item is rated at levels 2 to 4 and an item rated at level 1 indicates a sub-threshold problem.

This retrospective analysis has found no relationship between the length of an episode and the total HoNOS65+ score from the start episode, a finding similar to a multicentre study using the adult version of HoNOS on six private psychiatric hospitals in Australia.[20] The authors commented that HoNOS is unlikely to be of utility in predicting length of stay or in offering a 'gate-keeping' service in decision-making in regard to the allocation of resources for individual patients.

The large number of unmatched episodes in this retrospective review is concerning. This could be a compliance issue with inpatient staff not completing the ratings and/or completed paper-based ratings were lost.

Since January 2006, staff in our service can complete HoNOS ratings online and this will minimise loss of data. A process is now in place to generate reminders for uncompleted episodes. A follow-up review of the ratio of unmatched episodes: matched episodes will be useful in determining the effectiveness of this online system.

Furthermore, the New Zealand Ministry of Health is currently planning a nationwide evaluation of the implementation of the MH-Smart programme for mid-2007. This is aimed at assessing the effectiveness of the programme in terms of supporting clinical staff in the delivery of good quality care to service users.

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Heracleum mantegazzianum and *Toxicodendron succedaneum*: plants of human health significance in New Zealand and the National Pest Plant Accord

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Abstract

New Zealand's National Pest Plant Accord (NPPA) is a voluntary and cooperative agreement between industry, regional councils, and central government departments with biosecurity responsibilities (primarily the Ministry of Agriculture and Forestry and the Department of Conservation). Plant species included in the NPPA are declared unwanted organisms under the Biosecurity Act 1993, which prevents their sale, propagation, or distribution across the country.

Although MAF Biosecurity New Zealand (the lead agency in New Zealand's biosecurity system) has evaluated the potential human health impacts of 202 species considered for inclusion in the NPPA, two species were examined primarily due to their significance to human health: *Heracleum mantegazzianum* (giant hogweed, cow parsnip, wild parsnip) and *Toxicodendron succedaneum* (rhus tree, wax tree, Japanese wax tree). As a result of this process, *H. mantegazzianum* has been listed in the NPPA. In contrast, *T. succedaneum* was not included in the NPPA, as the latter was deemed to be an inappropriate mechanism for its control.

In this article the NPPA process is outlined, and the adverse impacts on human health of these two species are discussed—including symptoms, treatment, and possible management measures.

The National Pest Plant Accord

The National Pest Plant Accord (NPPA) is a voluntary and cooperative agreement between the Nursery and Garden Industry Association, regional councils, and government departments with biosecurity responsibilities (primarily the Ministry of Agriculture and Forestry [MAF] and the Department of Conservation [DOC]).

The NPPA seems to be the only agreement of its kind in existence (M Newfield, personal communication, 2006). All plant species listed under the NPPA are automatically declared unwanted organisms under the Biosecurity Act 1993, pursuant to section 2(1). This prevents their legal sale, propagation, or distribution within New Zealand.

When listing plants emphasis is given to species that are invasive, pose the greatest level of threat, are primarily spread by people, and have limited actual distribution relative to their potential distribution.[39]

The NPPA came into effect for the first time on 1 October 2001. It was revised in 2006, with signatory parties renewing their commitment to the Accord for another five years. Although the Accord is not a binding contract, it is intended to carry the same effect as a Memorandum of Understanding between the signatory parties. As a

result, these parties share responsibility to promote compliance with the rules of the Accord, and regional councils have specific responsibility for actively monitoring compliance (A Harrison, personal communication, 2006).

There were 202 species initially considered for inclusion in the NPPA during the 2006 revision of the Accord. The final approved list includes 109 individual plants species and all the species present in four genera.[40]

Following the creation of Biosecurity New Zealand within MAF in 2004, the Ministry has acquired greater biosecurity accountabilities including an oversight role for the biosecurity system and explicit accountability to protect the full range of societal values through its activities. As a result, MAF aims to ensure that human health values are adequately considered within its routine activities. Therefore, under this new role, apart from the potential impact on the environment and the economy, MAF Biosecurity New Zealand (MAFBNZ) was also accountable for evaluating the potential human health significance of all plants being assessed for inclusion in the NPPA.

Several introduced plants species in New Zealand can cause adverse reactions in humans via skin contact—e.g. *Urtica dioica* (perennial nettle) and *Ficus carica* (fig tree)—with a number of other species being poisonous if plant parts are ingested. However, two plant species in particular were evaluated for the NPPA as a result of their public health significance and the possible need to officially control them: *Heracleum mantegazzianum* Sommier et Levier (Apiaceae) (giant hogweed, cow parsnip, wild parsnip) and *Toxicodendron succedaneum* (L.) Kuntze (Anacardiaceae) (rhus tree, wax tree, Japanese wax tree).

Both species are introduced (i.e. not native) to New Zealand, and their adverse effects to human health have been widely documented. They are, for example, included in the list of plants in New Zealand that are poisonous to children compiled by Landcare Research.[37]

In this article, the relevance of these two species to human health are discussed, including symptoms and treatment, as well as the final decisions of the NPPA process and some possible management measures.

Heracleum mantegazzianum

Background—*Heracleum mantegazzianum* is a giant herb that varies in height from 2.0 to 5.0 m, and which may live for several years.[1] It is a popular plant in gardens due to its attractiveness (Figure 1).[2,3] *Heracleum mantegazzianum* is native to Asia, more specifically the western Caucasus but it is now widespread in Europe[1] and North America.[4]

In the United States this species escaped from cultivation and has become a public health hazard—being found in urban, suburban, and rural settings.[5] As a result, *H. mantegazzianum* is on the United States federal noxious weed list, which means that its importation into the country is illegal, as is the interstate and intrastate movement of this species.[6]

While *H. mantegazzianum* appears to be present in Dannevirke, Napier, Wellington City and in a few scattered localities in Marlborough, Canterbury, Otago, and Southland,[7] its actual current distribution in New Zealand is unknown.

Figure 1. *Heracleum mantegazzianum* with its large leaves and flowerheads (Photo courtesy of Rune Aanderaa, SABIMA)

Mode of action—The sap of *H. mantegazzianum* contains psoralens (furocoumarins) that lead to a relatively common type of dermatitis: phytophotodermatitis, which is produced by the interaction of such plant compounds with sunlight on human skin.[8–11] The psoralens are lipid-soluble and penetrate into the epidermis with ease.[12] The photochemical excitation of psoralens is induced by ultraviolet (UV) radiation, usually within the UVA wavelengths of 320–400 nm.[13] Note that the absorption of psoralens into the skin (and the consequent reaction) is enhanced by high humidity.[14]

A detailed study of the mechanisms of phytophotodermatitis has been provided by Pathak (1986).[14] In brief, two types of toxic reactions occur: one oxygen-independent where the ultraviolet-activated psoralens bind to RNA and nuclear DNA, and an oxygen-dependent reaction where the induced compounds cause cell membrane damage and oedema.[11,38]

These reactions consequently lead to cell death.[3,11,15,38] This is a phototoxic reaction, and not an allergic one so there is no immunological response. As a result, no prior sensitisation is necessary and anybody can be affected.[16] *Heracleum mantegazzianum* is one of the main causes of phytophotodermatitis in the United Kingdom and United States.[4]

The plant's clear watery sap is said to exude from all parts of the plant, so dermatitis is induced via contact with leaves, stem, seeds, and roots.[4] An analysis of

furocoumarin contents indicated that their concentration in *H. mantegazzianum* plants was highest in fruit, intermediate in leaves, and low in stems.[15]

Nonetheless, touching the plant or brushing against it appears to be enough to induce exposure to the sap, and all persons that come in contact with it seem to be affected to some extent. It should be noted that the content of furocoumarins in the sap of *H. mantegazzianum* varies with individual plants, season (being highest in spring), and probably also soil conditions and climate.[2,10,15]

Clinical features—The consequent symptoms of the induced phytophotodermatitis appear usually to have a benign character, but they can lead to severe blistering and painful burn-like lesions.[5,15] The diagnosis based on the symptoms displayed by the patient is difficult, and adequate diagnosis is clinically based on history and physical examination.[13,17] In some cases, the symptoms of phytophotodermatitis have been mistaken for child abuse.[4]

In addition, as a result of the aggressive progression of the symptoms, phytophotodermatitis induced by *H. mantegazzianum* is commonly mistaken for resistant staphylococcal infections or necrotising fasciitis.[5]

Contact with the plant sap and exposure to UV light leads to erythema, oedema, and burn-like lesions within 24 hours and possibly large, fluid-filled blisters within 48 hours.[3,8,10] The occurrence of pruritus is uncommon,[18] but secondary skin infection is a possible complication.[5]

The blisters can develop into purplish or blackened scars, with skin hyperpigmentation remaining visible for months or even years after exposure.[3,4,8,10,16]

In addition, the affected areas may remain hypersensitive to UV light for many years.[4,9,16] The occurrence of systemic manifestations is rare.[13] Children appear to be particularly attracted to playing with *H. mantegazzianum*'s large and hollow stems, which are used for play swords and telescopes.[5]

It is important to highlight that phytophotodermatitis may occur via indirect contact with *H. mantegazzianum* sap. For example, a woman developed phytophotodermatitis as a result of contact with her cat that had been playing with a specimen of the plant in the garden.[8] Dogs are also described as being frequent carriers of the plant's sap on their fur, which is then transferred to the owners' skin.[5] It seems that mammals other than humans may also be affected by phytophotodermatitis induced by this plant.[19]

Treatment—Immediately after exposure, the skin should be thoroughly washed with soap and cold water to remove plant sap.[5,9] The exposed skin should be protected from sunlight by covering and/or the application of sunscreen, until at least 48 hours post-exposure even if asymptomatic.[3] If sap enters the person's eyes, these should be thoroughly flushed with cold water or irrigation solution.[5] Although there is suggestion that exposure to furocoumarins can cause permanent blindness,[5] there appear to be no cases documented in the medical literature.

Since most people are unlikely to seek medical attention prior to the onset of symptoms, management is usually symptomatic and supportive.[3,5] Wet compresses, ice packs, and paraffin gauze dressings my assist to reduce swelling and inflammation.[3,5]

An effective treatment may consist of wound debridement and daily dressings with silver sulphadiazine, which seems to be effective and safe.[15] In any case, keeping blistered areas clean with the use of topical antiseptics is advisable to prevent the onset of secondary infection.[13]

In some cases, treatment with an oral or topical anti-inflammatory medication is advised,[5,15] with severe cases possibly requiring hospitalisation for analgesia and supportive care.[3,5] Where intense pruritus occurs, antihistamines may be used.[13]

The healing process can take up to 2 weeks,[15] but in some cases symptoms may last for over a month.[5] Moreover, it is necessary to monitor the patient for secondary infection, and educate the person to avoid future exposure.[18]

Although the subsequent hyperpigmentation requires no treatment, hydroquinones may be used.[18] Since affected areas may remain hypersensitive to sunlight for months or years, the continued use of sunscreen is advisable.[3,18]

***Heracleum mantegazzianum* in New Zealand and NPPA recommendations—** Unfortunately there is currently no information on the number of cases of phytophotodermatitis induced by *H. mantegazzianum* in New Zealand.

However, unlike other plants in New Zealand that cause adverse reactions in humans, *H. mantegazzianum* stands out as a particular threat for several reasons, including:

- Potentially severe symptoms caused by phytophotodermatitis;
- Its growth habit (giant herb) making it more likely for people to come into contact with the plant (compared with a tree or a low-growing herb);
- The fact that in other temperate countries it is a serious weed that invades high use areas likely to be used by people—such as river and stream banks, roadsides, and right-of-ways.

Although the distribution of *H. mantegazzianum* is currently limited in New Zealand, this species has the potential to become widespread along river and stream banks, especially in areas fenced off from grazing stock.

In addition, the use of *H. mantegazzianum* as a garden plant has been promoted in the past, even though current trade in this plant is very limited. Therefore, due to its significance to human health and its potential to become an invasive species in New Zealand, *Heracleum mantegazzianum* has been included in the list of plants covered by the Accord.

Toxicodendron succedaneum

Toxicodendron succedaneum is a relatively small deciduous tree, native to Eastern Asia, that grows to approximately 12 m.[20,21] It has attractive autumn foliage, which makes it sought after as an ornamental tree (Figures 2 and 3). However, *T. succedaneum* is as allergenic as poison ivy (*T. radicans*), although it seems to be less of a clinical problem than the latter since it grows as a tree rather than a creeper (M Rademaker, personal communication, 2005). Plants in the family Anacardiaceae are the main cause of allergic contact dermatitis (ACD) induced by plants worldwide,[16,22] with *Toxicodendron* spp. being by the far the most common cause.[23]

Figure 2. Canopy of *Toxicodendron succedaneum* showing the characteristic bright reddish colours of its deciduous autumn leaves (Photo courtesy of DermNet)

Figure 3. A sapling of *Toxicodendron succedaneum* showing green leaves (Photo courtesy of Auckland Museum)

In the United States, ACD caused by *Toxicodendron* spp. is a significant occupational hazard for agriculture and forestry workers as well as recreational wilderness users.[24] There are substantial associated medical costs, and major economic losses as a result of the consequent morbidity in particular amongst forestry workers.[24]

Mode of action—The sap of *Toxicodendron* spp. contains urushiols that are extremely potent sensitisers, in particular the allergen catechols.[16,21,25,26] Urushiol is described as colourless or slightly yellow, but once exposed to air it oxidizes and polymerizes turning black.[24]

It is estimated that at least 50% (but possibly as much as 75%) of the adult population in the United States is hypersensitive to the urushiol of *Toxicodendron* spp. and would likely develop clinical symptoms.[12,23,24,27,28] Note that the expression of ACD is partially dependent on genetic factors.[12] ACD is a cell-mediated response to exposure to an antigen of a relatively low molecular weight that can penetrate the epidermis.[23] *Toxicodendron* induced dermatitis is more intense in adults, although many severe cases can be observed in children.[12]

The antigens of all *Toxicodendron* spp. are essentially the same, so cross-sensitivity between different species occurs.[26] Urushiol is found in all parts of the *Toxicodendron* plant including stems, leaves, roots, and fruit skin.[24] ACD is induced following exposure to a damaged part of the plant,[24,25] and the latter is necessary to allow the urushiols (oleoresins) to contact the skin, as the uninjured plant is innocuous.[24,26] In addition, allergic contact dermatitis can occur following inhalation of the smoke of burning plants, as the urushiols may be present in the particulate matter.[21,26,29]

Inhalation of these particles may therefore result in an allergic response affecting mucous membranes for example, and ACD may also occur on skin where particles may settle. In some cases contact with urushiols may occur via wind transmission.[21]

Urushiol is non-volatile and dries quickly on fomites—persisting on clothes and equipment indefinitely.[24] It seems that pets, particularly long-haired dogs, may be responsible for transmitting the oleoresin from *Toxicodendron* plants to children.[12] Aggravating the threat posed by *Toxicodendron* spp. is the fact that dermatitis can occur following contact with dead plant tissue, as urushiol retains its antigenic potential in the dry state indefinitely.[26]

Note that most of the literature available on *Toxicodendron* refers to North American species, mainly *T. radicans* (poison ivy), *T. vernix* (poison sumac), and *T. diversilobum* (poison oak). Nonetheless, since, as previously mentioned, the antigens of all *Toxicodendron* spp. are essentially the same,[26] symptoms of exposure, treatment, and management are applicable to all species.

Clinical features—Although most contact allergens require repeated exposures to trigger an immune response, the catechols of *Toxicodendron* spp. are potent, and susceptible people may become sensitised after only two exposures.[23] After a sensitised person comes into contact with urushiol, the symptoms usually appear within 2 days.[12] However, symptoms may appear as early as within 6 hours, and may be delayed for as many as 12 days after contact with the plant's urushiol.[30]

Erythematous papular lesions that itch intensely are usually the initial symptoms,[23] and these may be associated with an intense burning sensation, and often advance to

raised lesions.[30] Pruritus is intense in all stages of the lesions and is characteristic of *Toxicodendron* ACD.[23] The severe itching may lead to scratching with excoriation and secondary lesions, possibly leading to infection.

Approximately 48 to 72 hours after exposure, vesicular lesions develop and erupt, releasing plasma that forms a crust.[23] Vesicles are often numerous and small but bullae can occur in severe reactions.[23] Facial oedema with marked periorbital swelling are particularly common in children.[21]

In moderately severe cases, oedematous swelling of various parts of the body may occur, while in severe cases *Toxicodendron* ACD is characterised by widespread symptoms and marked oedema of the extremeties and face.[26]

The fluid that exudes from vesicles and bullae do not contain the allergen and therefore the patient cannot spread the dermatitis to other persons or other parts of the body.[24,26,27,31] However, the rash may grow in size and new vesicles may develop during the first 2 weeks without further exposure to urushiol, which leads to the common belief that the serum from the vesicles contains the antigen.[23]

The severity of the response will also vary between individuals, some experiencing mild reddening while other patients may become temporarily disabled.[31] The extent and severity of the lesions will vary due to a number of factors, especially level of exposure (area of contact and amount of urushiol involved), patient's sensitivity to the allergen, site of contact, and skin thickness.[23,30,31] In addition, lesions may develop in areas not directly exposed to the plants due to secondary exposure (via contaminated hands, clothing, tools, etc.), and also due to the non-specific effect of the cell-mediated response.[23]

The ACD induced by *Toxicodendron* spp. usually resolves within 3 to 4 weeks,[23] but it may last for 6 weeks in more susceptible individuals.[24] Hyperpigmentation may occur in darkly pigmented individuals, which may last for months.[23,24] Although complications and systemic effects may occur, these appear to be uncommon.[24] Secondary infection, however, appears to be more common.

Ingestion of *Toxicodendron* plant material leads to symptoms that occur mostly within 1 day.[32] Chewing or ingestion of the leaves is likely to result in inflammation of the oral mucous membranes, and may cause severe gastroenteritis—with nausea, vomiting, diarrhoea, abdominal pain, and proctitis.[12] Other systemic symptoms may include fever, chills, headache, and fatigue—and, in very serious cases, hypotensive shock may occur.[32]

Ingestion of *Toxicodendron* plant material can also lead to systemic contact dermatitis, with symptoms as those resulting from direct skin contact.[32]

Treatment or management—Following contact with the sap of *Toxicodendron* plants, it is important to wash the affected area immediately with warm soapy water.[25] All clothing, tools, or other objects or pets that have been exposed to the urushiol should be adequately washed with common soap or detergent, which renders the urushiol-contaminated areas or fomites harmless.[26]

Urishiol may remain under a person's fingernails, which must be washed to prevent further self-exposure or contamination of other individuals.[26] Note, however, that the urushiol binds to skin proteins within a few minutes after exposure, and thorough washing would only remove the remaining oleoresin yet to bind.[26,30]

After approximately 30 minutes post-exposure, all urushiol is likely to have been absorbed into the skin.[24] However, there are some specific detergents that seem to optimise the removal of urushiol from human skin, which may be applied a couple of hours after exposure.[23,24,30]

Eruptions may be treated with topical corticosteroids, although severe cases may require hospitalisation and systemic administration.[25,28] In New Zealand, three-quarters of the patients confirmed to be affected by *Toxicodendron* dermatitis were treated with a reducing dose of prednisone, and the remaining with potent topical corticosteroids and systemic antihistamines.[33]

Note that such corticosteroids may alleviate but not prevent the development of symptoms.[30] Detailed discussions on the treatment of *Toxicodendron* ACD have been provided by other authors.[12,23,30]

***Toxicodendron succedaneum* in New Zealand and NPPA recommendations—** *Toxicodendron succedaneum* is without doubt the most allergenic plant species in New Zealand causing contact dermatitis, and one that certainly causes public harm (M Rademaker, Waikato Hospital, personal communication, 2006). In 1993 alone there were at least 20 cases of allergic contact dermatitis due to *T. succedaneum* recorded in the Waikato Hospital.[34] There were at least 92 cases of contact dermatitis due to *T. succedaneum* in the Waikato region between 1982 and 1994.[33]

At least 55 cases involved young people (0–20 years) who were affected during outside play, most of which involved lesions to the face.[21] In contrast, almost all cases involving those aged 21 or older occurred while gardening.[21]

Toxicodendron succedaneum is not yet officially controlled in New Zealand, but it is classified as a noxious weed in the Australian states of South Australia[35] and New South Wales,[36] where all specimens of this plant must be destroyed.

In Australia, *T. succedaneum* was sold for many years as a garden plant, but since its declaration as a noxious weed it can no longer be offered for sale;[36] a similar situation to Japan.[33,34] In addition, in New South Wales for instance, public education has assisted in leading to a considerable reduction in the number of trees.[36]

While the potential environmental impact of *T. succedaneum* in New Zealand is uncertain, there seems to be no naturalised population of this plant. However, in Sydney (Australia) *T. succedaneum* is considered to be a serious weed problem where birds spread the seeds in their droppings—and many thousands of seedlings were flourishing in home gardens, in public areas, and in urban bushland.[36] *T. succedaneum* can also be spread by movement of garden soil containing seed, which remains viable for many years.[36]

The Steering Group overseeing the NPPA process decided that there was no justification for including *T. succedaneum* in the NPPA list. According to the horticultural industry, "this plant is not a species that is currently being sold in New Zealand."

The NPPA's Technical Advisory Group also concluded that there is no evidence that this species is invasive in New Zealand or is spread by humans. Therefore, although *T. succedaneum* warrants some management due to its potentially serious effects on human health, it did not meet the criteria for inclusion in the NPPA, and this was

consequently deemed not to be the appropriate mechanism to address the risks to human health associated with this plant.

Hazard management and conclusion

As a result of the inclusion of *H. mantegazzianum* in the National Pest Plant Accord, this plant is now an unwanted organism. As a result, its sale, propagation, and distribution across the country are illegal. There is however, no requirement for existing plants to be destroyed.

Due to *H. mantegazzianum*'s threat to public health and its potential invasiveness, MAFBNZ encourages the general public, regional, and local authorities to destroy this plant. Nonetheless, extreme care should be exercised when removing these plants, and it must be stressed that contact with dead plant parts and with inanimate objects or pets that have been in contact with such plants is dangerous.

The use of protective water-resistant clothing and protective goggles is advisable when dealing with *H. mantegazzianum*, as is the simultaneous avoidance of exposure to sunlight.[1,15]

A detailed management plan for *H. mantegazzianum* was produced by Nielsen et al. (2005) who assessed various control methods.[1] The authors stated that: "currently used control methods comprise a variety of manual and mechanical methods, grazing and herbicide application", and that "rather than recommending a single control method, a control programme based on an integrated weed management strategy (IWMS) is preferred" (p30).

In regards to *T. succedaneum*, since it has not been listed in the NPPA, no official measures have been imposed on its sale or propagation in New Zealand. However, MAFBNZ encourages local and regional authorities to consider taking action against this species in the interest of public safety.

Specifically, MAFBNZ recommends that councils promote or carry out active removal of *T. succedaneum* (and also *H. mantegazzianum*) from public places—including schools, parks, reserves, and other high public use areas. It seems that the Hamilton City Council, for example, no longer plants *T. succedaneum* and has removed many such trees from public places or other areas on medical request.[34]

Regarding the removal of *T. succedaneum*, like *H. mantegazzianum*, dead plant parts or anything that has been in contact with the plant poses a risk, as ACD can be developed by contacting tools, pets, or clothing that have been in direct contact with the urushiol previously.

The removal of *Toxicodendron* plants consequently has to be done with care, and as much of the skin area as possible should be adequately covered. It seems that it is necessary to use heavy-duty vinyl gloves, as rubber gloves are not very protective as the catechols in urushiol can penetrate most, if not all, types.[12] The plants removed should be buried or burnt.[26] However, as previously pointed out, burning *T. succedaneum* may also lead to exposure, and any person in the vicinity should maintain a safe distance to avoid exposure to urushiol that may be carried in the ashes or smoke.

Finally, medical practitioners that come across cases of dermatitis as a result of contact with these plant species in private properties should recommend the removal

of the specimen(s). Moreover, when the particular plant is located on public land, the medical practitioner should inform the local authorities, as consideration should be given on whether the plant needs to be removed in the interest of public health.

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Rural hospital medicine in New Zealand: vocational registration and the recognition of a new scope of practice

Garry Nixon, Katharina Blattner; for the NZ Rural Hospital Doctors Working Party

Abstract

Establishing a professional body for rural hospital generalist doctors was identified as a priority by the Rural Hospital Doctors Working Party which was formed in July 2005. In May 2006 the Working Party lodged an application with the Medical Council of New Zealand (MCNZ) for Branch Advisory Body status and the recognition of rural hospital medicine as a new scope of practice. The Branch Advisory Body will sit within the Royal New Zealand College of General Practitioners. It will have an independent Board of Studies which will administer vocational training and reaccreditation programmes for these doctors.

These initiatives represent an opportunity to generate the skilled generalist medical workforce NZ rural hospitals need to move into the future. The application is currently before the MCNZ, and a final decision is expected later this year. This paper outlines some of the main points contained in the application document.

Introduction

In July 2005 a working party was formed to examine the vocational issues faced by doctors working in our small rural hospitals*, principally those with no or very limited specialist cover. In their hospital role, these doctors provide generalist secondary level care, across the entire spectrum of medical presentations.

The Working Party identified the formation of a professional body for rural hospital generalist doctors as a priority. This body will sit within the Royal New Zealand College of General Practitioners (RNZCGP). It will have an independent Board of Studies which will administer vocational training and reaccredidation programmes for these doctors .and act as a focus for their professional development. This will bring rural hospital medicine into line with other branches of medicine in New Zealand.

In May 2006 the Working Party lodged an application with the Medical Council of New Zealand (MCNZ) for Branch Advisory Body status and the recognition of rural hospital medicine as a new scope of practice. The MCNZ has undertaken stakeholder consultation, in particular consultation with other colleges, prior to considering the application. A final decision is expected later this year.

Rural hospital medicine is challenging, hands on, and varied. The hospitals are valued by their communities and can provide an excellent working environment that sits alongside the lifestyle advantages of rural living. These proposals will help create the right professional environment. Once this is done rural hospital medicine has the potential to become an attractive career choice.

The New Zealand rural hospital medical workforce

Limited relevant data exists on the New Zealand rural hospital medical workforce. There are about 120 rural hospital doctors who fall into three broad categories.

- About half are rural General Practitioners (GPs), usually with part time hospital appointments, and
- About half are fulltime Medical Officers Special Scale (MOSSs) working under general registration, often in the slightly larger rural hospitals. These doctors contribute 80% of the total number of medical hours worked in NZs rural hospitals.[1]
- There are also a small number of specialist surgeons, physicians and emergency medicine physicians. These doctors are not included in the vocational registration initiative outlined in this paper.

Workforce problems appear most serious with the fulltime MOSSs. A third of these positions are currently vacant and more than 50% of these doctors leave their positions within 2 years.[2]

Rural hospital MOSSs exhibit many of the risk factors that were associated with complaints and competence review under the old Medical Practitioners Act 1995; they are frequently overseas graduates, transient, and general registrants.[3] They work in relative isolation and are often unsupervised. The collegial relationships designed to monitor competence are usually provided by visiting urban-based specialists. These specialists work in only part of the MOSS's scope of practice, in the different urban context, and only visit the rural hospital occasionally.

Outside of these collegial relationships there are no recognised standards of training, experience, assessment, or ongoing professional development. Indeed, the systems of clinical governance and credentialling, which are now routine in larger hospitals, often do not exist in small rural hospitals.

About half of all rural GPs work some of the time in a rural hospital.[4] While many of these doctors are vocationally registered in General Practice, they too face significant training and workforce issues. The rural GP workforce is aging with the number of GPs currently considering retirement (30%) likely to increase over the next 5 years.[5] As with the MOSS group, overseas-trained doctors form a large proportion of the rural GP workforce.[7]

Many rural GPs have significant experience in rural hospital medicine. The introduction of defined scopes of practice and vocational registration has, however, meant that much of what they practise in rural hospitals falls outside the GP scope of practice, as currently defined by the MCNZ and the RNZCGP. This is increasingly significant for younger GPs and those training in General Practice.

General Practice vocational training does not in itself equip doctors to work in a rural hospital. Locums, often vocationally trained GPs, face the same difficulties when required to cover a GP's duties in the rural hospital. Rural GPs cite the lack of quality rural CME, the need for upskilling in trauma, emergency care, and rural hospital work as issues of concern.[4]

Rural hospitals require generalist doctors (GPs and MOSSs) to work in relative isolation far from complex diagnostic and specialist backup and across a broad generalist scope of practice. The systems in place to ensure the skills and competence of this workforce should therefore be one of medicines most, not one of its least, robust.

The special relationship between rural hospital medicine and rural general practice is the first of several important issues the working party has had to consider in developing proposals for vocational registration and a training programme.

While many rural GPs want better access to training and continuing education specific to their hospital work, they wish to avoid the costs and barriers that may be associated with a new scope of practice and professional body. Many rural GPs see rural hospital work as a seamless part of the care they provide their patients. Not only is the continued involvement of rural GPs an essential part of a sustainable rural hospital workforce, continued rural hospital practice may also enhance GP recruitment and retention. Practicing the full spectrum of care has often been cited by rural GPs as one of the most positive aspects of rural practice.[6]

The Working Party has concluded that the total workforce is too small to risk dividing it between fulltime MOSSs and GPs. It is also keen to avoid the division that has occurred in Australia with the formation of a separate rural college.

For these reasons the current proposal would see the formation of a Division of Rural Hospital Medicine within the new Rural Faculty of the RNZCGP. This will allow rural hospital training to be integrated with any future rural primary care training initiatives. It is expected that the reaccreditation programme will be closely integrated with that of the existing RNZCGP accreditation programme for GPs.

At the same time if the reaccreditation process requires rural hospital doctors to undertake specified activities (that they know should be part of their professional development but that are hard for them personally to organise, such as regular time upskilling at a base hospital), then helping doctors to complete these activities will become the responsibility of the rural hospital and its managers.

By creating the right vocational training pathway for rural hospital medicine, primary and secondary care in rural areas are less likely to drift apart. It will give rural GPs options to enhance their skills, as well as offer them support and training in rural hospital practice (regardless of whether or not they choose to become vocationally registered in the new scope). It will enable rural GPs to continue practising the full spectrum of care across the primary-secondary interface to the highest possible standards.

A special relationship exists between rural hospital generalists and their specialist colleagues. A defining feature of the scope of rural hospital medicine is its breadth. Many rural hospital doctors have procedural skills that in the urban setting would belong only to specialists. To practice safely across such a broad scope, rural hospital doctors need strong and healthy working relationships with their specialist colleagues—even though this will inevitably be at a distance. For the same reasons, their new professional body will need to develop strong relationships with the specialist colleges.

The current proposal will see these specialist colleges represented directly on the Board of Studies of the new RNZCGP Division of Rural Hospital Medicine. The training programme will include a minimum of 18 months of base hospital experience at registrar level. There will also be a strong emphasis on the skills necessary to appropriately and safely transfer patients and communicate clearly and effectively with base hospital specialists. The reaccreditation programme will require regular periods of time in a base hospital.

Metropolitan specialists are likely to be concerned that district health boards (DHBs) will employ doctors trained in rural hospital medicine in urban hospitals in the place of more appropriately trained generalists such as Emergency Medicine or General physicians.

The application to the MCNZ clearly ties the vocational scope of practice to its context: the small rural hospital. Vocational registration in this branch would not allow a doctor to practise independently in a metropolitan hospital. They would only be able to do so as general registrants and thus under the oversight of a relevant specialist. Similarly it would not allow independent practice in primary care.

Finally, there is a genuine concern that the creation of this new scope of practice with its associated regulations will place a further compliance burden on the existing and potential workforce; particularly those already vocationally registered in other scopes such as General Practice or Accident and Medical Practice or who have extensive overseas training and experience. That this initiative might exacerbate rather than improve the chronic workforce shortage was carefully considered in developing these proposals.

Flexibility is the key. Involving 4 years, the training programme is relatively short. There is a high level of recognition of prior experience and training, including the recognition of the Part 1 examinations of other colleges. Some of the academic requirements of the programme are shared with the Accident and Medical Practitioners Association (AMPA) programme. It will be possible to undertake General Practice and Rural Hospital training concurrently, without increasing the total time in training.

It should remain possible for doctors to enter the field from a variety of backgrounds, train concurrently in other scopes, and (if they choose) move out of rural hospital medicine with at least part of the necessary training in another scope of practice.

Doctors currently working in rural hospitals will be able to apply to be "grandparented", as is the usual process when a new category of vocational recognition is established.

It is likely that many doctors working in our rural hospitals on a permanent or temporary basis will continue to do so as general registrants. All that will change for these doctors will be that that the collegial relationship (that they are already required to have) will be more relevant, with an accredited peer who works alongside them across the same broad scope of practice.

Conclusion

The lack of vocational registration and a career pathway for rural hospital medicine are major barriers to both the recruitment and the ongoing development of a

workforce that can practise at a consistently high standard. The Rural Hospital Doctors Working Party is confident that the proposals that have been outlined and currently sit with the MCNZ will solve many of these issues.

Notes (from page 1):

* For the purposes of the application to the MCNZ for Branch Advisory Body Status a Rural Hospital has been defined as a hospital staffed by suitably trained and experienced generalists (both medical officers and rural general practitioners), who take full clinical responsibility for a wide range of clinical presentations. While resident specialists may also work in these hospitals, specialist cover is limited to 24 hr / 7 day cover in no more than one specialist area.

** A copy of the full NZMC BAB application can be obtained from Cathy Webber at the RNZCGP (cw@rnzcgp.org.nz) or on the Rural GP Network website (http://www.rgpn.org.nz/Site/Rural_Hospital_Doctors/default.aspx).

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New hospital consultant: surviving a difficult period

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Abstract

The first years of consultant practice are amongst the most stressful in a medical specialist's career. Recognising the likely difficulties is essential if measures are to be put in place to lessen their impact. In this article, recommendations are made on how to balance clinical and non-clinical duties and to obtain the support required for professional development. Self-care of mental and physical health is vital and planning is necessary to ensure that both personal health and a work/life balance are maintained.

Specialist-based medicine is a positive career option providing for a very satisfying working life. However, in retrospect at least, many medical specialists reflect on how stressful the first years of becoming a consultant were. Some would rate the difficulties as exceeding those of the internship, when one maintained the collegiality and friendship of the same graduating class. There is now a burgeoning interest in improving the conditions and work/life balance for junior doctors and an emerging awareness of the needs of new consultants.[1–3]

The newly appointed consultant is likely to carry the self-expectation of being fully trained and ready to shoulder the new personal responsibility that goes with having your name "on the end of the bed". This may be peculiarly burdensome, given the background of the culture of medicine which assumes strength, independence, misguided omnipotence and workaholism.[4]

The new consultant's work pattern includes being alone at clinics and in private practice, and conducting ward rounds with frequently changing junior doctors, all of which can promote the sense of isolation. Contact with other consultants in the clinical scene may be infrequent and cursory, with true opportunities to discuss clinical difficulties being scarce in many busy clinical settings.

As the new consultant, full of up-to-date knowledge and drive, you will be asked to undertake any number of commitments including running departmental in-service training, hospital quality/audit committees, college work, specialist societies, teaching at all levels, start or join a private practice, continue the research themes from your previous senior registrar/MD/PhD experience, and present at and organise conferences.

We suggest a number of strategies to assist the new consultant (Table 1). Firstly, find a colleague you respect to use as a mentor (not necessarily within your department), and meet regularly by scheduling appointments.

Secondly, take your role in your multidisciplinary team meeting and not defer this to juniors. Your "team" will support you and provide you with the necessary information and feedback to perform your role. In turn you should endeavour to be inclusive, respectful, and accessible.

Thirdly, meet with your departmental clinical leader/director individually, not just at departmental business meetings. Your personal development "plan" and Continuing Medical Education (CME) requirements are a priority. It is important to carefully negotiate your clinical commitments to around 70% of your total work time.[5]

The requirements for non-clinical time are increasing, and we propose a table of likely clinical and non-clinical duties for the new consultant to consider when structuring his/her job and time (Table 2). Clearly this job-sizing is variable over the range and diversity of services but the principle of work-based time management is the key to survival during this period, because there will be many calls upon your time. Ensure there is adequate secretarial support.

Table 1. Tips for new consultants

- Get a mentor
- Support your whole team
- Preserve your "non-clinical" time
- Use a personal development plan for CME
- On appointment:
  - Ensure full orientation
  - Avoid professional isolation
  - Agree to a manageable out-of-hours/call roster
- Upskill on management/leadership training
- Value/preserve your health
- Avoid unrealistic over-commitments (saying "no" to extra demands)
- Do not believe your clinical training is "complete", identify your "gaps"

Fourthly, when you get to your new post/hospital, it is vital that you have a proper orientation programme, despite the fact that many hospitals and new consultants assiduously avoid this matter. You should familiarise yourself with relevant hospital policies, information systems, and the wider team (including managers) with whom you will be working.

In this context you must work closely with your clinical departmental head and seek their leadership and assistance in brokering arrangements. Junior consultants are often ill equipped for leadership, clinician/managerial interface issues and frustrations over resource allocation, and procuring of newer medical devices may emerge.[6] Currently many DHBs offer training in clinical leadership and management skills, and doctors should be involved.[7,8]

Finally, self-care of your mental health is vital during this period of medical practice. It is important to get a general practitioner, have a life outside medicine and put in place a personal programme to preserve your mental/physical health (Table 3). The new consultant will often be in a setting of geographical change with young children adding to recognised work stresses.

Table 2. Considering the consultant's clinical/non-clinical time (based on a fulltime post with average job size of 50 hours per week)

Activity	Hours/week
1. *Non-clinical (about 25%):*	
• Supervision (educational) of junior doctors	1.0
• Departmental Administration/meetings	1.0
• Email/mail	2.0
• Journal club	1.0
• Grand round sessions	1.0
• Formal teaching sessions (undergraduate/postgraduate)	1.0
• CME	
- Reading journals	1.0
- Audit/Quality/Protocols	1.0
- Peer review/Morbidity/Mortality meetings	0.5
• Research	1.5
• College work/community service	1.0
	12.0
2. *Direct patient clinical time (face-to-face) (about 60%): i.e. ward rounds, clinics, theatre*	
• 6 sessions of 4 hours (for example)	24.0
• Acutes	5.0
	29.0
3. *Indirect patient clinical activity (about 15%):*	
• Dictation/reading dictation (+RMO letters)	2.0
• Signing laboratory reports (+taking action on some abnormals)	0.5
• Phone advisory (colleagues/GPs/RMOs)	1.0
• Triage (OP letter/admissions)	1.0
• Meetings of the multidisciplinary teams	1.5
• Handover meetings	0.5
• Radiology sessions	1.0
• Reading up on current clinical problems	0.5
• Complaints/administration	0.5
• Reports, e.g. coroner, authorities	0.5
	9.0

Table 3. Personal health tips

• Value/preserve your health
• Have your own general practitioner
• Spend enough time with immediate relationships, family
• Do not self-prescribe
• Maintain important friendships
• Enjoy regular exercise
• Enjoy hobbies that relax you
• Beware alcohol, drugs, depression

Work/life balance plans become submerged and your personal health can be jeopardised. Indeed, the setting may be just right for the doctors' three Ds—depression, drugs (alcohol), and dimming (burn-out) to emerge[4,9]—particularly if a significant medicolegal issue arises early in your consultant years.

In summary, the difficulties of the new consultant position may be major, yet are often neglected. Recognition of the potential difficulties is essential if strategies are to be put in place to lessen their impact.[10–12]

Competing interests: None.

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Phytophotodermatitis caused by contact with a fig tree (*Ficus carica*)

José G B Derraik, Marius Rademaker

Abstract

Two arborists presented acutely with blistering eruptions affecting their forearms, hands, and fingers. The previous day, both men had pruned branches from a large fig tree, *Ficus carica*, which had sustained damaged during a storm. The following morning, both complained of a burning discomfort which rapidly evolved into erythema and bullae on skin that had been in direct contact with the tree branches. These symptoms gradually resolved over 4 to 6 weeks. Although phytophotodermatitis from *Ficus carica* has been reported, it is often poorly recognised and there is a need to raise awareness amongst arborists, orchardists, forestry workers, gardeners, and health professionals.

Plant dermatitis (phytodermatitis) is caused by a reaction in the skin following contact with certain plants or plant parts. They can be irritant such as cactus spine injuries, urticarial (e.g. from stinging nettles), allergic from plants such as *Primula obconica* or *Toxicodendron succedaneum*, or they can be phytophototoxic in nature.[1]

Phytophotodermatitis is generally a toxic reaction due to direct skin exposure to certain plants or plant parts, followed by exposure to ultraviolet (UV) light. The most common plants to cause phytophotodermatitis belong to the Apiaceae (Umbelliferae) family.[1] Other plant families that can cause phytophotodermatitis include Rutaceae, Moraceae, and Fabaceae.

Case report

Two male arborists were cleaning up storm damaged limbs from a large fig tree, *Ficus carica,* which was heavily laden with fruit (e.g. Figures 1 and 2). The work took place in Auckland between 10:30 am and noon, on a dry, relatively clear summer day (80% relative humidity, temperature 24°C, total UV exposure in 1.5 hours 3.58 mJ).

Both workers were dressed in short-sleeved shirts or singlets. During the removal of the storm damage, they wrapped their arms (predominantly their right arms) around the fig tree branches when dragging them to the wood chipper.

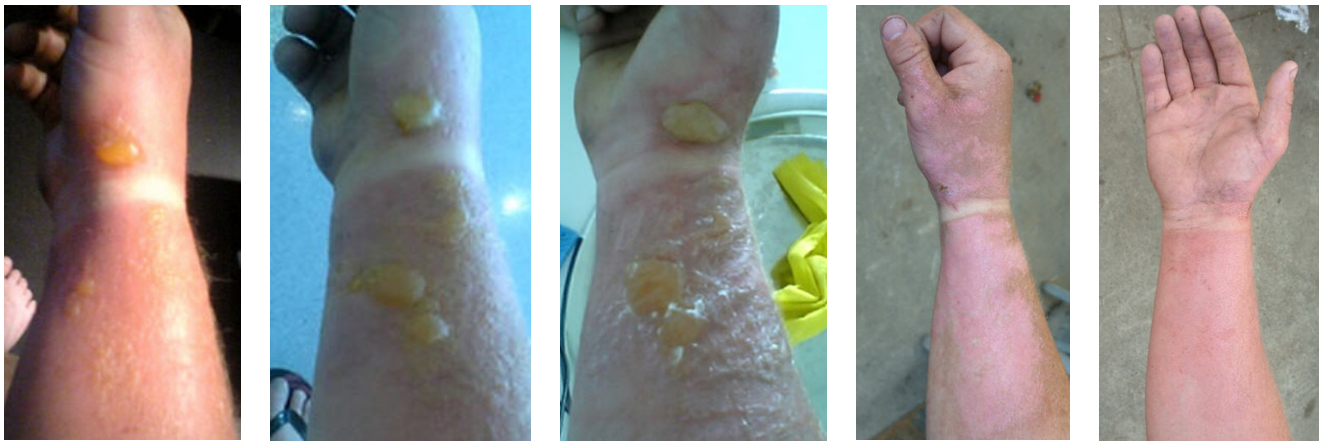
Some 9 hours later, the first arborist noted a burning sensation on his right arm, which he attributed to sunburn. However, over the next 12 hours, the skin on this arm became swollen, erythematous, and was sore to touch. Within 24 hours of contact with fig tree parts, bullae appeared on the forearm, wrist, and back of the hand

Figure 1. The fig tree, *Ficus carica* (Photo courtesy of Petr Kocna)

Figure 2. The leaves and fruit of *Ficus carica* (Photo courtesy of Petr Kocna)

Figure 3. Second arborist's forearm approximately 36 hours (A), 48 hours (B), 72 hours (C), 12 days (D), and 35 days (E) after contact with *Ficus carica* tree branches and exposure to sunlight. (Photos courtesy of Alex White and Gerald Collett)

(A) **(B)** **(C)** **(D)** **(E)**



Note: In the photos a 'ring' can be observed in the wrist region where an armband presumably prevented exposure to sunlight and the consequent occurrence of phototoxic reaction.

These symptoms on the first arborist persisted for over 2 weeks despite the use of alternative remedies, including a mixture of lavender oil and *Aloe vera* gel. As the acute erythema settled, post-inflammatory pigmentation developed, which slowly resolved over a month.

The second worker also experienced a burning sensation on his right forearm some 9 hours after working with the fig tree. Blistering of skin was noticed approximately 31 hours after contact with the fig tree (Figure 3), at which point the arborist covered the blisters and bullae with manuka honey. His condition progressively worsened with swelling and formation of large bullae on the affected arm (Figure 3). Circa 51 hours after contact with the plant, he attended the accident and emergency (A&E)

department at the local hospital. Initially the pain and blistering were restricted to the arm which had been wrapped around the fig branches—but subsequently he developed pain and swelling on the left arm, chest, and legs which had also been in contact with the fig tree. The discomfort progressed such that he was unable to work for approximately 10 days.

He responded slowly to topical corticosteroids and oral non-steroidal anti-inflammatories. The symptoms gradually resolved over 4 to 6 weeks (Figure 3).

Discussion

The history and clinical appearances was pathognomonic of a phytophotodermatitis which, in these two cases, was secondary to contact with the fig tree. Phytophotodermatitis is the interaction of plant compounds, most often psoralens, with sunlight on human skin; this results in an acute dermatitis.[1] It is usually a phototoxic reaction, as opposed to a photoallergic reaction. As a result, no prior sensitisation is necessary and anybody can be affected.[2] Other types of phytodermatitis include urticarial dermatitis, irritant contact dermatitis, and allergic contact dermatitis.

The eruption of phytophotodermatitis usually begins 24 hours after exposure and peaks at 48–72 hours. Phytophototoxicity may be amplified by both humidity and perspiration. It typically manifests as a burning erythema that may subsequently blister, and post-inflammatory hyperpigmentation lasting weeks to months may ensue. In some patients, the preceding inflammatory reaction may be mild and go unrecognised by the patient.

Phytophotodermatitis occurs most commonly in the spring and summer when furocoumarins are at their highest concentration in plants, and when UV levels are also at their peak. The incidence of phytophotodermatitis is unknown, but will vary according to the risk of exposure to psoralens. Because furocoumarins are found in a wide range of wild and domestic plants (Table 1), a variety of patient groups may become exposed.

Table 1. Examples of plants known to cause phytophotodermatitis, and the main sensitising compounds associated with them

Family	Species	Common Names	Main Compounds
Apiaceae	*Ammi majus*	Bishop's weed, large bullwort	5-MOP, 8-MOP, imperatorin
	Apium graveolens	Celery	Psoralens, 5-MOP, 8-MOP
	Heracleum sphondylium	Cow parsnip, common hogweed	5-MOP, 8-MOP, imperatorin, phellopterin
	Heracleum mantegazzianum	Giant hogweed	5-MOP, 8-MOP, imperatorin,
	Pastinaca sativa	Parsnip	5-MOP, 8-MOP, imperatorin, isopimpinellin
Fabaceae	*Psoralea corylifolia*	Babchi, scurf pea	Psoralens
Moracea	*Ficus carica*	Fig	Psoralens, 5-MOP
Rutaceae	*Citrus bergamia*	Bergamot	5-MOP
	Citrus maxima	Pomelo, pummelo, shaddock	5-MOP
	Dictamnus albus	Gas plant	5-MOP, 8-MOP

5-MOP = 5-methoxypsoralen, 8-MOP = 8-methoxypsoralen.

The two cases presented were at very high risk of developing phytophotodermatitis because of their prolonged and significant contact, high summer levels of

furocoumarins in the plant, peak summer UV levels, exposed skin, warm temperature, and perspiration.

Whilst photoallergic reactions are a cell-mediated immune response in which the antigen is the light-activated photosensitising agent, phototoxic reactions result from direct damage to tissue caused by light activation of the photosensitiser.

The main photosensitisers in plants are furocoumarins and consist of psoralens (5-methoxypsoralens, 8-methoxypsoralens), angelicin, bergaptol, and xanthotal (Table 1).[1–3]

The photochemical excitation of these furocoumarins is induced by UV radiation, usually within the UVA wavelengths of 320–400 nm (peak activity is around 335 nm).[13]

Two types of toxic reactions occur: one oxygen-independent, where the UV-activated furocoumarins bind to RNA and nuclear DNA; and the other: an oxygen-dependent reaction where the induced compounds cause cell membrane damage and oedema.[1,3–6] These reactions consequently lead to cell death (sunburnt cells and apoptotic keratinocytes).

Ficus carica, which is believed to have originated in western Asia, was brought to the Mediterranean as early as 5000 BC. In New Zealand, it is commonly cultivated as a fruit tree in home gardens and appears to be widespread in the North Island (particularly in northern regions); it can also be found in some areas of the South Island, particularly in those areas that experience long, hot summers (Melanie Newfield, personal communication, 2007).

Ficus carica belongs to the Mulberry family (Moraceae). The leaves and unripened fruit of figs contain the furocoumarins, psoralen, and bergapten, as well as the coumarins, umbelliferone, 4',5'-dihydropsoralen, and marmesin. The furocoumarins are lipid-soluble and can penetrate the epidermis with ease.[7–10]

There are a number of other *Ficus* species which may cross react with *F. carica* including Weeping fig (*F. benjamina*), Cluster fig (*F. racemosa*), and Sycamore Fig (*F. sycomorus*).

Eating figs does not cause photosensitisation, unless the juice is smeared onto the face. However, anaphylaxis has been reported after eating figs; in some of these cases, this may represent a cross-reaction with natural rubber latex.[11–12]

Although phytophotodermatitis from *Ficus carica* has been previously reported, it is often poorly recognised. As the cases reported here illustrate, contact with fig and other plant sources of furocoumarins can cause severe local reactions.

It is important that awareness is raised amongst the general public—especially those people whose occupations lead to a greater likelihood of exposure: arborists, orchardists, forestry workers, and gardeners.

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Three students exposed to *Uraba lugens* (gum leaf skeletoniser) caterpillars in a West Auckland school

José G B Derraik

Recently, Derraik has discussed in detail the public health issues regarding *Uraba lugens* Walker (Lepidoptera: Nolidae).[1] Commonly known as gum leaf skeletoniser, the caterpillars of this Australian moth feed on the foliage of gum trees (*Eucalyptus*) and other closely related plants.[2] *Uraba lugens* is firmly established in the greater Auckland region of New Zealand and, as a result, eradication is deemed to be not feasible.[1]

Most cases of harmful exposure to caterpillars seem to occur in young children, as these creatures are a source of curiosity to children due to their easy accessibility and slow mobility, and also the caterpillars' generally bright colours.[1,3] It is therefore not surprising that cases of exposure of school children to *U. lugens* are emerging in New Zealand.

This case report describes a recent incident in a West Auckland school.

Case report

The cases were observed at a school in Avondale in mid-February 2007. Three girls of approximately 10 years of age were climbing a *Eucalyptus* tree within school grounds. Girl #1 allowed a caterpillar to crawl onto her arm, which she eventually shook off. Shortly afterwards the girl described feeling a "sting" on her arm.

Girls #2 and #3 do not appear to have handled the caterpillars intentionally but were exposed to them while playing on the tree. They account that a short time after the initial stinging sensation, this turned into an itch.

Approximately 15 minutes later they were taken to the principal's office where they exhibited some distress as a result of the intense itching they described as "horrible". Although the girls appeared to be in considerable discomfort, once reassured, they were relatively calm. Note, however, that the itching did not appear to be localised, but instead seemed to have been experienced throughout the girls' bodies.

The principal observed that Girls #2 and #3 had welts (a couple of millimetres in diameter) on their skin: Girl #2 on the back of the ankles, and Girl #3 on her forearm and thigh. Girl #1 also had welts, which were located on the on the back of her hands and lower arm. The latter welt was larger than all others that were observed and it was elongated, in the approximate shape and size of the caterpillar itself. Several skin rashes were also observed, but it is not clear whether these were a result of the girls scratching themselves, or the exposure itself.

A topical antihistamine was administered to the girls, which apparently offered them some relief. Once released from school their parents seem to have offered the girls some extra care, with one girl being bathed in Dettol® (antiseptic solution) and other being treated with a traditional Pacific Island herbal concoction.

The following day all the girls appeared to have no lingering itchiness or any form of discomfort. At least Girl #1 had visible welts on her wrists after 5 days, although these were slight.

The number of cases of child exposure to *U. lugens* in the Auckland region is unknown. Based on the information currently held by MAF Biosecurity New Zealand these appear to be rare, although there has been at least one previous case of exposure of kindergarten children in Manukau City. It seems that there have also been a few incidents of children exposed while on private properties.

It should be noted that no severe adverse reactions have yet been observed, as previously pointed out by Derraik.[1] Although exposure to *U. lugens* may still cause considerable discomfort, the reactions seem to be relatively minor.

Further information on *Uraba lugens*

Uraba lugens has been the focus of a long-term management programme aimed particularly at filling current knowledge gaps and controlling the existing population.[4] Although MAF Biosecurity New Zealand is no longer directly involved in the management of *U. lugens*, further information on this pest can be obtained from the organisation's website: www.biosecurity.govt.nz/pest-and-disease-response/pests-and-diseases-watchlist/gum-leaf-skeletoniser

Finally, the author would be interested to receive information from health professionals on any reports of exposure to gum leaf skeletoniser in New Zealand.

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Laparotomy for Obstructed Labour: Uterine (Caesarean) Section

By Ernest C Barnett. Published in the N Z Med J 1907;6(24):26–8

The case was that of a woman Mrs B., of Taihape, age 33—5 children. I saw the woman first on June 23rd, she was then in full time labour. The labour pains were strong and frequent and had been so for four days previous to my seeing her.

PREVIOUS HISTORY.

Former labours easy: After birth of last child patient had a ventral fixation and amputation of the ~cervix performed in Wellington Hospital.

Examination externally: The fundus of the ‘uterus was as high up as the ensiform, no falling of the womb having occured.

Per vaginam: No sign of any external os, the presentation was a vertex one, but the head could not be felt.

The pains continued all day Monday and Tuesday, no show had been seen and the pains, though severe, were without effect. Tuesday afternoon I examined the woman under chloroform, introducing my hand into the vagina, but the most careful search even by the use of a catheter, failed to reveal any sign of dilatation, or the opening of the cervical canal.

Pains were severe and constant, temperature normal, pulse 90. Dr Whitton was called in consultation and Laparotomy was decided on. With the patient under chloroform I performed the operation that evening, June 29th, my incision reaching down as far as the fixation, the adhesions of which were tough and fibrous.

After delivery of the child and removal of placenta and membranes, the uterus was swobbed out with 1-4000 perchloride, the incision was then closed with catgut sutures, the abdominal wound with silk-worm gut.

Before closing the uterus, I passed my finger down to the internal os and attempted to find an opening, but I had to pass my other hand into the vagina and make an opening from there. For a week following the patient ran a hectic temperature, 100 to subnormal with no pain or vomiting, taking nourishment very well.

The following Monday night, the temperature suddenly rose to 104, pulse 135, due to bronchopneumonia. The next day, Tuesday July 1st, the patient was seen by Dr A. A. Martin of Palmerston North.

From the above date until July 16th the temperature varied between 104 at night and normal or subnormal in the morning, but after the 16th it became normal throughout the twenty-four hours. Just before leaving the Nursing Home the patient developed slight albuminuria.

The child, a well nourished boy, has thriven remarkably well since its birth.

Further notes on the above case by Dr A. A. Martin:

On July 1st at Dr Burnett's request I saw Mrs B. at Taihape. She was then running a high temperature, 103° to 104-5° with a rapid and feeble pulse, dry furred tongue and a sharp painful and irritating cough. There were several patches of bronchopneumonia in the base of left lung and a small patch in the middle lobe of the right side. Her temperature, cough and general "unwellness" I attributed to the lung condition. I examined the laparatomy wound. It was quite clean and healthy. The fundus of the uterus could just be felt in the hypogastrium.

A vaginal examination was negative. The cervix had been removed by a previous operation in Wellington.

Dr Barnett is to be congratulated on so successfully carrying out an important and hazardous operation. His prompt surgical interference undoubtedly saved the life of the mother and the infant. The result is the more satisfactory when one considers that the surroundings and appliances did not by any means approach the "surgical ideal."

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What is wrong with my right arm and leg?

Rayid Abdulqawi, Khaled Ashawesh, Piers Newman

A 41-year-old man presented with 2-year history of right-hand spasms and a 14-year history of clawing of the toes of his right foot when walking. He underwent three operations on the right foot with partial success. There was no history of birth trauma and no family history of note. On examination, there was no neurological deficit except dystonic posturing of the right arm and leg when walking (Figure 1A and 1B).

Figure 1A. The patient's dystonic posture of right arm on walking

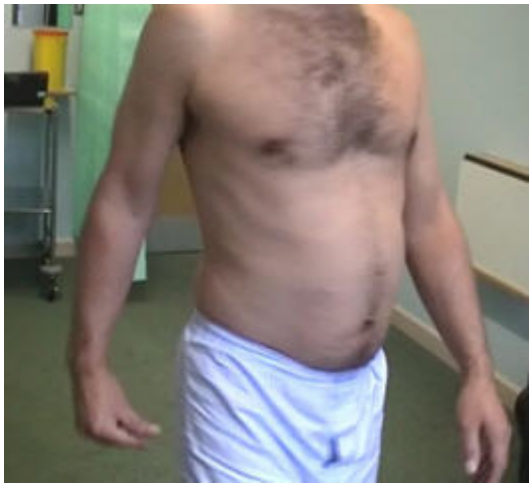
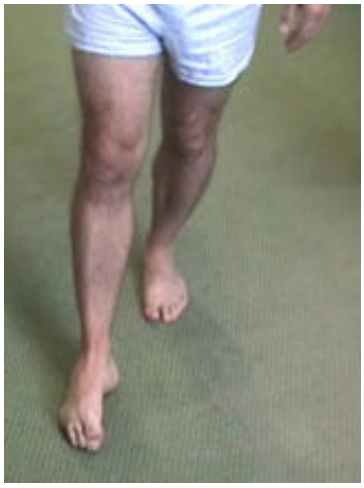
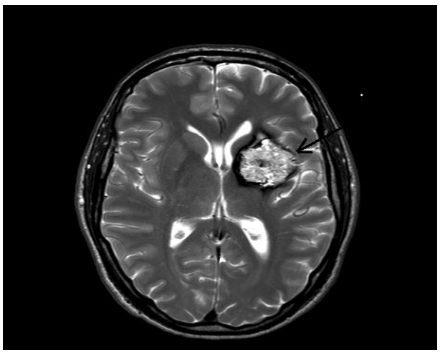


Figure 1B. The patient's dystonic posture of right leg on walking



A magnetic resonance imaging (MRI) scan of the brain revealed a cavernous haemangioma in the left temporo-parietal lobe with involvement of the basal ganglia (Figure 2).

Figure 2. T2 brain MRI showing typical popcorn appearance of cavernous haemangioma (arrow), measuring 3.2 cm, with peripheral haemosiderin bloom involving putamen, globus pallidus, head of caudate, anterior and lateral thalamus, and internal capsule



Discussion

Major causes of hemidystonia are stroke, trauma, and perinatal injury.[1] Hemidystonia secondary to cavernous haemangioma of the basal ganglia is extremely rare. To our knowledge, only one previous case is reported in the literature.[2]

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Folic acid for the prevention of colorectal adenomas—a paradoxical outcome

Apparently considerable epidemiological evidence suggests that a low-folate diet is associated with an increased risk of colorectal neoplasia, particularly in concert with alcohol, which can antagonize the metabolism of folate. Hence a trial to assess the safety and efficacy of folic acid supplementation for preventing colorectal adenomas.

After 8 years, the trialists report that, far from preventing colorectal adenomas, those in the folate supplement arm developed more adenomas than those in the placebo cohort. Clearly such supplementation cannot be recommended.

An editorial commentator points out that chemoprevention with single agents is problematic.

JAMA 2007;297:2351–9 & 2408–9

Osteoarthritis pain and the weather

Individuals with osteoarthritis often assert that change in the weather influences their pain but the evidence is inconclusive.

As someone (possibly Mark Twain) said, everyone complains about the weather but no-one does anything about it. Well the authors of this paper have done some research about it—a complex study involving 200 patients with osteoarthritis of the knee. They have related their pain and the weather.

Their results show that pain severity in individuals with knee osteoarthritis is modestly influenced by the weather. Both an increase in barometric pressure and colder ambient temperature are associated with greater pain.

The American Journal of Medicine 2007;120:429–34

Anticoagulation for venous thromboembolism—3 vs 6 months?

The British Thoracic Society has favoured the lesser option for many years but is disappointed that their advice is unheeded. Hence this randomised trial involving 749 patients in 46 UK hospitals. In each arm of the trial the subjects were treated with low molecular weight heparin and then either 3 or 6 months warfarin.

At 12 months, recurrent fatal or non-fatal venous thromboembolism occurred in 8% of patients in each treatment group (p=0.80). However major bleeding occurred in significantly more patients taking warfarin for 6 months than for 3 months (p=0.008).

The take-home message seems obvious—3 months anticoagulation is adequate and less dangerous, provided there are no known risk factors for recurrence. The message is reinterated in an editorial from across the Atlantic.

BMJ 2007;334:645 & 674–7

Purulent sputum predictive of bacterial infection in chronic obstructive airways disease (COPD)

It would seem self evident that the development of purulent sputum during an exacerbation of COPD would call for the exhibition of antibiotics. This study compares sputum culture results with culture results from secretions obtained at bronchoscopy. There was high concordance between these cultures. In addition, the patient's report on their purulent sputum also correlated well with the cultures—i.e. purulent sputum relates positively to bacterial culture reports. This study was confined to admitted patients but the lesson surely applies to those in the community—i.e. COPD exacerbation and purulent sputum is an indication for antimicrobial treatment.

Thorax 2007;62:29–35

Problems with erythropoietin?

When erythropoietin won market approval in 1988, it was hailed as a wonder drug because of the selectivity of its action and its resulting relative freedom from untoward effect.

So patients with renal failure and anaemia were saved from the hazards of repeated blood transfusion. A limiting factor, particularly in New Zealand, is the cost. But over the ensuing years, functional erythropoietin receptors have been shown to be present on other types of cells. And there have been reports of tumour progression in some recent clinical trials of erythropoietin for treatment of the anaemia associated with cancer chemotherapy.

So the Food and Drug Administration (FDA) in the US has become involved and may suggest restriction of use of erythropoietin in those with cancer.

N Engl Med J 2007;356:2445–51

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Cheaper than chicken: protein foods ranked by supermarket prices

Background to our study—Food safety scientists in New Zealand have identified chicken meat (especially fresh chicken) as the main source of human infection with *Campylobacter* in New Zealand.[1] Similarly, poultry is an important source of human infection in Europe,[2] and consumption of fresh chicken meat the leading risk factor in Denmark.[3] *Campylobacter* infection has been the most commonly reported notifiable disease in New Zealand for many years, and continues to increase.[4,5,6]

Consumers may wish to lower their risk of *Campylobacter* infection which currently estimated at around 1 in 35 per year (based on an estimate of 120,000 community cases per year[6]). Consuming frozen rather than fresh chicken is likely to reduce the risk.[5 6] But since frozen chicken still has some *Campylobacter* contamination, consumers could also reduce overall chicken consumption. Given that chicken meat is a relatively low-cost protein food, it may be useful to identify protein foods which are cheaper than chicken.

Our study's methods—The Woolworths Supermarket website[7] provided data on the three categories of fresh (i.e. "chicken whole", "chicken breast", and "chicken kebabs") and frozen (i.e. "chicken whole", "chicken breast", and "chicken nuggets") chicken products with the most items listed. The cheapest fresh and frozen items by weight, within these six categories, were identified in a Wellington Woolworths store (on 21 July 2007).

To identify potential alternatives, we searched the New Zealand Food Composition Database[8] to find food categories with items containing at least 10% protein (by weight). We then checked the in-store prices to identify the lowest cost items by weight. From all the selected lowest cost items we abstracted the protein and saturated fat levels from the product nutrition labels (on the packaging or bulk bin labels).

Results—The results indicate that 10 categories of food had at least one item that provided cheaper protein than the frozen chicken (Table 1). Two additional categories (e.g. including processed meats and seeds) provided cheaper protein than fresh chicken as well. Six of the 10 foods that provided cheaper protein than frozen chicken also had better ratios of protein to saturated fat (over 20 times better for the pulses and cereal items).

Discussion—This analysis was limited to one supermarket at one point in time. Also the range of chicken products examined was not the entire range (i.e. there were actually 572 products in the Woolworths online store that included the word "chicken"). A more sophisticated analysis could also address the complex issue of the nature of the protein quality as plant protein is nutritionally inferior to animal protein (though this problem can be minimised by combining plant protein sources within the same meal e.g. combining pulses and grains).

Table 1. Cost of protein-containing foods (≥10%) relative to the cheapest identified frozen and fresh chicken meats, in order of ascending cost per 100 g of protein (for one NZ supermarket on a single day)

Food category	Cheapest brand & details	Price ($)[a]	Weight (g)	Protein (g per 100g)	Satur-ated fat (g per 100g)	Price per 100g weight ($)	Price per 100g protein ($)	Ratio of protein to saturated fat
Nuts	*Homebrand* roasted & salted peanuts	2.99	750	24.8	5.4	0.40	1.61	5
Fish	*Homebrand* tinned tuna in spring water	2.10	425	26.4	0.3	0.49	1.87	88
Spreads	Peanut butter *Basics smooth*	4.99	1000	25.0	11.4	0.50	2.00	2
Pulses	Red lentils (BB)	0.45	100	21.0	0.2	0.45	2.14	105
Cereal	Rolled oats (BB)	0.35	100	16.0	0.1[b]	0.35	2.19	160
Fresh red meat	Fresh ox liver[c]	4.99	1000	21.2[d]	2.8[d]	0.50	2.35	8
Bread	*Signature* Highland – wholemeal bread	1.99	800	10.1	0.3	0.25	2.46	34
Milk	*Basics* skim milk powder	9.49	1000	35.5	0.5	0.95	2.67	71
Cheese	*Alpine* Colby - block	8.37	1000	23.9	23.7	0.84	3.50	1
Eggs	*Signature* 30pk, size 7	7.36	*30 eggs*	*6.9 per egg*	*1.7 per egg*	–	3.56	4
Frozen chicken	Whole chicken, *Signature*, extra-large, Size 22, (range 2.1-2.3kg)	11.50	2200	18.3	3.8	0.52	3.97[e]	5
Seeds	Sunflower kernels (BB)	0.85	100	19.3[d]	5.2[d]	0.85	4.40	4
Processed meat	*Hellers* pre-cooked sausages[f]	22.00	4000	12.0	8.0	0.55	4.58	2
Fresh chicken	Whole chicken, *Signature*, size 18, (range 1.7 - 1.9kg)	10.99	1800	18.3	3.8	0.61	4.63[e]	5
Other protein foods	Other chicken items, other protein foods (>10% protein)[g]	-	-	-	-	-	>5.36	-

[a] Prices were those listed on the Woolworths website, or failing that an actual supermarket (with all prices ignoring "specials"); [b] Increased from "nil" on the packet to "0.1" to allow for the ratio calculation; [c] The next cheapest fresh red meat was sheep's heart at $3.02 per 100 g of protein (i.e. also cheaper than the frozen chicken); [d] From the NZ Food Composition Database,[8] since there was no data on the package or bulk bin label; [e] Based on the mid-point of the chicken weight range on the packet and adjusted for the estimated 28% of the weight which is bone (based on the US Department of Agriculture Database[9]). The protein and saturated fat levels are for the edible portion only; [f] These were comprised of 66% meat with the order of listed meat types being: lamb, beef, pork and chicken; [g] Including all canned red meats, fresh and frozen fish, soft cheeses, whey protein drinks, some canned vegetarian foods, and some types of muesli. BB – bulk bin.

The results are slightly biased in favour of the cost-effectiveness of protein from chicken since it is likely some chicken meat gets discarded with bones and other unused parts of the chicken (i.e. the US Department of Agriculture's food nutrient database assumes that 49% of the weight of a whole chicken is "refuse" consisting of 28% bone, 14% skin and 7% separable fat[9]). Furthermore, we have not considered how chicken sometimes gets cooked relative to the other foods—e.g. with the addition of extra fats for frying.

Despite the study limitations, the availability of many protein sources that are cheaper than chicken (by weight), suggests that consumers can avoid chicken in their diet without cost or nutritional disadvantage. This is an important consideration for household food choices and national policy options. None of the selected alternative protein foods are implicated as major risk factors for *Campylobacter* in New Zealand (or for *Salmonella* infection as chicken is also sometimes contaminated with this organism in this country[10]). Most of the alternative foods are also less hazardous for saturated fat intake than chicken (per mass of protein obtained).

Some of the cheaper protein foods also have other nutritional advantages relative to chicken meat—e.g. for various micronutrients (especially the eggs, liver, milk, and nuts); beneficial fatty acids (the fish and nuts); and dietary fibre (the wholemeal bread, pulses, and cereals).

One study has reported better nutrient density scores for a range of foods in comparison with poultry meat (e.g. fish, organ meats, vegetables, and legumes).[11] Nevertheless, there are concerns about some of these alternative protein foods in this analysis—the high levels of saturated fat and salt in some foods such as cheese; the mercury levels in some fish species; and the environmental sustainability of fish. Even so, chicken meat may be less sustainable than some other meats in New Zealand as up to 70% of chicken feed is imported.[12]

In summary, this relatively small study provides some preliminary guidance to consumers who want cheap protein foods but without the high risk of *Campylobacter* infection associated with consuming contaminated chicken meat.

Competing interests: There was no external funding for this work. One of the authors (NW) has had two previous research contracts with the NZ Food Safety Authority (NZFSA) in 2005 and another author (MB) has provided technical advice to the NZFSA.

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Self-applied treatment of persistent plantar wart with 5% imiquimod cream

Plantar warts are predominantly caused by human papillomavirus (HPV) types 1, 2 and 4.[1] Three-quarters of plantar warts spontaneously regress within 2 years, however they can persist and may be resistant to treatment.[2]

There are currently various approaches in the treatment of plantar warts including cryotherapy, salicylic acid, cytotoxic agents, immunotherapy, surgical excision, and laser therapy. The recurrence rate following these treatments can be high due incomplete eradication of the virus from the site of infection. We describe a case of successful treatment of a recalcitrant plantar wart using 5% imiquimod (Aldara™) cream in combination with salicylic acid and duct tape occlusion.

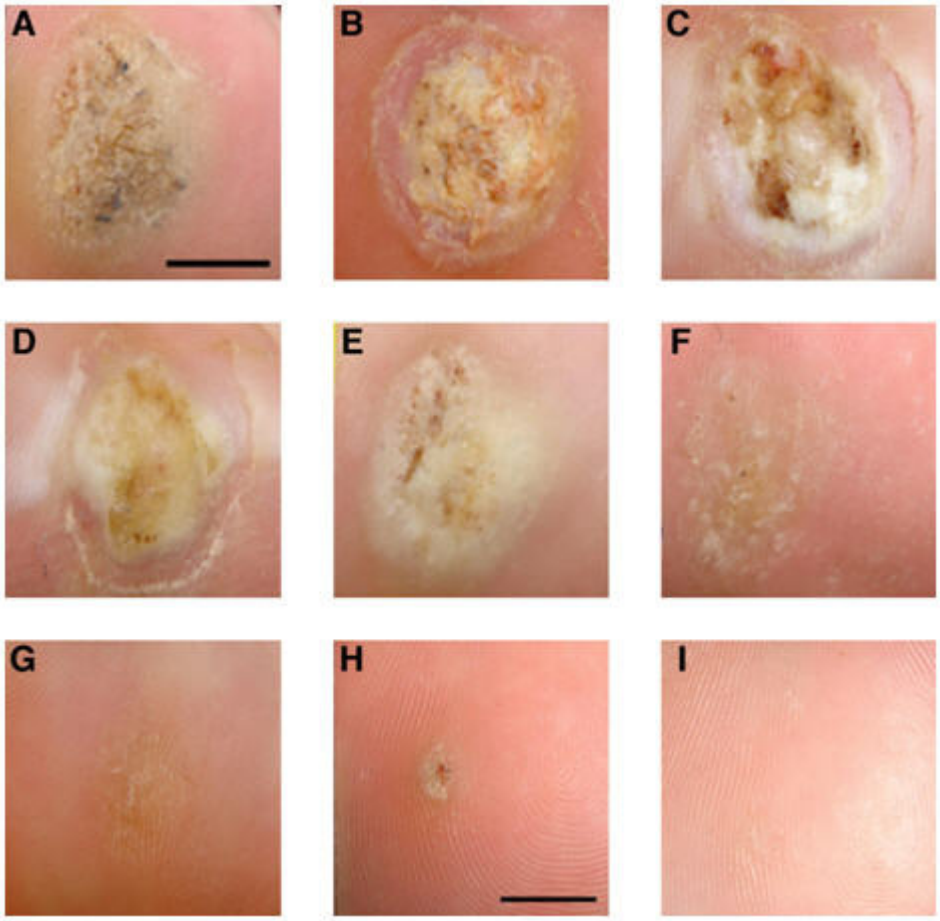
A healthy 26-year-old male presented with a 2-year history of plantar warts measuring approximately 2 cm in the longest dimension on the left foot and 0.4 cm on the right foot. The larger wart had previously been treated by cryotherapy with liquid nitrogen and with topical administration of podophyllin and salicylic acid, which were all ineffective. The wart continued to grow and caused discomfort when pressure was applied.

The treatment regimen, which was applied only to the wart on the left foot, involved initial destruction of the thickened surface skin with topical application of salicylic acid for 2 weeks. The 5% imiquimod cream was then applied daily at a dose of 12.5 mg/week for 6 weeks, with duct tape occlusion.

The patient experienced some swelling, redness, and itching at the site of infection during treatment. A reduction in lesion size was observed 2 weeks following the initiation of the treatment with imiquimod. After 8 weeks of treatment there was near complete resolution of the wart with only some flaky surface skin remaining.

Three weeks after the imiquimod was last applied, the treated wart had completely healed and the untreated wart on the right foot had spontaneously resolved. At 19-months follow-up there was no evidence of recurrence of either wart. See Figure 1.

Figure 1. Presentation of recalcitrant plantar warts. Plantar wart on the left foot (A) and a smaller wart on the right foot (H). Destruction of the thickened skin of the left foot lesion observed 2 weeks post-salicylic acid treatment (B). Lesion on left foot following commencement of imiquimod treatment: 4 days (C); 2 weeks (D); 6 weeks (E); 8 weeks (F). Clearance of the wart on the left foot (G), and the right foot (I), observed 3 weeks after completion of imiquimod treatment. The scale bar on (A and H) represents 1 cm.



Discussion

In this report, we demonstrate the successful treatment of a plantar wart with imiquimod in combination with salicylic acid and duct tape occlusion. Imiquimod is a topical immune modulator licensed for the treatment of anogenital warts. It functions through toll-like receptor-7-mediated activation of the immune.[3]

Imiquimod also stimulates migration of Langerhans cells (LC), the antigen presenting cells of the epidermis, enhancing presentation to T-cells.[4] It has been proposed that HPV-specific cell-mediated immunity is activated, reducing recurrence of warts.[5]

In this case, complete resolution of the untreated wart at a site distant from the treated infection suggests that a systemic response against HPV was activated.

Skin thickening may contribute to the recalcitrance of plantar warts to treatment. Initial tissue destruction with salicylic acid may have aided the penetration of the imiquimod into the infected tissue. In addition, duct tape occlusion has been reported to promote resolution of warts.[6] It has been proposed that duct tape induces a proinflammatory response at the site of application, increasing infiltration of immune cells, including LC.[7] This may have contributed to the efficacy of the treatment regimen used here.

Although the patient experienced a mild inflammatory response, there were no systemic or long-term adverse effects following the imiquimod treatment. Cost may be a limitation of imiquimod treatment for plantar warts, however to minimise costs, imiquimod was used sparingly and a single-use packet (12.5 mg) was used for each week of treatment.

Plantar warts are frequently persistent and debilitating and in severe cases can cause hospitalisation.[8] This study shows that imiquimod in combination with salicylic acid and duct tape occlusion can be used to treat plantar warts.

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Normal vaginal deliveries

I am writing in response to Misty Curry's plea "Would somebody please have a normal vaginal delivery?"—*NZMJ* 15 June 2007;120(1256). http://www.nzma.org.nz/journal/120-1256/2595

Like many midwives it does concern me that so few of our future doctors have the opportunity during their education to participate in a normal birth. However in my experience interns like Misty who are in it for the long haul not just for the action are few and far between! On many occasions I have been approached by students and asked very bluntly "do you have any vaginals I can come and watch". It appears that most have no interest in participating in the labour care and just want to be called once the woman is pushing so they can log a 'catch'.

I would like to reassure Misty that normal births DO still happen most of the time, but as she has discovered NOT in the big hospitals! I am an independent midwife working in a rural area and of the 40–50 women I care for each year most will have a physiological birth with only 2–3 requiring a caesarean delivery due to complications.

However unfortunately for Misty and her classmates they are unlikely to get the opportunity to attend most of these births because to achieve this high rate of physiological birth I actively encourage women who are experiencing normal pregnancies to give birth at home or in primary maternity facilities.

With this in mind I think the real issue here is not midwives keeping interns from attending normal births, but that placements for interns are not occurring in the very places that normal birth does still happen!

Misty I love sharing my passion for normal birth so if your future O&G training would allow you to get some out-of-hospital experience I'm just over the Bombay Hills so give me a call!

Sheryl Wright
Independent Midwife
www.birth.net.nz

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Prevalence of new and known diabetes mellitus, impaired glucose tolerance, and impaired fasting glucose

Sundborn et al reported a prevalence of known diabetes of 6.7% known diabetes and a further 2.6% with newly diagnosed diabetes,[1] giving a total prevalence of 9.3% for the Auckland population. This was a rigorous cross-sectional, population-based study and indicates a far higher burden of diabetes in the New Zealand population than previous Ministry figures, based on inadequate data had suggested.[2] It also approximates far more closely to the prevalence figures for diabetes reported in Australia.[3]

Other mortality data (quoted by Sundborn et al) originating from the Ministry of Health,[4] predicting around 1700 deaths due to diabetes in the year 2006, are also likely to be a gross under-estimate of the true picture, especially given that the reliability of death certification in New Zealand has been brought into question.[5,6]

In particular, we showed in a retrospective review of 600 death certificates (during 1999) that 104 cases (17%) had previously documented diabetes, of which only 47 (45%) had diabetes recorded on either the death certificate or the coroner's report.[5] In addition, we found that there were 159 (32%) of those people not known to have had previous diabetes that showed a highest random plasma glucose ≥11.1 mmol/L in their clinical records.[5]

Similar findings have also been reported from Otago[6] where diabetes was mentioned on only 55% of death certificates. Furthermore, in our study,[5] we found 24 cases with vascular disease cited as the primary cause of death for which diabetes was not recorded at all on the death certificate despite being present.

It is remarkable that diabetes is often not recoded as a contributory cause of death in vascular cases, despite the fact that New Zealand Guidelines cite diabetes as a cardiovascular risk factor.[7] If we take this under-estimate into consideration against Ministry figures,[4] then there would be more than 3500 deaths where diabetes is present during the time period in question.

Ministry figures may be flawed, but we are pleased to see the emergence of data[1] that give a more representative indication of the true burden of diabetes in New Zealand.

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PHARMAC's funding of 9 weeks Herceptin: many assumptions in a high-risk decision

Metcalfe et al[1] recently presented in the *Journal* the reasoning behind PHARMAC's decision to fund 9 weeks of trastuzumab (Herceptin®) as adjuvant therapy for early stage HER2-positive breast cancer.

They state that 'the evidence for the 9 week concurrent regimen was sufficient to justify funding' and put this 9-week regimen forward as the standard of care for the treatment of women with early stage HER2 positive breast cancer in New Zealand. By contrast, regulatory authorities in 23 other OECD countries have instead accepted 12 months of therapy as standard treatment. We strongly maintain that there is currently insufficient evidence to be confident a shorter duration of therapy offers equivalent benefits.

The use of Herceptin as adjuvant therapy for HER2-positive breast cancer has been heralded as a major advance in breast cancer treatment. The benefits, as described by Metcalfe et al, are documented by four large international studies[2–4] of adjuvant Herceptin which recruited >12,000 patients in studies of 12 months Herceptin treatment duration, while there have been two small trials looking at 9–10 weeks of therapy using a non-standard chemotherapy regimen.

One of the short duration trials,[5] described by Metcalfe et al, was a cardiac safety study that was not designed and had too few patients to assess efficacy. Furthermore, of the 227 patients in this trial, only 157 were HER-2 positive on central review. Thus the only published short duration trial of treatment efficacy is FinHer,[6] which enrolled 232 patients in total, with only 54 in the PHARMAC-mandated New Zealand treatment arm of Herceptin concurrent with docetaxel.

This study lacks the statistical power in its own right to be used as the sole justification for a standard of care, and PHARMAC's assessment of its significance relies heavily on the findings from 12 month studies and an implied, but far from proven, assumption that 9 weeks has equivalent efficacy.

The large 12-month duration studies have all shown not only statistically significant disease-free, but also overall survival benefits at a remarkably early stage. This pattern of benefit has now been maintained at 4 years follow-up in two of the large studies,[2] predicting persistent and potentially curative benefits—as seen with other adjuvant systemic therapy studies of tamoxifen and chemotherapy at 15 years follow-up.[7]

These findings emphasise the efficacy of Herceptin when given in a 12-month schedule. Critically, and most disappointingly, Metcalfe et al do not discuss the fact that all the published 12 month trials have shown statistically significant overall survival benefits, while the 9 week FinHer trial did not.

Overall survival benefits are still recognised as the 'gold standard' of oncological treatment efficacy. Put bluntly, New Zealand women are to be denied a treatment regimen which has been robustly shown to save lives and are instead to be offered one which has not.

Metcalfe et al make much of the apparent waning of benefit with longer follow-up in the sequential HERA study,[3] but the benefits remain large at 2 years median follow-up and are supported by an overall survival benefit.

Smith et al have calculated that the chances of the disease-free survival results losing significance are <20%.[3] The primary concern regarding maintenance of the survival differences in that trial is that many patients in the control arm of this study are now crossed over to Herceptin, following the reporting of the 1-year follow-up statistics. If there are any uncertainties over the durability of the disease-free and overall survival benefits of 12 months therapy, these uncertainties must be many-fold higher for the 9-week therapy (FinHer) that has not been demonstrated to have a mortality benefit.

Metcalfe et al also discuss minor methodological concerns in the different studies. In fact such analyses highlight the serious imbalance between the Herceptin and chemotherapy-alone arms in the FinHer study, including the findings that the patients in the non-Herceptin arms had more often less favourable characteristics, like larger tumour size, more grade 3 tumours, more oestrogen receptor-negative tumours and a slightly younger median age.

Further scrutiny of the FinHer study shows that only 33 of the 54 assigned to docetaxel with Herceptin actually received the protocol chemotherapy. This further highlights the risks associated with basing a funding policy on the results of a single small study and ignoring the results from a number of much larger trials. It is ironic that PHARMAC argue against a 12 month therapy, partly because they state the true long-term effects of 12-month therapy may never be known. In fact, this is because it is now viewed internationally to be unethical to continue studies where there is a control patient group without 12 months Herceptin treatment.

We remain perplexed as to why the Medical Advisory CaTSoP committee of PHARMAC was told that 12 months was not an option when considering their recommendations. We note that CaTSoP expressed a strong preference for 12 months of therapy, but were given the option of 9 weeks or nothing. Subsequent to the CaTSoP adjudications the evidence supporting 12 months treatment has further strengthened as studies continue to collect more follow-up data.[2–4]

Why, with CaTSoP's stated preference for 12 months of therapy did PETAC, the PHARMAC Board, and the DHBs not actively pursue additional funding from the Health Minister, rather than try to restrain spending within their current budget?

Health care is becoming inevitably more expensive with many other new drugs and technologies becoming available and offering potentially significant benefits. Beyond the issue of cost, if we do not adopt proven new therapies the quality of our care will inevitably fall further in comparison to other countries, at a time when our take up of new therapies is well behind most other OECD countries.[8]

In this setting, it is disappointing that PHARMAC appears unable to weigh up the narrow (drug costs), short-term fiscal imperative against available research evidence

together with the wider and longer-term health care costs, in a logical, systematic, and transparent fashion.

A major role for PHARMAC here, which has proven to be very effective in the past, should be to push for optimal pricing of Herceptin by negotiation with the pharmaceutical industry.

It is clear that Herceptin improves the outcome of patients with early stage HER2-positive breast cancer, but the current international standard of care remains 12 months of therapy. PHARMAC have now introduced a regimen for funded treatment on the basis of very poor evidence, which is one small trial with serious statistical concerns, in preference to regimens supported by robust clinical trial data.

While PHARMAC have a difficult job in balancing pharmaco-economic benefits of treatment, we believe in this instance they have placed too little weight on compelling scientific evidence. The risks they have taken with their decision to fund 9 weeks of Herceptin are not so much with their limited budget, but much more significantly with patients' lives.

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THE NEW ZEALAND MEDICAL JOURNAL
Vol 120 No 1259 ISSN 1175 8716

Criticism of the decision not to fund the HPV vaccine for pre-adolescent females in New Zealand, and Response

Letter from two concerned doctors—We were very disappointed when (in the recent Budget) the Government failed to fund HPV vaccine for pre-adolescent females, contrary to the scientific advice of the Ministry's advisors.[1]

It was not clear from the Minister of Health's statements whether the decision not to vaccinate was simply financial or whether there was a scientific basis for his decision. It now appears that individuals within Ministry of Health have chosen to ignore its own scientific advice.

The Ministry's very own Director of Cervical Cancer Screening has penned an article, published in *Screening Matters*,[2] supporting the Minister's decision not to fund the vaccine. The Director pointedly lists the gaps in knowledge about HPV vaccines, but in our opinion, fails to weigh the evidence in a way that presents a balanced scientific view.

We consider it ironic that it is the Ministry's very own Director of Cervical Cancer Screening that has adopted a position contrary to its own Immunisation Scientific Advisory Committee.

The Cervical Screening Director's article lists five key gaps in knowledge that she considers argue against funding the vaccine for pre-adolescent females at present.

Firstly, she reminds us that the duration of protection and the requirement for booster doses is not known. This is true—however there appears to be very promising stability of the immune response, and protection has already been observed for 5 years following vaccination.

We are then reminded that the relative contribution of HPV genotypes to cervical cancer in New Zealand is not known. However she conveniently fails to commit the Ministry to funding the vaccine should the attributable risk of HPV types 16 and 18 reach a critical threshold.

Then there is the red herring of male vaccination. Whilst vaccinating boys may "break the cycle of transmission"[2] the likely additional cost/benefit of vaccinating males in addition to females is likely to argue strongly against male vaccination until vaccine costs reduce substantially.[3] This is not an argument for denying vaccination to girls now.

And what about genotype replacement? Will the beneficial effects of reduction in infection by types 16 and 18 be offset by an increase in infection rate by other oncogenic HPV types?

We accept that if genotype replacement does occur, then it may be necessary to change the formulation of the vaccine. But it is important to note that this effect, if it were to occur at all, can only be observed if the vaccine is widely used.

Finally, it is declared that the effect on the cervical screening programme is not known and it will not be until the vaccine is in use. Surely this is a communication and education issue rather than an excuse not to introduce the vaccine at present.

In our view the Ministry of Health's Cervical Screening Directors' public stance is short-sighted and disappointing. It is important to note that there are gaps in our knowledge about all vaccines, including those currently in routine use. This is not a reason to withhold them from the population but rather emphasizes the importance of careful post licensure surveillance of all the effects of vaccination.

Every year of delay means approximately 30,000 women cannot benefit from receiving an HPV vaccine. Who is the Ministry really advocating for; treasury or women?

Dynes McConnell and Stewart Reid
Ropata Medical Centre
Lower Hutt

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Letter from the Immunisation Advisory Centre—It is with disappointment that we read the article by Dr Hazel Lewis published in the July 2007 newsletter of the National Screening Unit on the potential impact of mass HPV vaccination (*Screening Matters*, Issue 10, July 2007).

Aspects of this article appear to be more around individual opinions and not supported by the scientific evidence. A few particular points we wish to highlight:

First, the comments in this article around "the potential to increase inequities" have missed a crucial point in the consideration of introducing HPV vaccine: Universal immunisation programmes have the potential to reduce inequity gaps significantly. This has been demonstrated in New Zealand following the reduction in the inequity gap in the meningococcal B epidemic programme.[1] Leaving HPV vaccines on the private market, and not on national programmes is much more likely to increase inequities.

Second, the statement that "long-term safety of these vaccines can only be determined from surveillance over many years" is misleading. There is already extended follow-up to 5 years' surveillance safety data on HPV vaccines in clinical trials, showing an excellent safety profile.[2] Safety surveillance is continuing, however 5 years' surveillance data is already considerably better safety data that any pharmaceutical and vaccine data we are aware of prior to licensure.

Third, the comment "reductions in the rates of smear abnormalities and colposcopies will make it more difficult for laboratories and colposcopists to maintain quality" is extraordinary. Are we going to avoid introducing an effective vaccination programme so we can keep other parts of the health establishment in business? Furthermore it is extremely unlikely that colposcopists will go out of business.

With the current rate of 27,000 colposcopies in the public sector (Data provided from The National Cervical Screening Programme – July 2007) and an unknown amount in the private sector, an estimate in a reduction of approximately 50% in smear abnormalities requiring intervention[3] will clearly still leave a great deal of work for colposcopists nationwide.

Fourth is the issue around the likelihood of ecological replacement. While seroreplacement is always a theoretical possibility to bear in mind, there are a range of reasons and evidence to date as to why this is unlikely to become a problem. Viral type replacement requires two conditions to be fulfilled. These conditions are:

- The existence of partial competition of different types during natural infection, and
- No cross-protection from the vaccine against types competed against.

Evidence to date indicates that the former is not fulfilled in the case of HPV and preliminary data from efficacy studies have found varying degrees of cross-protection.[4]

The article implies that introducing universal HPV immunisation will somehow be detrimental to cervical screening services. Surely the objective of decreasing the burden of cervical abnormalities and deaths within our communities must be the priority.

HPV vaccination can protect against two types of HPV that lead to approximately 70% of cervical cancer. Hence vaccination will not prevent the need to continue to screen for (and treat) cervical abnormalities.

A well-delivered universal immunisation programme, however, has the potential to reduce the persisting equity gap for those underserved by the current screening programme.

Both screening and immunisation should be apolitical, receiving the full and energetic support of all our health services to improve women's health.

Nikki Turner
Senior Lecturer

Helen Petousis-Harris
Senior Lecturer

Natalie Desmond
Advisor (Research and Communications Department)

Immunisation Advisory Centre, University of Auckland, Auckland

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NZMJ Note: Both the above letters were received by the NZMJ separately and then combined due to the common response (below).

Response—Potential impact of mass HPV vaccination on cervical cancer prevention: reply to Turner et al and McConnell et al

The purpose of the National Screening Unit's newsletter, *Screening Matters*, is to inform practitioners and the community about developments in screening. The article on the potential impact of HPV vaccination on cervical screening, which appeared in the July 2007 edition[1] raised a number of issues for debate, particularly around possible operational issues for screening, and should be read in this light.

This letter is a response to the six issues mentioned in the article that Turner et al and McConnell et al have chosen to highlight in their letters to the editor.

Like any preventive technology, HPV vaccination has the potential to cause harm at the population level while providing a degree of protection to individual women. This risk can be avoided by careful design and implementation of a strategy that effectively combines screening with vaccination.[2]

The key will be to ensure that vaccination does not lead to a reduction in the participation of women (especially disadvantaged women) in regular screening. Provided current rates of participation can be sustained, if not further improved, the combination of screening plus vaccination could provide an additional advantage over screening alone.

Sustaining high screening participation rates among immunised women will be no easy task. Rather this will require ongoing, costly, and highly innovative health promotion strategies.

Turner and colleagues correctly point out that universal vaccination programmes can narrow, rather than widen, inequalities. The reality, however, is that coverage of most vaccines has generally tended to be higher in more privileged groups, so worsening inequalities. This issue will need to be carefully addressed in the design of any HPV vaccination programme.

The 4–5 year safety profile of HPV vaccines appears reasonable to date. This does not, however, negate the need for post marketing surveillance to ensure that delayed adverse effects do not emerge with wider use over a longer period of time.

The importance of the impact of HPV vaccination on cytology laboratory and colposcopy quality is one we need to raise now so we can be thinking collectively about how to address it. It is widely accepted that quality declines with falling volumes. Minimum volumes are a core quality standard for the National Cervical Screening Programme.[3] Again, this issue will need to be carefully managed as universal HPV vaccination will lead to declining rates of abnormal smears.[4]

The risk of ecological replacement (i.e. that oncogenic genotypes of HPV not included in the vaccine may become more prevalent over time) is low. The point made in the article is that this needs to be monitored. The NSU hopes to shortly commission an HPV prevalence study to provide a baseline for such surveillance.

McConnell and Reid point out that protection from vaccination has been shown to persist for up to 5 years. Again, this does not negate the need for studies to monitor long term duration of protection. Such studies will determine whether booster doses are needed and if so when.

McConnell and Reid acknowledge that vaccinating boys will be necessary to "break the cycle of transmission", but doubt whether male vaccination will be cost effective. This was precisely why this point was raised in the *Screening Matters* article.

Contrary to the statement made by McConnell and Reid in their letter, the *Screening Matters* article does not contradict the advice of the Immunisation Technical Working Group. Rather the article raises important operational issues that will need to be addressed as we move from a pure screening era to a screening plus vaccinating strategy. All novel medical technologies—whether drugs or vaccines—require robust but rational debate if they are to be wisely used.

An additional and very important issue not addressed in the *Screening Matters* article is whether the extra benefit of screening plus vaccinating over screening alone can be achieved cost effectively.

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National Screening Unit
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Wellington

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THE NEW ZEALAND MEDICAL JOURNAL
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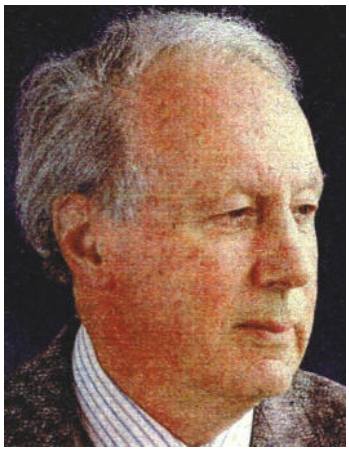
Richard Nichols Akel

10 April 1916 – 14 December 2006

Richard Akel loved flying, golf, and the land—and as a doctor and paediatrician he touched many Eastern Bay of Plenty lives. During his time in Whakatane he made an amazing contribution, not only to medicine but to a huge range of other organisations and causes.



Two of his three children, Elizabeth and William, describe their father as a "gentle and unassuming man, who always saw the positive side of things…his passion was for the education and health of children; many people were recipients of his kindness and generosity. He didn't blow his own trumpet. He was from a generation of service."

Dr Akel grew up in Wellington where, after attending Wellington College with his two brothers, he studied medicine for 5 years at Otago University. He returned to Wellington and the capital's hospital for his final training year.

Dr Akel then moved to New Plymouth, where, in 1943, he met his future wife, Lillian Livingston, a trainee nurse at the hospital.

That same year, Dr Akel joined the New Zealand Air Force. He spent 3 years as a medical officer overseas on Norfolk Island, and at the air force base at Mt Maunganui. He moved to Whakatane after the war.

In Bee Dawson's book *Spreading their Wings – New Zealand WAAFS in Wartime* nurse Judy Fraei, nee Giles, remembers Dr Akel's generous nature while he was posted on Norfolk Island: "We had to wear uniform, both Joe and me. I held a little prayer book. The other nurse, Mae Speedy, was my bridesmaid, and Doc Akel gave me away."

After arriving in Whakatane in 1946, Dr Akel first worked out of a surgery in Commerce Street. Daughter Elizabeth clearly remembers the little building: "It was an old-style house, white with a red roof. It was right between the old garage and the McGlashen's car yard."

The family then bought a house on Domain Road from Dr Freddy Appleby and for a time Dr Akel worked from the front room. Later, he bought the house next door.

During this time the baby boom was in full swing and Dr Akel travelled to Murupara, Nukuhou, Kutarere, Matahi, and Waiotahi to deliver them.

According to William, "He travelled great distances and had a penchant for big, fast American cars. It was quite funny as he usually didn't like a lot of show."

Dr Akel developed a real love for all his patients and his children say he knew all the farmers in the district and they would wave to him as he drove by.

In 1953 Dr Akel went to England to undertake postgraduate studies at Great Ormond Street Children's Hospital. He was in the crowd during the Queen's coronation.

His medical involvement in Whakatane was immense. He worked for about 20 years at Whakatane Hospital as a paediatrician, was in charge of the children's ward for a time, helped to train nurses, was a patron of the Stroke Club, and was the police and ACC doctor.

He served as president of the Bay of Plenty branch of the New Zealand Medical Association for two terms and was secretary for a time. He was also the honorary Agricultural and Pastoral Show doctor for many years.

Aviation was a passion and his interest in aero medicine meant he provided medical care for air force personnel and civilian pilots in the area.

Dr Akel's wife suffered a heart attack in 1993 and passed away shortly after.

Aside from his medical involvement, Dr Akel was a member of the tennis club, swimming pool, and squash club committees; the Apanui School PTA; IHC; and Rotary—and he helped establish the Wine and Cheese Society.

He was a keen sportsperson and loved golf. He played until he was 88, and he was passionate about rugby. He also loved the land, especially dairy farming and agriculture—an interest he developed through his wife, a farmer's daughter, and his association with farmers.

Dr Akel donated a block of land to Edgecumbe College for horticulture, one of the first to be accredited in New Zealand, and he established a family orchard at Edgecumbe which he enjoyed visiting in later life.

This obituary was adapted slightly from the version that appeared in the *Whakatane Beacon* newspaper. We are grateful for their permission to reprint this obituary in the *NZMJ*.

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Philip Matthews Goodrich

5 June 1922 – 27 April 2007

Alcoholics Anonymous (AA) became Philip Goodrich's family. An alcoholic himself, he was twice "struck off" as a doctor, once in England and once in New Zealand, but was re-instated both times. In each case, his misdemeanour was related to excessive drinking.

The English doctor arrived in New Zealand alone, in 1974. After beating his alcohol addiction, he dedicated himself to working with AA. His tireless efforts endeared him to hundreds with alcohol and addiction problems.

At his packed funeral service in Christchurch, one AA member after another rose to pay tribute to "Dr Phil", their valued helper and adviser. Goodrich's ashes were later sent to England for interment in his home village of Steyning, West Sussex.

Colleague Dr Norman Walker says Goodrich was probably unique in having been "struck off", then later re-instated in England and New Zealand. Walker said Goodrich became a fervent AA member and a rehabilitation patient at Sunnyside Hospital. "He made a good recovery." He lived the last 28 of his 84 years without a sip of alcohol.

Born and raised in England, Goodrich studied at Queen's College, Cambridge, and at London's St Bartholomew's Hospital. After completing his medical training in Chichester, he joined the Royal Air Force and served with its Air Ambulance, based in Hong Kong.

During the Korean War, Goodrich was involved in missions to bring wounded troops from conflict zones to Hong Kong for hospital treatment. He changed course in 1953, returning to England to specialise in obstetrics and gynaecology. He qualified in this field but then turned to general practice, owning and leading a medical centre in his home county of Sussex. He worked there from 1957 to 1974, acting also as an examiner for Civil Aviation.

Goodrich married Jeane Oxlade, who had two children from a former marriage. The couple separated and Goodrich moved to New Zealand. Although Jeane never visited New Zealand, they remained on good terms.

He worked first as a GP in Christchurch. Then, while recovering from alcoholism, he became involved in Sunnyside Hospital's Mahu Clinic. He began working with fellow patients and, when a medical position at the clinic became available, it was offered to him. "We were very pleased to get him re-instated," Walker said.

Goodrich worked at Mahu Clinic from 1978 to 1987, first as a registrar and then as a medical officer. Walker says he gave exemplary service there, but his greatest contribution was to AA. "He was a great force in AA. His contribution to AA was his strong point. He helped a lot of people. He went out of his way to help alcoholics. AA was his recovery and his family."

Friend Tony McCaffrey says Goodrich was "committed to AA and totally dedicated to the service of others".

Retiring from Sunnyside in 1987, Goodrich became medical officer to the newly formed Vincentian Recovery Centre, which was run by a charitable trust in Christchurch.

McCaffrey says Goodrich maintained a high work rate, driven by his desire to help rehabilitating alcoholics. He became involved also in advising on developments in alcoholism and drug addiction. "He was at the leading edge of work in the area of binge drinking. People involved in drink-driving were often referred to him by the courts. He was very open-minded, even into advanced age. He studied alternative theories, such as rebirthing, into his 80s."

Goodrich was active in many medical and professional groups, especially those connected with alcoholism. He was a member of a committee that offered advice and services to fellow doctors with health and addiction problems.

Walker says Goodrich was "a decent person, a nice fellow, a good chap". He lived a quiet life, with little involvement in the wider community. However, he demonstrated a pleasant singing voice in his membership of a neighbourhood choir. He had a gift for words and was sought-after as a speaker.

Mike Crean wrote this obituary; it appeared in *The Press* newspaper (Christchurch) on 12 May 2007 under the heading *Untiring in his help for alcoholics*. We are also grateful to Bruce Rennie of *The Press*.

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Geoffrey Noel Ferner

25 December 1924 – 28 May 2007

The Depression years made a deep impression on Geoffrey Ferner, in spite of a comfortable upbringing. His father, an Auckland lawyer, was mayor of Mount Albert, so his family was often approached by the desperate. The Depression also gave him an excuse to go barefoot to school, as nobody else had shoes.

In 1936 his father was appointed magistrate in Greymouth, where Ferner continued his schooling. On the outbreak of war, in 1939, the family moved to Whangarei.

He studied medicine at Otago University, moving to Christchurch for his final year in 1947, as his family was living there. That year he received the Ardagh Memorial Prize for the best student. On graduating, he served at Christchurch Hospital as a junior, then as a senior house surgeon.

He had met Margaret at Otago University and they were married in Stoke, Nelson, in 1949.

In 1950 Ferner was posted to the coal-mining town of Kaitangata, near Balclutha, as a special area doctor to complete his medical bursary obligations. He delivered most of the local babies at the Kaitangata maternity home and learned to love obstetrics.

The family moved to Christchurch in 1953 and Ferner set up in general practice in Papanui. He continued his interest in obstetrics and, in 1960, achieved the new New Zealand Diploma in Obstetrics. Pressure on him eased when he took a partner in about 1958. His interest in obstetrics continued and, at his peak, he was delivering 100 babies a year.

When the demands of obstetrics became too great, he turned his interest to the causes of infertility. At the age of 55 he went to England to study for his Diploma of Venereology and was a venereologist with the Canterbury Hospital Board until compulsory retirement at 65.

In 1991 he was made a Fellow of the Royal New Zealand College of General Practitioners. He remained an active family doctor until his retirement in 1994.

Ferner's practices was typical of many at that time. It was obstetrics-based and personal. It involved home visits as well as evening and weekend duties. GPs worked long hours, so what leisure time they had was very precious.

In spite of his busy professional life, Ferner had many other interests. Soon after coming to Christchurch he took up gliding and earned the right to fly solo. As usual, enthusiasm took over and soon he and a group of friends were building their first plane.

Many summers followed in "luxurious" settings at Omarama, which at that time offered dust, wind, and heat to the families. His adventures included circling the summit of Mount Cook with friends and being in the team that rescued a fellow glider pilot from the top of Mount St Cuthbert at Omarama.

He was president of the gliding club for a term and editor of *The Gliding Kiwi* magazine for several years. And because of his interest in aviation, he was appointed a Designated Medical Examiner in 1971—responsible for mandatory medical checks of senior pilots.

Rebellion in the family ended the gliding days so the next enthusiasm was for jet boating. He bought a clapped-out jet boat with a friend, Dr Tony Goldstein. They were kindred spirits being compulsive tinkers and perfectionists, with no idea of time. The pair spent a year of late nights getting it ready for action. They then negotiated many South Island rivers. Many ingenious repair jobs were done by the side of the river to get them or other boats home.

After teaching advanced first aid to Red Cross people, Ferner became doctor for the Red Cross Disaster Relief Team in 1969. Here he took part in exercises designed for people 20 years younger than he was. Eventually good sense prevailed. He served for 3 months in Vietnam with a Red Cross team in 1973.

Ferner played a weekly game of golf with a friend, skied in the winter, and water-skied in the summer with his family. Through his family involvement he learned to love classical music. He served as president of the School of Instrumental Music when his children were small, and for several years was a vice-president of the Christchurch Symphony Orchestra.

He had a close relationship with the Papanui Anglican Parish of St Paul’s and was a trustee of Life Education Trust Canterbury for several years.

Geoffrey Ferner was a fine example of the GP of the latter half of the 20th century. These folk will not be replaced, as their style of practice does not fit the current mould. In effecting change we might well have lost a great deal. In particular, the passing of the GP obstetrician has severed the bond between doctors and families.

Geoffrey Ferner served his practice and profession well. He is survived by his wife Margaret, daughter Barbara, and sons Tony and John.

This obituary is based on *The Press* obituary entitled *Versatile doctor liked a challenge* (23 June 2007). Roy Holmes adapted it after discussion with Margaret Ferner.

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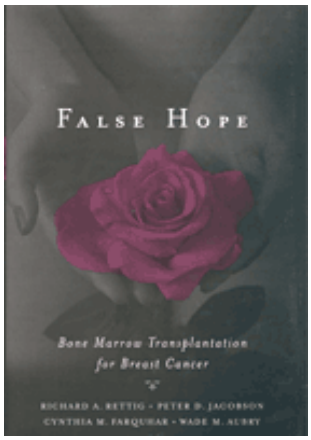
False hope: bone marrow transplantation for breast cancer

Richard Rettig, Peter Jacobson, Cynthia Farquhar, Wade Aubry. Published by Oxford University Press, 2007. ISBN13: 9780195187762. Contains 368 pages. Price US$49.95

This book is a compelling cautionary tale of how a medical intervention (high dose chemotherapy and autologous bone marrow transplant [HDC/ABMT]) became widely utilised for women with breast cancer ahead of any substantive evidence of benefit.

Up to 40,000 women (predominantly in the USA) may have received this procedure over a 12-year period between 1989 and 2001. Perhaps only 2–3% of these procedures were conducted within one of the handful of phase III clinical trials which would eventually demonstrate its ineffectiveness.



"False Hope" is a meticulously researched case study of clinical medicine "jumping the gun" by introducing an expensive and toxic procedure in response to the desperation of women (and their families and physicians) with metastatic and high risk early breast cancer. The book chronicles the alignment of irresistible forces which drove HDC/ABMT into clinical practice on the back of hope, clinician beliefs and evidence extrapolated beyond legitimacy. One of the main themes of the book is the struggle by proponents of an Evidence Based Medicine (EBM) approach against the overwhelming forces which led to the proliferation of this treatment.

This mismatch is clearly described as the views of EBM “experts” were cast aside in favour of the opinion of individual clinicians and the emotional appeal of individual patients and their cases.

The authors chose to adopt the methodology of a case study (not always valued in EBM circles) to tell their story. The resounding “success” of their account is a reminder to EBM purists that even they need to tell stories to communicate and illustrate their arguments.

The book is the combined effort of 4 authors and is divided into 4 parts: Initial Conditions, Drivers for Clinical Use, The Struggle for EBM and The Significance of the Story. This leads to some inevitable repetition of facts and viewpoints but there is remarkably little variation in narrative style given the different backgrounds of the authors. There is extraordinary detail in parts (especially the sections on the court cases and the utilisation data on the HDC/ABMT procedure) which underlines the extensive research effort the authors undertook.

“Initial Conditions” outlines the clinical and societal background on which the story begins. The second section “Drivers for Clinical Use” recounts the court trials and litigation strategies (generally patients & families vs Health Insurers refusing to fund HDC/ABMT) which culminated in the extraordinary awarding of $US89 million

(including a swingeing $77 million in punitive damages) in the *Fox vs HealthNet* case. The role of Entrepreneurial Oncology is outlined and is summarised later in a wonderfully understated way by a quote from Gabriel Hortobagyi: "Enthusiasm overtook discipline…"

"The Struggle for EBM" details how technology assessments cautioning the lack of evidence for HDC/ABMT were largely ignored until the tide slowly began to turn as the evidence (of ineffectiveness) from phase III trials began to emerge. Details of the case of scientific fraud by Dr Bezwoda from South Africa provided a colourful distraction as the "fall from grace" became complete.

The authors conclude (The Significance of the Story) by promoting their own solution to the deficiencies in the USA regulatory, scientific, insurance and clinical settings which allowed this unfortunate chain of events to unfold. They suggest that a future collaboration between the medical profession, health insurers, the National Cancer Institute and other NIH institutes be created to "change the dynamic surrounding initial conditions associated with the emergence of a new medical procedure".

This proposal seems strangely naïve given the preceding chapters which emphasized the pervasive and destructive influence of the both the media and the legal profession in this saga. It seems unlikely that those influences could be tamed by such a collaboration.

There is a NZ connection for readers from this country. One of the authors is Cindy Farquhar from Auckland who researched the (over) utilisation of HDC/ABMT for breast cancer in the USA during her time as a Harkness Fellow based at the Agency for Healthcare Research and Quality in USA. Whilst the NZ health system is often lambasted in our media for being behind the times and "third world" this case study is a sober reminder that being close to the cutting edge of medicine is not always the most desirable place to be. To my knowledge no NZ woman had a HDC/ABMT procedure for breast cancer except as part of the Anglo-Celtic randomised controlled trial. In this case our relative "isolation" minimised any harm to NZ breast cancer patients.

I recommend this book to all Oncology health professionals and anyone with an interest in health technology assessment of new medical procedures. It is a surprisingly "good read".

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Oncology Service, Christchurch Hospital
Christchurch

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Differential diagnosis of internal medicine

Walter Siegenthaler. Published by Thieme, 2007. ISBN 9781588905512. Contains 1104 pages. Price NZ$229.34

This weighty tome (1104 pages) originates in Switzerland. Apparently previous editions have been in German and this edition is the first English translation. It is intended for medical students, physicians, and all other people interested in the diagnosis of medical problems.

I decided to sample four different medical problems. The first of these was transient ischaemic attacks (TIA). This topic was dealt with in less than one page and I found it to be totally inadequate. For example, I found no suggestion that risk factors for stroke should be looked at, nor was there any suggestion that brain imaging was required. And of course there were no suggestions about how the patient should be managed.

I next looked at rheumatoid arthritis and found that this subject had one page devoted to it. I was not impressed with this. I was surprised that the American College of Rheumatology (ACR) criteria of diagnosis were not mentioned, or for that matter, any similar criteria.

The topic of myeloma warranted three pages, and this was extremely poor. The translation from German to English was not very successful and many unfamiliar and confusing expressions were noted.

The subject of the acute coronary syndrome was dealt with in seven pages, and this was reasonably satisfactory. Unusual terminology was also noted and I felt that there was too much detailed ECG discussion.

Based on my sampling of this book I could not recommend it. In addition to the points that have already been mentioned, I find it very strange that a text book of this size should specifically go out of its way to exclude management of the problems under discussion. Most seekers of the truth would appreciate the principles of management to be included with the description of the disease in question.

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White Gujaratis: Bramwell and Dorothy Cook

Published by H. Bramwell Cook, 2007. ISBN 9780473121129. Contains 238 pages. Price $45. Available from The Salvation Army Bookshop, 202 Cuba Street (PO Box 6015), Wellington

The White Gujaratis, Bramwell and Dorothy Cook, were Salvation Army medical missionaries who devoted most of their professional lives to working in the town of Anand in the Indian state of Gujarat.

Their story, and that of their extended family, is told by their son, Dr H Bramwell Cook, a well known and respected Christchurch gastroenterologist. In his words "This then is the story of Mansukh (Bramwell), the happy heart, and Yuddha Bai (Dorothy), the warrior. A story of love and courage and of lives richly blessed through the years".

Bramwell Cook has written a meticulously researched and detailed account with extensive quotations from family letters and oral recording. The large number of interesting photographs and the excellent indexing are notable features. The result is a fascinating family saga and a fitting dedication to two remarkable New Zealanders. Although the prime purpose of the book is to record the personal history of the Cook family and their work as members of the Salvation Army, there is much to interest the medical reader. The descriptions of Dr A Bramwell Cook's early schooling at Waitaki Boys High School in the years after World War I and life as a medical student in Dunedin capture well the educational and social mores of the time.

The description of Dr Cook passing the FRCS(Edin), MRCP(London) and Diploma of Tropical Health and Hygiene examinations, all within 15 months and without a hospital appointment or regular income, is a testament to his commitment to his calling.

The detailed descriptions of the medical work and social changes in India over 30 years from the early 1930s are fascinating to anybody with a love of India or an interest in tropical and nutritional diseases. The workload for a doctor in the India of the 1930s would make most modern doctors quail. Dorothy Cook wrote to her mother in 1936 "Yesterday – a record – saw 250 outpatients and about 12 operations, besides the 130-140 inpatients."

Bramwell senior studied the diarrhoeal illness then called sprue and described a variant which he called idiopathic steatorrhoea but which is now known as coeliac disease. Interestingly, Bramwell the author has a special interest and expertise in the management of this condition in New Zealand patients.

Those with an interest in India, medical history or the Salvation Army's contribution to social welfare will enjoy this book.

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