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This Issue in the Journal

Behaviours and beliefs about pain and treatment among Chinese immigrants and New Zealand Europeans

Ping (Carolyn) Ho, Malcolm H Johnson

This study investigates cultural differences on pain experiences, coping strategies and beliefs among general public between Chinese immigrants and New Zealand Europeans. We found the Chinese population reported more persistent pain than New Zealand Europeans and the management of their pain was different between the two groups. However, both cultures seem to share very similar attitudes and beliefs towards pain. Levels of acculturation were looked into and yet did not find an impact on the Chinese population. Interestingly, some participants in both groups actually behaved in a way that is different from their beliefs and attitudes.

Prevalence of diagnosed and undiagnosed diabetes and prediabetes in New Zealand: findings from the 2008/09 Adult Nutrition Survey

Kirsten J Coppell, Jim I Mann, Sheila M Williams, Emmanuel Jo, Paul L Drury, Jody C Miller, Winsome R Parnell

Diabetes is a common chronic disease with significant morbidity, mortality and cost, and the prevalence continues to increase worldwide. This study updates diabetes prevalence data for New Zealand. Data from the 2008/09 Adult Nutrition Survey provided an opportunity to estimate national prevalence of both diabetes and prediabetes in New Zealand for the first time. The prevalence of diabetes was 7.0% and the prevalence of prediabetes was 18.6%. Diabetes was more frequent in men (8.3%) than in women (5.8%). The prevalence of diabetes was higher among the obese group (14.2%) compared with the normal weight group (2.4%), and one-quarter of those who were obese had prediabetes. The prevalence of diabetes differed markedly among the three ethnic groups - Pacific (15.4%), Māori (9.8%) and NZ European and Other (6.1%). The high frequency of prediabetes suggests diabetes is likely to become more common, particularly in high risk groups. Implementation of effective evidence based diabetes prevention strategies is required to reduce the increasing costs of the diabetes epidemic.

The effectiveness of the Complete Health Improvement Program (CHIP) in Australasia for reducing selected chronic disease risk factors: a feasibility study

Darren P Morton, Paul Rankin, Peter Morey, Lillian Kent, Trevor Hurlow, Esther Chang, Hans Diehl

The Complete Health Improvement Project (CHIP) is a group-based lifestyle modification program that occurs in a community setting. A unique element of CHIP is that non-health trained volunteers can administer the program. CHIP shows promise for the management of several chronic diseases including heart disease and type 2 diabetes. Significant overall reductions in participant's body mass, blood pressure, total cholesterol and fasting plasma glucose levels were recorded in this study.

Binge drinking among Māori secondary school students in New Zealand: associations with source, exposure and perceptions of alcohol use

Terryann C Clark, Elizabeth Robinson, Sue Crengle, Janie Sheridan, Nicki Jackson, Shanthi Ameratunga

A nationally representative health and wellbeing study of secondary school students through New Zealand, found that among Māori students who currently drink alcohol, 31.5% reported binge drinking (5–9 drinks) and 30.4% reported heavy binge drinking (≥ 10 drinks) in a 4-hour session within the previous four weeks. Compared with non-binge drinkers, binge drinkers more frequently reported 'drinking alcohol was okay for people their age', had friends that drank alcohol, had sourced alcohol from friends or from 'other adults' and buy their own alcohol. Binge drinking was associated with poorer school performance, unsafe sex, unwanted sex, an injury, injuring someone else, motor vehicle crashes and 'doing things that could cause trouble'. Binge and heavy binge drinkers reported greater difficulty accessing drug and alcohol services.

In summary, binge drinking is associated with a range of poor health and social outcomes for Māori youth. The ease of access to alcohol for secondary school students to alcohol and associated poorer access to drug and alcohol services contributes toward poorer health and social outcomes for Māori youth.

A new podiatry service for patients with arthritis

Keith Rome, Kathryn Erikson, Anthony Ng, Peter J Gow, Hazra Sahid, Anita E Williams

The article shows that specialist in foot-care (podiatrist) can reduce pain and disability related to patients with rheumatic disease. Treatments such as footwear advice, removal of hard skin and shoe inserts have been shown to improve patient's foot problems. Patients found the service very helpful for their foot problems.

Accuracy of visual acuity testing in New Zealand primary health care

Nishanthan Ramachandran, Gordon Sanderson, Tui H Bevin, Giles Wynn-Williams

Visual acuity measurement is a general screening tool used by doctors and allied health professionals, with obvious medico-legal implication. We compared visual acuity scores of 15 to 26 “eyes” at 17 visual acuity testing centres, located in 9 general practices and Emergency Department of Dunedin Hospital in Dunedin, New Zealand, with their scores obtained in the Eye Department of Dunedin Hospital. We measured variables of testing centres, such as lighting and distance. There were inconsistencies in visual acuity measurements, which may be partly explained by the overall poor compliance with lighting and distance standards by primary health care providers. These factors are potentially easily modifiable and their change should lead to improvements in visual acuity testing and potentially more appropriate referrals to optometrists and ophthalmologists.

The pain experience and sociocultural factors

Edward A Shipton

Pain is a complex sensation that involves sensory, motivational, and cognitive components.¹ The experience of pain is characterised by immense inter-individual and group variability.^{2,3} The pain response is not restricted to a physiological reaction to noxious stimuli or tissue injury, but encompasses emotional and behavioural responses as well.

These responses have as their foundation variations in cultural perceptions, expectations, and past experiences that are known to differ among race/ethnic groups.^{4,5}

Substantial literature suggests that diverse biological, psychological, and sociocultural mechanisms account for differences by race and ethnicity in the experience, epidemiology, and in the management of pain.⁶

Race/ethnicity seems to have a larger impact on later stages of pain processing, including emotional and behavioural responses associated with chronic pain.⁷

Culture is defined as "the customary beliefs, social norms, and material traits of a racial, religious, or social group."^{5,8} Culturally-specific attitudes and beliefs about the origin, role, and meaning of pain not only influence the manner in which individuals view and respond to their own pain, but can affect how they perceive and respond to the pain of others.⁵

Cultural factors related to the pain experience include pain expression, pain language, lay remedies for pain, social roles, and expectations and perceptions of the medical care system."^{5,9} The extent to which culture can influence pain perception and response depends in part on the degree to which individuals identify with their ethnic or cultural group.^{5,9} Culture can in turn influence the request for medications or treatments to assist in ameliorating the pain.¹⁰

The perception and experience of physical pain and the meaning pain has to one's existence will vary by culture.¹¹ In the Chinese culture, pain has been understood as a result of blocked *Qi* (life energy or force).¹² To resolve the pain, the blockage must be removed and the patient must return to a state of harmony with the universe.¹²

Research has been performed on the relationship between expectations and pain experience.¹ Expectations about treatments and about painful stimuli have been shown to profoundly influence brain and behavioural markers of pain perception.¹

Another psychosocial factor that may influence differences in pain sensitivity response is the gender role.¹³ Individuals who considered themselves more masculine and less sensitive to pain have been shown to have higher pain thresholds and tolerances.¹³

The pain experience is therefore shaped by a dynamic interplay between physiological, psychological, and sociocultural factors.¹⁴ For example, heightened

pain reactions have been found among individuals of Asian ethnicity relative to those of European ethnicity, both in North America and Europe.¹⁵

Experimental pain measures may facilitate identification of biological, psychological, and sociocultural contributions to ethnic differences in pain processing. A recent systematic review analysed the use of experimental pain stimuli in assessing pain sensitivity across multiple ethnic groups.¹⁶ It found that race/ethnicity contributed significantly to variability in pain responses across most pain stimulus modalities.

In this issue of the *New Zealand Medical Journal*,¹⁷ Ho and Johnson importantly investigate how pain is construed and managed cross-culturally. Two groups, New Zealand Europeans and Chinese people, were defined for cultural comparison. The study anonymously recruited 165 participants from the general public (57.0% Chinese, and 43.0% New Zealand Europeans); the participants completed a questionnaire that measured the following characteristics: demographics, experiences of persistent pain, use of pain management and alternative treatment, as well as pain attitudes and beliefs.

Cultural differences did not appear significant for pain experiences, but influenced perceptions about pain as well as how people managed their pain. The study identified numerous cultural differences among New Zealand Europeans and Chinese immigrants in terms of beliefs about persistent pain and its treatment.

Acculturation, which entails adaptation to a new set of cultural norms, beliefs, and values,¹⁸ is inherently stressful, especially for first-generation immigrants.¹⁹ Stress of acculturation, in turn, may influence pain sensitivity.¹⁵

Two recent studies have demonstrated this.¹⁵ In one study, first- and second-generation Asian Americans and European Americans took part in a cold pressor task. Evidence of heightened pain responses was found only among first-generation Asian Americans.¹⁵ The second study was further controlled for ethnicity. It replicated this pattern in finding heightened pain reactions among mainland Chinese students in Hong Kong relative to Hong Kong Chinese students.¹⁵

These findings suggested a role for acculturation in accounting for ethnic differences in physical pain sensitivity.¹⁵ However, in the study by Ho and Johnson, acculturation levels did not reveal any substantial impact on the pain frequencies.

As stated by Ho and Johnson, “culture plays an important role in determining various aspects of pain experience and response.” A growing multicultural society presents healthcare providers with a difficult task of providing appropriate care for individuals who have different life experiences, beliefs, value systems, religions, languages, and notions of healthcare.¹⁰ Cultural practices and spiritual beliefs form the foundations on which many lives are based.

As the patient population in the New Zealand becomes increasingly multicultural, cross-cultural training, the use of cross-cultural principles, and the appreciation of the needs of immigrant patients and families becomes increasingly important.¹⁰

The challenge to healthcare professionals is to strive to become more culturally sensitive and culturally competent. As proposed by Ho and Johnson, further cross-cultural investigations using randomised samples instead of self-selected survey populations are awaited.

Competing interests: Nil.

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Behaviours and beliefs about pain and treatment among Chinese immigrants and New Zealand Europeans

Ping (Carolyn) Ho, Malcolm H Johnson

Abstract

Aims To investigate how pain is construed and managed across Western and Chinese cultures.

Methods Adults from the general public completed an anonymous survey developed for this study. Participants responded to recruitment posters and handouts that were distributed to Auckland community centres, libraries and relevant social organisations.

Results 165 participants were recruited with slightly more Chinese respondents (57.0%). 128 participants (77.5%) reported having experienced persistent pain which did not recover within expected periods in the last 5 years, and occurred more among Chinese (60.2%) than New Zealand Europeans (39.8%). Pain behaviours and coping strategies were found to be significantly different between Europeans and Chinese. However, differences in perceptions regarding pain and treatment were not substantial. Interestingly, for both cultures some participants reported behaving differently than expected according to their perceptions. Acculturation levels, however, did not show any great impact on Chinese immigrants. The high incidence of persistent pain reported in the study compared to random population surveys suggest individuals who had pain experiences were more likely to respond to the study.

Conclusions It is evident that culture plays an important role in determining various aspects of pain experience and response, although further investigation using randomised samples instead of self-selected survey populations is required to clarify the picture. The effect of acculturation levels particularly should be further investigated.

It is now well accepted that the unpleasant physical experience we call pain incorporates psychological, social and cultural influences.¹⁻³ It is also likely to be the case that these influences, unless understood and incorporated into assessment and treatment of painful conditions, will perturb patient management.

Literature shows that different cultural backgrounds influence attitudes towards pain medication and patients' beliefs and expectations of other pain treatments.^{4,5} This in turn may affect pain managing behaviours, and at times cause barriers to effective pain management if medical professionals do not incorporate awareness of cultural values and beliefs in relation to their patients' pain experiences.

Thus, understanding the beliefs about pain and illness that are embedded in different cultures may be vital to provide effective intervention.

Culture is defined as beliefs, values, practices and social behaviours shared by members of a certain group.⁶ It helps people to construct basic assumptions about reality that closely interrelate to identity, social life, as well as health care.⁷ The literature consistently indicates that the perception and management of illnesses are different across cultures, and that good health is constructed via diverse assumptions from different ethnicities.^{8,9}

The Western culture

The Biomedical model is the predominant medical model used in the Western world.¹⁰ In this model symptoms, in the absence of identifiable pathophysiology, are often attributed to psychological dysfunction.⁸

Western medicine has been largely concerned with anatomical and biological constructs with human bodies viewed as comparable to working machines. Pain is therefore conceived as indicating defective body parts, and medical practitioners are expected to relieve pain by mending or replacing the broken 'machine'.

Physicians practicing from the biomechanical perspective often consider mentioning psychological or emotional wellbeing inappropriate,¹¹ a standpoint also held by much of the general public. People visit their primary care physicians when they believe they require medical consultation for their symptoms, and expect a diagnosis and remedies after physical examination.¹²

Patients are often unsatisfied when medical practitioners cannot find pathological reasons for ill health and become particularly upset if physicians suggest their illnesses are psychologically based.¹³

The Chinese culture

Traditional Chinese Medicine (TCM) on the other hand perceives that an illness is a common outcome of both pathogenetic factors and internal maladjustments within the body. The Chinese viewpoint of health is based upon harmony within the macrocosm—which implies human beings living between heaven and earth—as well as balance inside the microcosm, a miniature universe within an individual.¹⁴ These different philosophies of aetiology and pathology from Western medicine provide different approaches to pathology.

Health is achieved by maintaining the harmony of external and internal values such as psychosocial and ecological factors.¹⁴ The overall functional wellbeing of a person is especially focused on the bodily response to pathogenetic factors, rather than diseases characterised by pathological changes and mechanisms.¹⁵

The concept of 'yin' and 'yang' is one of the primary health paradigms in the Chinese culture. It describes the mutual correlation between contrasting phenomena in which a healthy individual will have a balanced yin-yang maintained by an energy flow within their own small universe.¹⁴ An imbalance occurs when one phenomenon is deficient and triggers the surplus of the other.

Traditionally, this is how Chinese people categorise illnesses and malaise. An invasion of excess yin energy such as cold and dampness will obstruct the yang energy causing aches and movement difficulties. This is known as the Painful Obstruction Syndrome.¹⁶

It is believed such force disrupts the harmony of the microcosm and causes energy blockage at certain parts of the body, which therefore triggers physical suffering. As a result, the correction of internal maladjustments and the restoring of self-regulatory capacity of the body are major principles for TCM.¹⁵

Immigrants and acculturation

The phenomenon of change in the cultural behaviour and thinking of a person or a population through contact with another culture is known as 'acculturation'; a term that was introduced by anthropologists to refer to the occurrence of intercultural contact.¹⁷ Acculturation might occur through gradual acceptance of host-society norms, or it could be the frequent exposure to shared factors in the physical environment or both.

Acculturation, as reflected in awareness of different lifestyles, food options and medical resources after settlement by immigrants is also influential on health maintenance.¹⁸ The possibility that pain perception and expectations about pain management are influenced by acculturation has also been suggested,¹⁹ yet no clear conclusion has been drawn.

Bates, Edwards and Anderson¹ consider ethnocultural effects might influence pain responses through social learning and social comparison. These mechanisms are similar to processes of acculturation where behaviours and emotional expressions are learned through observing and through social interaction with others.

Study rationale and aims

Western views of the aetiology of pain are more biomechanical and tend to have specific physiological diagnoses as well as considering the body and mind as separate entities. In contrast, Chinese people tend to have a more holistic perception towards health. Separate treatments and coping strategies have been developed according to these perceptions and beliefs.

This study aims to investigate cross-cultural aspects of pain, specifically how pain is construed and managed across Western and Chinese cultures by individuals that have experienced a pain condition that persists longer than expected to recover which is not easy to explain (persistent pain). Since the New Zealand resident Chinese sample in the study will be variously acculturated, it is also important that the impact of acculturation on pain beliefs and behaviours is investigated.

Method

Adults from the general public within the Auckland region participated in the current study by responding to recruitment posters and handouts that were distributed to Auckland community centres, libraries and relevant social organisations. Two groups were defined for cultural comparison: New Zealand Europeans and Chinese people. There were 7 surveys completed and returned by New Zealand born Chinese; as the number was too low to create an additional group, these 7 participants were included in the Chinese group rather than excluded. 179 of the 310 sent surveys were returned, giving a

response rate of 57.74%. However, 14 surveys were excluded due to insufficient demographic data and late arrivals.

The study materials consisted of a questionnaire developed for this study measuring the following characteristics: demographics, experiences of persistent pain, use of pain management and alternative treatment, as well as pain attitudes and beliefs.

There were two parallel versions of the questionnaires, one for New Zealanders and one for Chinese. For the Chinese questionnaires, an additional 12 items regarding levels of acculturation were included. The development of these items were informed by an acculturation study reported by Matsudaira.²⁰

The items are on a Likert scale from 1 to 10, where 10 indicates strongly agree. Items included Chinese participants' identification with New Zealand culture and their own culture, the adoption of New Zealand culture and lifestyles, the use of language and status of current social life and overall satisfaction level living in New Zealand. The minimum and maximum score the participants can obtain is 12 and 120 respectively.

Data were first screened to explore the normal distributions of the scores. All variables, except for the items examining levels of acculturation, were not normally distributed since there was evidence of skewing and kurtosis. Data transformation failed to resolve these problems. Non-parametric tests were therefore applied for most analyses with parametric tests applied when possible. An alpha level of 0.05 was used for all statistical analyses.

Demographic characteristics were analysed by one-sample Chi-square tests. A two-way contingency table analysis was conducted on pain behaviours. Mann-Whitney tests were conducted to investigate other pain experiences—pain intensity, disability and impact—as well as pain perceptions and beliefs between cultures.

A Spearman's correlation was performed to explore the association between levels of acculturation and pain perceptions. A series of independent samples t-tests were conducted to assess the relationship between levels of acculturation and choices of pain management.

Results

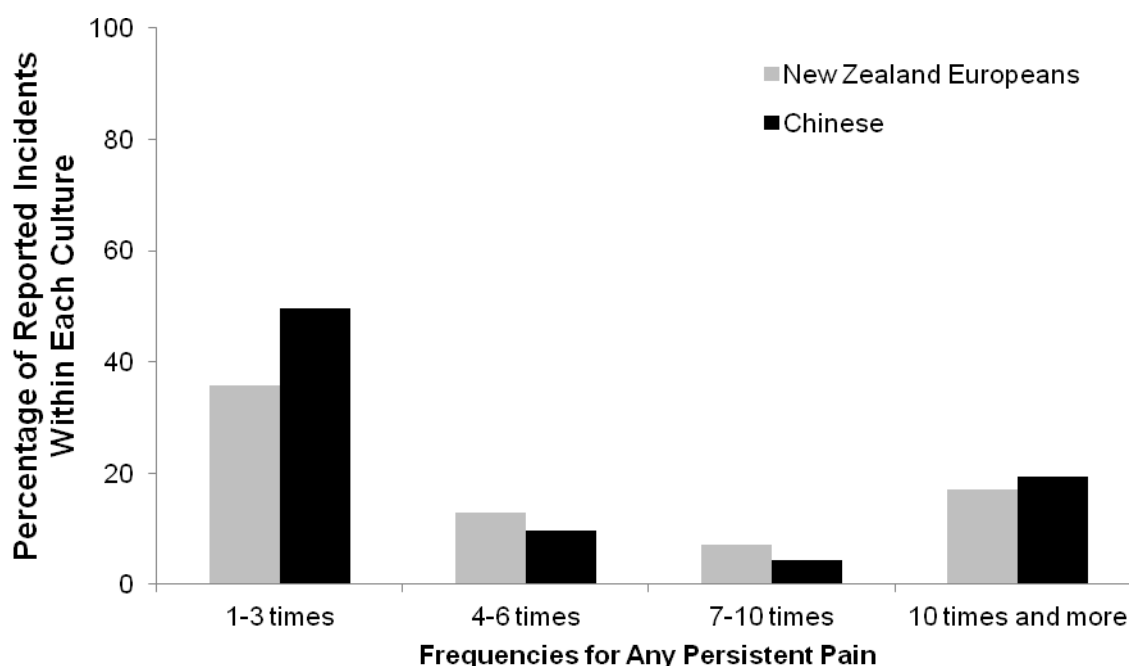
There were more Chinese respondents ($n=94$, 57.0%) than New Zealand Europeans ($n=71$, 43.0%). The mean age for the study sample was 35.5. The youngest participant was 18, and the oldest 66.

128 participants (77.5%) had experienced any form of persistent pain which did not recover within expected periods in the past 5 years. Of these, 51 (39.8%) were New Zealand Europeans and 77 (60.2%) were Chinese although pain experience was not different between the groups, $\chi^2(1, N=128)=.126$, $p>0.05$.

The mean age for these 128 participants was 36.19; with the New Zealand Europeans (39.73) being slightly older than the Chinese (33.91). There were more female participants than males for both cultural groups; 18 males and 33 females for New Zealand Europeans and 30 males and 47 females for Chinese.

Figure 1 shows there was no significant difference between cultures in reported frequencies of persistent pain either. Acculturation levels did not reveal any substantial impact on the pain frequencies when further analyses were performed, $F(3,71)=0.24$, $p=0.995$. The main results of the study will be based on participants who have reported persistent pain.

Figure 1. The frequencies of any persistent pain experience between New Zealand Europeans and Chinese in the past 5 years



Of the 128 participants reporting persistent pain, 47.7% (n=61) reported having disability associated with their pain, where the pain experience disabled, stopped or prevented them from doing other things; but again no significant differences were found between the two cultures, $z=1.691$, $p=0.091$ (Table 1).

On the 10-point Likert scale, the mean reported pain intensity of the most severe persistent pain condition for sufferers was 4.86, indicating moderate pain experience. A mean of 4.05 was reported for the impact of pain on their lives, where the survey asked how significantly their pain condition has impacted their lives, suggesting a moderate influence. There were no differences between the two ethnicities for either pain intensity, $z=-0.258$, $p=0.796$ or the impact of pain on life, $z=-1.159$, $p=0.246$.

Table1. Comparison of impacts of pain between New Zealand Europeans and Chinese who reported persistent pain in the past 5 years.

Variables	New Zealand Europeans (n=51) Mean Rank	Chinese People (n=77) Mean Rank	z	p value
Pain disability	70.39	60.60	-1.691	0.091
Pain intensity	65.53	63.82	-0.258	0.796
Impact in Life	69.11	61.45	-1.159	0.246

Table 2 indicates the most frequent pain management and behaviours were obtaining a diagnosis for their pain (58.6%), receiving alternative treatments (57.0%), seeing their general practitioners (GPs: 46.9%), and taking medication (46.9%).

The results indicated New Zealand Europeans acted to manage their pain more than the Chinese in various ways: they were more likely to see health professionals, ask family and friends for help, take medication, obtain a prescription for medication, seek physical therapy, seek other alternatives, and obtain diagnoses for the pain conditions. Interestingly, acupuncture as a treatment option was not significantly different between the two groups although acupuncture is a Chinese traditional treatment.

Table 2. The numbers and percentages of participants who reported engaging in pain management and behaviours for their persistent pain condition

Variables	New Zealand Europeans (n=51)		Chinese People (n=77)		Total		χ^2	p value
	n	(%)	n	(%)	n	(%)		
I ignored it	17	33.3	36	46.8	53	41.4	0.133	0.131
I saw my GP	32	62.7	28	36.4	60	46.9	-0.259	0.003
I saw a specialist	22	43.1	11	14.3	33	25.8	-0.323	0.000
I asked family and friends for help	10	19.6	5	6.5	15	11.7	-0.200	0.024
I took medication to relieve pain	28	54.9	27	35.1	55	43.0	-0.196	0.026
– Prescribed medication	17	33.3	12	15.6	29	22.7	-0.208	0.019
I prayed	2	3.9	9	11.7	11	8.6	0.136	0.125
I sought alternative treatment	29	56.9	44	57.1	73	57.0	0.003	0.975
– Massage	8	15.7	23	29.9	31	24.2	0.162	0.067
– Acupuncture	6	11.8	18	23.4	24	18.8	0.146	0.099
– Physical therapy	14	27.5	8	10.4	22	17.2	-0.221	0.012
– Traditional healing	1	2.0	3	3.9	4	3.1	0.054	0.538
– Herbal healing	1	2.0	8	10.4	9	7.0	0.161	0.068
– Yoga	3	5.9	4	5.2	7	5.5	-0.015	0.867
Other alternatives	15	29.4	7	9.1	22	17.2	-0.264	0.003
Diagnosis for the pain condition	36	70.6	39	50.6	75	58.6	-0.198	0.025

Eleven of the 16 items assessing pain attitudes and beliefs were different between the two groups, with the Chinese sample holding stronger beliefs in each case (Table 3).

Table 3. Comparison of pain attitudes and beliefs between New Zealand Europeans and Chinese who reported persistent pain in the past 5 years

Questionnaire items and numbers		New Zealand Europeans (n=51) Mean Rank	Chinese People (n=77) Mean Rank	z	p value
C1	Pain can only be managed by medication.	54.36	71.21	-2.578	0.010
C4	If the doctors cannot tell me what is causing the pain, I believe it is punishment for some things I have done wrong.	56.01	70.12	-2.778	0.005
C5	Other than physical damage, disruption of the harmony among elements within the body can also cause pain.	56.72	67.81	-1.683	0.092
C6	I believe family disruption is the main cause of my pain.	61.50	66.49	-0.790	0.429
C7	There must be a clear diagnosis for pain.	50.69	72.64	-3.325	0.001
D1	Seeing doctors for a clear diagnosis and treatment is the best way to manage pain.	58.20	68.68	-1.592	0.111
D2	I expect that when I visit my doctor, there is hope the doctor will cure my pain condition.	59.46	67.84	-1.276	0.202
D4	I am disappointed when doctors cannot find out what is causing my pain.	53.77	70.64	-2.567	0.010
D5	I believe there is always a cure for pain.	46.64	70.73	-4.491	0.000
D6	When doctors tell me that there is nothing they can do to make the pain go away, it is because they do not try hard enough.	55.10	70.73	-2.378	0.017
D7	I believe managing pain with my family is more powerful and helpful.	46.65	76.32	-4.500	0.000
D8	I dislike how physical discomfort and pain is diagnosed and treated in New Zealand	52.87	72.20	-3.005	0.003
D10	Understanding my culture is an important part of understanding my physical illness and pain.	61.75	66.32	-0.701	0.483
E2	If Western medicine does not seem to work, I would seek out traditional medicine.	50.51	72.76	-3.389	0.001
E4	I value traditional treatment more than Western medicine	51.21	71.32	-3.096	0.002
E6	The reason I choose traditional medicine over Western medicine is because of communication and language difficulties	51.67	65.61	-2.221	0.026

Acculturation level in relation to pain management and beliefs

Out of the 77 Chinese participants who reported persistent pain in the past 5 years, 75 (97.4%) fully completed the additional acculturation items. The mean level was 64.40 with a minimum of 26 and a maximum of 94. Most participants scored between 60 and 70, indicating the study had sampled more acculturated participants. This is not surprising since, other than the 7 New Zealand born Chinese, 58.1% of the Chinese

participants reported they have lived in New Zealand for over 10 years; 30.2% lived in between 4 to 6 years and 11.6% just under three years.

A series of Independent Sample T-Tests were conducted to compare Chinese participants in relation to pain management and behaviours at different acculturation levels. Only one item revealed significant difference where more acculturated participants were less likely to take the traditional healing approach, $t(73)=-2.929$, $p<0.05$.

Spearman's correlations were conducted to investigate correlations between acculturation levels and pain attitudes. Table 4 shows the 4 (of 23) items that were significantly correlated with acculturation levels. More acculturated participants believed non-physical factors influenced pain while less acculturated participants preferred to see a culturally similar doctor.

Table 4. Spearman's rho correlations for pain beliefs significantly correlated with acculturation levels

Questionnaire items and numbers		Acculturation Level	P value
C3	I believe there is a psychological factor leading to may pain, such as tension and stress.	0.360	0.002
C5	Other than physical damage, disruption of the harmony among elements within the body can also cause pain.	0.241	0.038
D10	Understanding my culture is an important part of understanding my physical illness and pain.	0.350	0.002
E3	Regardless of traditional or Western medicine, I prefer to have a doctor who is of the same culture and speaks the same language as I do.	-0.286	0.013

Discussion

The present study found 77.5% of the participants had experienced persistent pain in the past five years. The percentage reported was much higher than the findings of Frohlich, Jacobi, and Wittchen, 8.1%;²¹ or Gureje, Von Korff, Simon, and Gater, 22%.²² Additionally, the 2006/7 New Zealand Health Survey, a nationally representative cross-sectional survey, reported that 16.9% experienced chronic pain.²³

The dramatic differences in the percentages of persistent pain reported in these studies probably means either the various research populations are not comparable or the definitions of persistent pain are different, or both. Our study defined persistent pain as “a pain condition in the past 5 years that has persisted when you expected it to recover” while the definition used for the NZ Health Survey was “pain that is present almost every day ...that has lasted or is expected to last 6 months or more.”

Frohlich et al applied the even more stringent DSM-IV diagnostic criteria.²¹ Also, it seems likely that because it was about pain, our study attracted more people who have persistent pain in contrast to Gureje et al who recruited consecutive patients from health centres.²² It was difficult to compare the rate with other studies in China since they tend to look at specific pain conditions and hardly ever conduct nationwide

prevalence research. The most recent study conducted on the prevalence of chronic pain was amongst Hong Kong adults where 10.8% of the sample reported pain.²⁴

Overall, it was found that cultural differences were not evident regarding pain experiences but dissimilarities were found in responses to pain, such as more New Zealand Europeans would seek diagnoses than Chinese people. Rhodes et al., report that pain patients often expect a pain examination in order to discover evidence of biogenic cause, localisation and possible medical remedies.²⁵

The finding in the current study suggests that this is the case for New Zealand Europeans who sought diagnoses more and also had more visits to health professionals for pain. This is in accord with the literature that indicates that Westerners have more physically-oriented perceptions of health and tend to attribute their sickness to biological factors.^{11,12}

Taking medication is perceived as the most common and easiest way to manage pain in Western societies.^{26,27} The study participants reported moderate medication use with more New Zealand Europeans reporting taking medication—both prescription and other medications, confirming earlier research that suggests that Western patients frequently obtain prescriptions and use various analgesics for pain.^{28,29}

Although there were no cultural differences between groups in seeking alternative treatments, the rate of this pain behaviour nevertheless supported previous findings in New Zealand studies as over half of the study population reported seeking alternative treatment. For example, GPs' referral rate to alternative therapies in Auckland 20 years ago was 68.7%, where chronic pain syndromes and musculoskeletal disorders were most frequently treated.³⁰ Two years later, a study reported 52.4% of chronic pain patients consulted complementary therapists in the same region.³¹

More recently, it was found 95% of GPs had referred their patients to one or more alternative medicine sources in a nationwide cross-sectional study.³² These findings reveal increasing recognition and acceptance of CAM in New Zealand society, and the high overall rate may also explain why there were no cultural variances in seeking alternative treatments between the two cultures.

Because acupuncture is a traditional Chinese treatment it was surprising that there was no significant difference between cultures in the endorsement of this as a treatment. Again this probably reflects the general acceptance of CAM in New Zealand society and possibly also the availability of funding for acupuncture through the Accident Compensation Corporation (ACC). In contrast, physical therapy, also funded through ACC, was more endorsed by the European sample, perhaps reflecting a more active approach to managing pain.

Although the Chinese sample seemed less challenged by the impact of pain in their lives, they reported higher means for all perception items. In this context it was unexpected to find items suggesting family disruption may be a trigger for pain, or disharmony of bodily elements might cause pain, or the understanding of one's culture is important to understand pain, were not more highly endorsed by the Chinese participants as these appear to be in accord with traditional beliefs.

The findings indicated that New Zealand Europeans hold some pain perceptions that are different from their actual pain behaviours. While they were more likely to take

medication to control pain and to request diagnoses, this group actually agreed less with the perception items stating pain can be managed by medication and that there must be diagnoses for pain.

These findings contradict reports from other studies that indicated Westerners tended to believe analgesics were an effective way to relieve pain but agree with evidence they obtain more prescriptions and have higher medication usage.^{11,25,28,29} This is perhaps another example where people's behaviours are not in accord with their beliefs. At times people behave in a way that would be less beneficial to themselves such as not doing enough exercises or eating properly despite having the knowledge of how to live a healthy life.

The cost of seeking medical care as well as its accessibility may also play a role in how people manage their pain. In addition, it is possible that people are genuinely ready to seek medical assistance in spite of lacking knowledge and confirmation of how effective the medications are. More investigation is required to further explore and determine the underlying mechanisms that lead to such conflicting outcomes.

Conversely, results found Chinese participants strongly believed that there is always a cure for pain, when it is suggested only Westerners would hold such strong attitudes.^{11,13} These findings do not indicate that the studied Chinese participants are less holistic according to the Chinese health model, rather, degrees of holistic health perceptions vary across different Chinese people and in this study were not substantially affected by acculturation, perhaps because the sample were relatively acculturated overall. Future studies will be required to identify actual factors.

It is possible for less acculturated Chinese to hold extensive knowledge and attitudes which lead to responses similar to those who are highly acculturated. It is found in China, medical students during their clinical years tended to show negative attitudes toward TCM and more faith in Western medicine than the pre-clinical students.³³ This somewhat contradicts previous assumptions suggesting immigrants need to become habituated to the society and lifestyles of their host country in order to be acculturated and leaves open the possibility that immigrants are 'preacculturated' to some extent.

Yijala and Jasinskaja-Lahti proposed acculturation to a new culture can occur prior to migration, based on the potential migrants' perceptions of the preferences of the future hosts and the level of contacts with the hosts.³⁴ If this is the case the lack of substantial differences between the two groups found in our study might be expected.

Conclusion

Cultural differences did not appear significant for pain experiences, but did influence perceptions about pain as well as how people manage their pain. While acculturation levels were explored in relation to pain behaviours and attitudes, no substantial impact was reported.

An unanticipated finding was revealed when people reported managing and responding to pain differently from the way their perceptions would seem to predict. Given the wealth of evidence that health behaviours are not always in accord with the health beliefs.³⁵⁻³⁷ This result, although unexpected, is not surprising; and the inconsistency may offer useful information for health professionals to better understand and help people who experience persistent pain.

Currently, the Ministry of Health in New Zealand aims to improve consideration of the social and cultural determinants in health,³⁸ in order to strengthen the cultural base of health interventions and reduce health inequalities. In addition, meeting people's needs regarding chronic conditions including chronic pain has been deemed important.³⁹

Clearly, recognising patient's "cultural cues" will ensure cultural or ethnic differences are not overlooked in considering treatment options, and will facilitate effective clinician-patient communication and optimal uptake of healthcare resources.

In conclusion, the study has identified numerous cultural differences among New Zealand Europeans and Chinese immigrants in terms beliefs about persistent pain and its treatment. As far as we are aware this is the first cross-cultural study in New Zealand regarding persistent pain, and it has indicated the importance of providing resources and support for people who experience unexplained persistent pain with an understanding of their cultural background.

Future investigations are required to further understand the unexpected findings. Additionally, seeking similar information from a random sample might indicate the extent that the present findings are unique to Chinese and European New Zealanders who have experienced persistent pain.

Competing interests: Nil.

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Prevalence of diagnosed and undiagnosed diabetes and prediabetes in New Zealand: findings from the 2008/09 Adult Nutrition Survey

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Abstract

Aim To describe the prevalence of diagnosed and undiagnosed diabetes and prediabetes for New Zealand adults.

Methods The 2008/09 New Zealand Adult Nutrition Survey was a nationally representative, cross-sectional survey of 4,721 New Zealanders aged 15 years and above. Self-reported diabetes and the 2010 American Diabetes Association cutoffs for HbA1c were used to define diagnosed diabetes, undiagnosed diabetes and prediabetes. Prevalence rates were calculated and age-specific diagnosed diabetes rates were compared with those from the Virtual Diabetes Register.

Results Overall, prevalence of diabetes was 7.0%, and prevalence of prediabetes 18.6%. Prevalence of diabetes was higher in men (8.3%, 95% CI: 6.4, 10.1) than in women (5.8%, 95% CI: 4.7, 7.0), and was higher among the obese (14.2%, 95% CI: 11.6, 16.9) compared with the normal weight group (2.4%, 95% CI: 1.4, 3.6). Prevalence of undiagnosed diabetes was highest among Pacific people (6.4%, 95% CI: 3.8, 9.1) compared with Māori (2.2%, 95% CI: 1.2, 3.1) and New Zealand European and Others (1.5%, 95% CI: 0.9, 2.1).

Conclusion The high prevalence of prediabetes indicates the prevalence of diabetes will continue to increase in New Zealand. Implementation of effective evidence-based prevention strategies is required to reduce the increasing costs of the diabetes epidemic.

Diabetes is a common chronic disease with significant morbidity, mortality and cost, and the prevalence continues to increase rapidly worldwide.^{1,2} Estimates of the prevalence of diabetes in New Zealand have limitations. The four national health surveys, undertaken in 1992/93, 1996/97, 2002/03 and 2006/07, examined self-reports of doctor-diagnosed diabetes only.³⁻⁶

New Zealand's Virtual Diabetes Registry (VDR), established by the Ministry of Health over the past 10 years, counts known diabetes cases as follows: individuals with diabetes are identified using the National Health Index from six databases with information about hospital admissions, attendance at diabetes outpatients or retinal screening, diabetes-specific medication prescriptions, laboratory HbA1c testing and mortality.⁷

As national diabetes prevalence estimates have not included undiagnosed diabetes cases, the actual burden of disease has been underestimated. A number of local or workplace-based prevalence surveys conducted since 1967 have reported both

diagnosed and undiagnosed diabetes,⁸⁻²² but the study populations were not necessarily representative of New Zealand's population.

National and international reports of diabetes prevalence in New Zealand have involved assumptions and modelling rather than direct measurements.^{23,24}

The 2008/09 New Zealand Adult Nutrition Survey (2008/09 NZANS) enquired about doctor diagnosed diabetes and a blood sample was taken for the measurement of glycated haemoglobin (HbA1c). Thus the 2008/09 NZANS has provided an opportunity to report the national prevalence of diabetes and prediabetes in adult New Zealanders using American Diabetes Association (ADA) criteria.²⁵

We also compared the prevalence of diagnosed diabetes in the 2008/09 NZANS with that obtained from New Zealand's national VDR.

Methods

The 2008/09 NZANS was a nationally representative, cross-sectional survey of 4,721 New Zealanders aged 15 years and above.²⁶ Ethical approval to undertake the survey was obtained from the New Zealand Health and Disability Multi-Region Ethics Committee (MEC/08/04/049).

The survey methods are described in detail elsewhere.²⁷ In brief, an area-based sampling frame was used based on 32,173 small geographic areas (meshblocks). 607 meshblocks were selected using probability-proportional-to-size design.

Within each selected meshblock private dwelling households in both urban and rural areas were randomly selected, then a single individual within each household was randomly selected. To ensure adequate samples for analysis, increased sampling occurred for the following groups: Māori, Pacific and the age groups, 15-18 years and 71+ years. The survey was conducted from 13 October 2008 to 4 October 2009. Informed written consent was obtained from the participant. The response rate for the survey was 61%.^{26,27}

Data—Data were obtained at participants' homes by trained interviewers using computer-assisted personal interview software. All measurements were taken by trained interviewers using calibrated instruments. Data collected included: demographics, tobacco use, alcohol consumption, medical history including a specific question about diabetes. Ethnicity was self-reported, with the option to choose up to nine different groups using the Statistics New Zealand standard ethnicity question.²⁷

Standing height was measured to the nearest 0.1 cm using a stadiometer (Seca 214) and weight was measured to the nearest 0.1 kg using electronic scales (Tanita HD-351, maximum weight 200kg). Height and weight were both measured twice, then if each duplicate measurement differed by more than 1%, a third measurement was taken. The mean of the two closest measurements was calculated and used in analyses.

A blood sample was obtained from 3,348 participants (71% of the survey respondents). Participants attended a local health clinic, where a non-fasting blood sample was collected in EDTA-treated vacutainers. These were kept at 4°C, until transported to Canterbury Health Laboratories. HbA1c was determined in whole blood using an ion-exchange high performance liquid chromatography method (Bio-rad Variant II). Samples were not collected from pregnant women, as pregnancy alters biochemical indices.

Definitions—For this study 'diagnosed diabetes' was defined as self-reported doctor-diagnosed diabetes. To allow international comparisons we used the 2010 ADA cutoffs for HbA1c to define 'undiagnosed diabetes' and 'prediabetes'.²⁵ Undiagnosed diabetes included those who had an HbA1c \geq 6.5% (48mmol/mol), but did not self-report doctor-diagnosed diabetes.

Prediabetes included those who had an HbA1c result between 5.7% (39mmol/mol) and 6.4% (46mmol/mol) inclusive, but did not self-report doctor diagnosed diabetes. The 2010 ADA criteria differ from the recently revised 2012 New Zealand criteria, which coincided with a change in HbA1c units from % to mmol/mol. The New Zealand cutoffs are - diabetes: HbA1c \geq 50mmol/mol (6.7%) and prediabetes: HbA1c 41-49 mmol/mol (5.8% - <6.7%).²⁸

Statistical analysis—Survey weights²⁷ were used in all analyses so that no group was under- or over-represented. The weights reflect the probabilities of selection of each respondent, and correct for any discrepancies between the survey sample distribution and the population with respect to age, sex and ethnicity. For this survey the estimated resident population aged 15 years and over living in private dwellings in New Zealand at 30 June 2007 was used.^{26,27}

Age-specific rates of self-reported diabetes, undiagnosed diabetes, total diabetes and prediabetes were calculated for men and women by 10 year age groups (15-24, 25-34, 35-44, 45-54, 55-64, 65-74, ≥ 75 years). Data were extracted from the VDR as at 31 December 2010. Age-specific diabetes rates were calculated for men and women by 10-year age groups as specified above. The 10-year age-specific self-reported diabetes rates using the 2008/09 NZANS data were compared with those obtained from the VDR and the 2006/07 New Zealand Health Survey.⁵

450 participants reported more than one ethnic group.^{26,27} We used prioritised ethnicity,⁴ and categorised participants into three ethnic groups: Māori, Pacific, and New Zealand European and Other (NZE), where 'Other' includes mainly Asian, Middle-Eastern, Latin-American and African ethnic groups. Because of small numbers in some age groups within each ethnic group, broader age groups were used when calculating the ethnic-specific rates (15-24, 25-44, 45-64, 65-74, ≥ 75 years).

Body mass index (BMI) was calculated as weight (kg) / [height (m)]². The World Health Organization BMI cutoff points were used to define the following categories for participants aged 19 years and over: normal weight (BMI 18.50–24.99 kg/m²), overweight (BMI 25.00–29.99 kg/m²), obese (BMI ≥ 30.00 kg/m²).²⁹ For participants aged 15–18 years, the Cole gender and age-specific BMI cutoff points were used.^{30,31} Diabetes and prediabetes rates were calculated for men and women for each of the three body weight categories.

Previous diabetes prevalence surveys—A literature search was undertaken to identify all published diabetes prevalence studies undertaken at a regional or national level in New Zealand. The methods and results for each study were summarised and tabulated.

Results

Overall the prevalence of diabetes was 7.0% (95% CI: 6.0, 8.0). Diabetes was more common among men (8.3%; 95% CI: 6.4, 10.1) compared with women (5.8%; 95% CI: 4.7, 7.0). The prevalence of diagnosed diabetes was 6.0% (95% CI: 4.5, 7.5) among men and 4.0% (95% CI: 3.1, 4.8) among women, and the prevalence of undiagnosed diabetes was 2.1% (95% CI: 1.2, 3.0) among men and 1.5% (95% CI: 1.0, 2.0) among women.

Table 1 shows the age-specific rates for diagnosed diabetes, undiagnosed diabetes, total diabetes and prediabetes for men and women aged 15 years and over. For both men and women, the prevalence of diagnosed diabetes and total diabetes increased with increasing age, notably from the 35–44 year age group, for whom the prevalence of total diabetes was 5%.

The age-specific undiagnosed diabetes rates varied among the age groups. Among men aged <45 years with diabetes, a high proportion had undiagnosed diabetes, particularly men aged 25-34 years for whom the ratio of diagnosed diabetes to undiagnosed diabetes was 1:15. Prediabetes increased with increasing age, and was higher than diabetes in all age groups.

Diabetes and prediabetes were prevalent among Māori and Pacific peoples, and particularly high among Pacific peoples (Table 2). One-third or more of Pacific people aged 45 years and over had diabetes and a further one-third had prediabetes. Age-specific rates of undiagnosed diabetes were highest among Pacific peoples, for whom the ratio of diagnosed to undiagnosed diabetes was 5:4 compared with 10:3 for Māori and 10:1 for NZEO.

Table 1. The age-specific rates for self-reported doctor diagnosed diabetes, undiagnosed diabetes, total diabetes and prediabetes by 10-year age groups for men and women aged 15 years and over

Sex	Age groups (years)	Diagnosed Diabetes [†] % (95% CI)		Undiagnosed Diabetes [‡] % (95% CI)		Total Diabetes [‡] % (95% CI)		Prediabetes [‡] % (95% CI)	
All	15–24	0.1	(0, 0.1)	0.1	(0, 0.3)	0.1	(0, 0.4)	4.5	(2.4, 6.7)
	25–34	0.8	(0, 1.6)	1.3	(0.1, 2.6)	2.4	(0.7, 4.1)	9.9	(6.2, 13.5)
	35–44	1.9	(0.8, 2.9)	2.1	(0.7, 3.5)	4.8	(2.6, 6.9)	13.1	(9.3, 16.8)
	45–54	3.9	(2.1, 5.9)	1.9	(0.4, 3.4)	6.0	(3.2, 8.7)	21.2	(15.9, 26.4)
	55–64	11.0	(7.2, 14.7)	2.9	(1.3, 4.6)	13.4	(9.3, 17.4)	31.1	(25.2, 37.0)
	65–74	12.8	(9.2, 16.4)	1.8	(0.8, 2.8)	14.7	(10.9, 18.6)	35.0	(29.7, 40.3)
	75+	15.4	(11.6, 18.2)	4.6	(2.8, 6.4)	21.3	(17.0, 25.6)	37.3	(32.9, 41.7)
	Total	4.9	(4.1, 5.7)	1.8	(1.3, 2.3)	7.0	(6.0, 8.0)	18.6	(16.8, 20.4)
Men	15–24	0.1	(0, 0.2)	0.2	(0, 0.5)	0.3	(0, 0.7)	5.6	(1.8, 9.3)
	25–34	0.1	(0, 0.3)	1.5	(0, 3.6)	1.8	(0, 3.9)	11.3	(5.3, 17.3)
	35–44	2.0	(0.4, 3.6)	2.2	(0, 4.6)	5.0	(1.4, 8.6)	16.9	(10.7, 23.1)
	45–54	4.5	(1.2, 7.9)	2.7	(0, 5.6)	7.1	(2.5, 11.8)	20.7	(12.9, 28.5)
	55–64	15.2	(8.1, 22.2)	3.5	(0.5, 6.6)	17.7	(10.2, 25.3)	27.9	(18.4, 37.3)
	65–74	16.4	(10.4, 22.3)	1.9	(0.5, 3.3)	18.7	(12.4, 25.1)	29.9	(22.3, 37.6)
	75+	19.4	(12.5, 26.3)	4.8	(2.1, 7.5)	25.4	(18.2, 32.6)	33.9	(27.5, 40.3)
	Total	6.0	(4.5, 7.5)	2.1	(1.2, 3.0)	8.3	(6.4, 10.1)	18.4	(15.7, 21.1)
Women	15–24	–*		–*		–*		3.5	(1.3, 5.7)
	25–34	1.5	(0, 3.0)	1.1	(0, 2.7)	3.0	(0.3, 5.7)	8.5	(4.1, 12.8)
	35–44	1.8	(0.5, 3.2)	2.0	(0.6, 3.4)	4.6	(2.1, 7.1)	9.7	(5.6, 13.8)
	45–54	3.4	(1.3, 5.6)	1.1	(0.1, 2.1)	4.8	(1.9, 7.7)	21.6	(14.5, 28.6)
	55–64	7.3	(3.7, 10.9)	2.4	(0.8, 4.0)	9.8	(5.5, 14.0)	33.8	(26.4, 41.2)
	65–74	9.6	(5.5, 13.6)	1.7	(0.3, 3.1)	11.1	(6.5, 15.7)	39.5	(32.0, 47.0)
	75+	12.0	(8.6, 15.5)	4.4	(1.9, 6.9)	17.6	(13.0, 22.2)	40.4	(34.3, 46.4)
	Total	4.0	(3.1, 4.8)	1.5	(1.0, 2.0)	5.8	(4.7, 7.0)	18.8	(16.4, 21.2)

† Total number completing the survey = 4721; ‡ Total number providing a sample for blood analysis = 3348; * Insufficient data to calculate rate.

Table 2. The age-specific rates for self-reported doctor diagnosed diabetes, undiagnosed diabetes and prediabetes by age group for Māori, Pacific and New Zealand European and Other (NZEО) ethnic groups

Ethnic Group	Age groups (years)	Diagnosed Diabetes[†] % (95% CI)		Undiagnosed[‡] Diabetes[‡] % (95% CI)		Total Diabetes[‡] % (95% CI)		Prediabetes[‡] % (95% CI)	
All	15-24	0.04	(0, 0.2)	0.1	(0, 0.3)	0.1	(0, 0.4)	4.5	(2.4, 6.7)
	25-44	1.4	(0.7, 2.1)	1.7	(0.8, 2.6)	3.7	(2.3, 5.0)	11.6	(8.8, 14.3)
	45-64	7.0	(5.0, 9.0)	2.3	(1.2, 3.5)	9.2	(6.8, 11.6)	25.5	(21.5, 29.4)
	65-74	12.8	(9.2, 16.4)	1.8	(0.8, 2.8)	14.7	(10.9, 18.6)	35.0	(29.7, 40.3)
	75+	15.4	(11.6, 19.2)	4.6	(2.8, 6.4)	21.3	(17.0, 26.0)	37.3	(32.9, 41.7)
	Total	4.9	(4.1, 5.9)	1.8	(1.3, 2.3)	7.0	(6.0, 8.0)	18.6	(16.8, 20.4)
Māori	15-24	-*		-*		-*		3.8	(0.4, 8.1)
	25-44	2.3	(0.8, 3.9)	2.3	(0.8, 3.8)	5.5	(2.8, 8.2)	18.8	(13.7, 23.9)
	45-64	16.4	(10.6, 22.1)	4.4	(1.6, 7.1)	20.8	(13.7, 27.9)	34.0	(25.1, 42.9)
	65-74	28.8	(15.0, 42.3)	1.1	(1.0, 3.2)	34.7	(18.3, 51.0)	45.9	(29.6, 62.1)
	75+	36.5	(15.6, 57.5)	-		40.1	(15.3, 64.9)	44.4	(16.8, 71.9)
	Total	7.0	(5.2, 8.9)	2.2	(1.2, 3.1)	9.8	(7.4, 12.2)	20.5	(16.8, 24.2)
Pacific	15-24	-*		-*		-*		10.9	(2.2, 19.5)
	25-44	4.5	(2.3, 6.7)	6.0	(1.8, 10.3)	10.7	(5.7, 15.7)	21.9	(15.5, 28.1)
	45-64	18.0	(11.9, 24.2)	12.7	(5.7, 19.7)	32.9	(21.7, 44.2)	33.3	(22.7, 43.8)
	65-74	27.6	(13.4, 41.9)	10.7	(1.0, 22.4)	34.2	(16.6, 51.8)	52.0	(32.5, 71.5)
	75+	39.0	(6.2, 71.7)	-*		55.8	(14.6, 97.0)	32.7	(5.8, 71.2)
	Total	8.1	(6.0, 10.3)	6.4	(3.8, 9.1)	15.4	(11.5, 19.4)	24.0	(19.4, 28.5)
NZEО	15-24	0.04	(0, 0.1)	0.1	(0, 0.3)	0.2	(0, 0.5)	4.1	(1.6, 6.7)
	25-44	1.0	(0.2, 1.8)	1.3	(0.2, 2.4)	2.8	(1.2, 4.4)	9.5	(6.2, 12.7)
	45-64	5.5	(3.3, 7.7)	1.7	(0.5, 2.9)	6.9	(4.3, 9.4)	24.2	(19.9, 28.5)
	65-74	11.6	(7.9, 15.3)	1.6	(0.5, 2.6)	13.2	(9.2, 17.2)	34.0	(28.3, 39.6)
	75+	14.5	(10.6, 18.3)	4.8	(2.9, 6.7)	20.3	(15.9, 24.6)	37.1	(32.6, 41.6)
	Total	4.5	(3.5, 5.4)	1.5	(0.9, 2.1)	6.1	(5.0, 7.3)	18.1	(16.0, 20.1)

† Total number completing the survey = 4721. ‡ Total number providing a sample for blood analysis = 3348; * Insufficient data to calculate the rate.

Table 3 shows prevalence rates for normal weight, overweight and obese groups. The rates of self-reported doctor diagnosed diabetes, undiagnosed diabetes and prediabetes were all higher among those categorised as obese compared with the overweight and normal weight groups.

Among the obese 14.2% (95% CI: 11.6, 16.9) had diabetes and 25.3% (95% CI: 21.4, 29.1) had prediabetes. Similar patterns were observed in both men and women.

Table 3. The age-specific rates for self-reported doctor diagnosed diabetes, undiagnosed diabetes and prediabetes by body weight category (normal weight, overweight and obesity)

Sex	Body Weight Category	Diagnosed Diabetes [†]		Undiagnosed Diabetes [‡]		Total Diabetes [‡]		Prediabetes [‡]	
		%	(95% CI)	%	(95% CI)	%	(95% CI)	%	(95% CI)
All	Normal weight	1.4	(0.6, 2.2)	0.7	(0.2, 1.3)	2.5	(1.4, 3.6)	13.9	(10.9, 16.9)
	Overweight	4.6	(3.2, 5.9)	1.3	(0.5, 2.0)	5.9	(4.3, 7.5)	18.7	(15.7, 21.7)
	Obese	9.8	(7.5, 12.0)	4.0	(2.7, 5.3)	14.2	(11.6, 16.9)	25.3	(21.4, 29.1)
Men	Normal weight	2.0	(0.3, 3.7)	1.0	(0, 2.2)	3.5	(1.1, 6.0)	12.6	(7.8, 17.3)
	Overweight	5.2	(3.2, 7.2)	1.8	(0.4, 3.2)	6.7	(4.3, 9.1)	17.8	(13.8, 21.8)
	Obese	11.4	(7.5, 15.3)	3.9	(1.8, 6.0)	15.9	(11.2, 20.5)	26.7	(20.5, 32.8)
Women	Normal weight	0.9	(0.4, 1.5)	0.5	(0.1, 1.0)	1.7	(0.8, 2.5)	14.8	(10.8, 18.7)
	Overweight	3.8	(2.1, 5.6)	0.6	(0.2, 1.1)	4.8	(2.8, 6.9)	19.7	(15.6, 23.9)
	Obese	8.2	(5.9, 10.5)	4.1	(2.4, 5.8)	12.7	(9.6, 15.7)	23.9	(19.0, 28.8)

[†] Total number completing the survey = 4721; [‡] Total number providing a sample for blood analysis = 3348.

Figures 1 and 2 compare the 10-year age-specific diagnosed diabetes rates between three sources of national diabetes prevalence data.

Overall, for men the age-specific rates are similar for most age groups. For women the age-specific diabetes rates using the VDR dataset are higher for women aged 55 years and over.

Tables 4–6 summarise and compare regional and national diabetes prevalence surveys undertaken in New Zealand since the late 1960s.

Figure 1. The 2006/07 New Zealand Health Survey, 2008/09 New Zealand Adult Nutrition Survey and the Virtual Diabetes Register age-specific diagnosed diabetes rates, by 10-year age groups for men aged 15 years and over

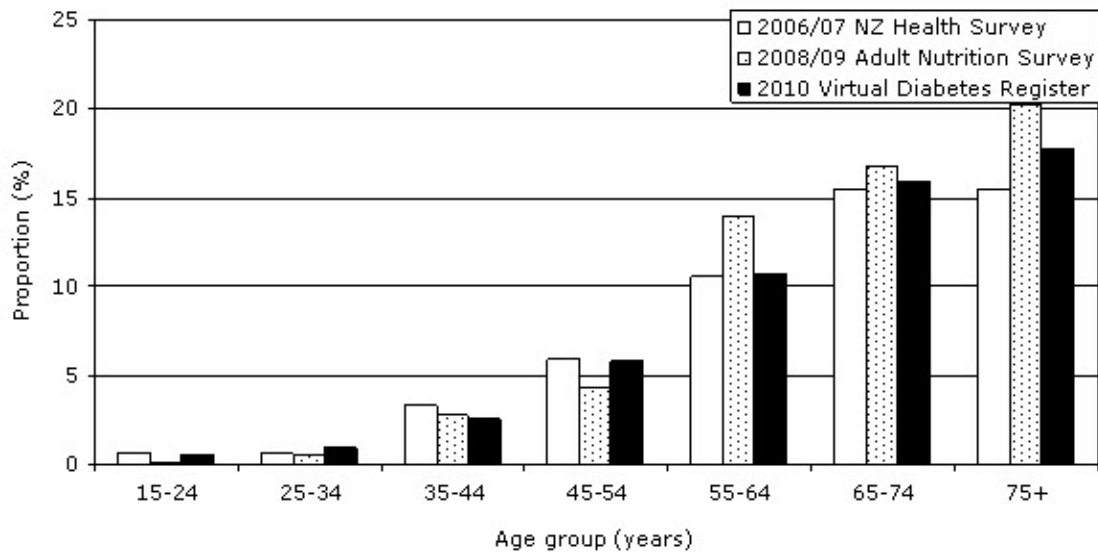


Figure 2. The 2006/07 New Zealand Health Survey, 2008/09 New Zealand Adult Nutrition Survey and the Virtual Diabetes Register age-specific diagnosed diabetes rates, by 10-year age groups for women aged 15 years and over.

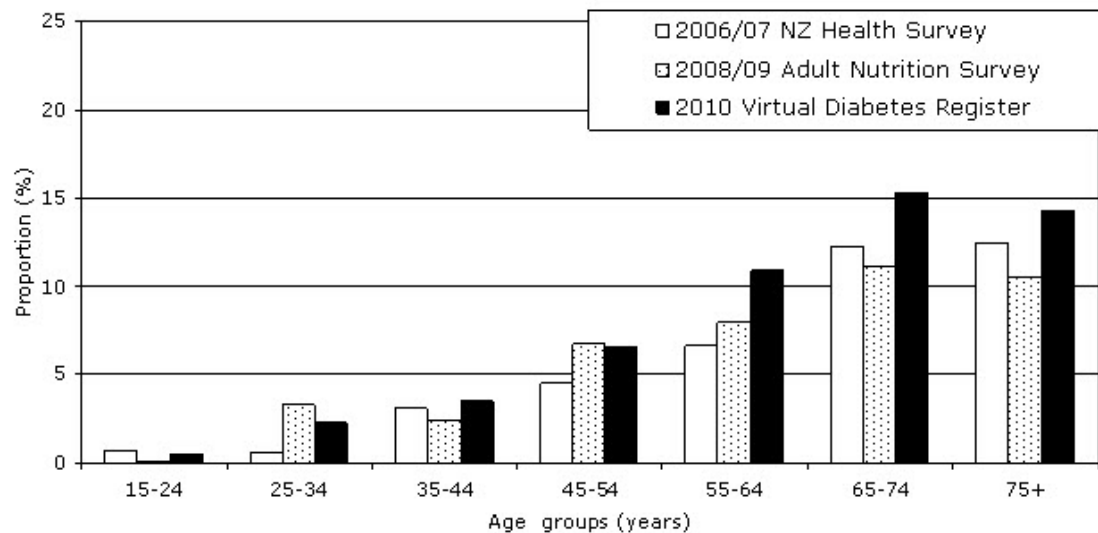


Table 4. Reported prevalence of diagnosed diabetes for different regional and national studies in New Zealand, 1967–2009

Place	Study period	Diabetes definition	Recruitment method	Number of participants	Response Rate	Age group (years)	Prevalence diagnosed diabetes (%)			
							European	Māori	Pacific	All ethnic groups
Rangiora community ¹⁴	Apr 1967	Self-report		2,670	93%	>20	-	-	-	1.7
Single large multi-departmental workplace, Christchurch ¹⁰	Dec 1982 to Feb 1983	Self-report	Workplace invitation	969	93%	≥ 15	-	-	-	1.6
41 worksites in Auckland and 5 work sites in Tokoroa ¹⁵	May 1988 to April 1990	Self-report	Workplace invitation	5,677	67%	40-44	0.4	3.6	0.8	0.8
						45-49	0.8	4.0	4.8	1.5
						50-54	1.0	4.1	11.9	2.7
						≥ 55	2.4	10.5	5.4	3.4
						40-64	1.1	5.3	5.3	1.8
Dunedin general practice ⁹	Dec 1989 to June 1990	Medical record documentation	-	-	-	Men	-	-	-	
						39-49				0.8
						50-69				7.0
						Women				
						39-49				1.9
						50-69				8.1
Large urban medical centre, Christchurch ¹³	Not stated	Medical record documentation	Random selection from practice age/sex register	595	69.4%	Men	-	-	-	
						65-69				8.0
						70-74				7.7
						75-79				3.2
						80+				4.0
						Women				
						65-69				8.5
70-74				3.3						
						75-79				2.0
						80+				0
Otaru, South Auckland ¹⁶	Apr 1992 to Oct 1992	Self-report and local GP diabetes register checks.	All households in area visited.	22,651	92.7% of 4,707 households	<20	0.4	0.1	0.1	-
						20-39	0.8	1.5	0.7	
						40-59	2.9	10.8	7.2	
						≥ 60	9.3	16.5	10.1	
						≥ 20+	3.8	5.3	3.3	
Otaru and Mangere,	Apr 1992 to	Self-report and	All households	55,518	91.2% of	20-29	0.3	0.4	0.2	-

South Auckland ¹⁸	Dec 1993	local GP diabetes register checks.	in area visited.		12,770 households	30-39 40-49 50-59 60-69 70+ ≥ 30†	0.7 2.0 4.5 7.5 8.5 4.2	2.6 7.7 14.5 16.7 11.5 7.9	1.0 5.0 12.0 13.1 10.0 5.5	
Three inner suburbs, South Auckland ¹⁹	1991 to 1994	Self-report	Random selection all households with members aged 40-79 years	1,899	69.4%	40-59 60-79	4.4 8.7	11.7 16.2	13.1 22.0	-
1996/97 NZ Health Survey ³	Oct 1996 to Oct 1997	Self-report	Stratified cluster sampling	7,862	73.8%	≥ 15*	3.1‡	8.3	8.1	3.7
2002/03 NZ Health Survey ⁴	2002 - 2003	Self-report	Stratified cluster sampling	12,929	72%	Men ≥ 15 [§] Women ≥ 15 [§]	3.4‡ 2.4‡	9.5 6.7	8.1 11.9	4.5 3.7
East Coast north of Gisborne ^{11,21}	May 2003 to Dec 2003	Self-report	Random selection local health provider age sex register	289	48.7%	25+	-	7.1**	-	8.4
Auckland region ²⁰	Jan 2002 to Dec 2003	Self-report	1. Cluster sample, and 2. Random sample from electoral roll	3,770	1. 61.3% 2. 65%	35-74*	3.9	12.0	19.5	-
East Coast north of Gisborne ¹¹	May 2006 to Jan 2007	Self-report	Random selection local health provider age sex register	235	47.7%	25+	-	-	-	7.6
2006/07 NZ Health Survey ⁵	Oct 2006 to Nov 2007	Self-report	Multi-stage, stratified, probability proportionate to size sample design	12,488		≥ 15†	4.3‡	5.8	10.0	5.0
Rotorua General Practice Group ²²	1 July 2007	Validated diabetes cases identified in	Invitation of Rotorua General	45,500	10 of 15 (66.7%)	0-9 10-19	0.2 0.3	0.1 0.2	0.0 0.0	

		general practice	Practice Group practices		invited general practices	20-29	0.6	0.9	0.7	
						30-39	1.0	1.8	4.6	
						40-49	2.2	5.8	10.6	
						50-59	4.8	14.9	19.2	
						60-69	9.3	22.6	27.3	
						70-79	12.3	27.8	-	
						80+	13.1	18.2	-	
						All ages ^β	3.1	7.0	8.9	
Wairoa Community Heart Study ¹²	May 2007 to Dec 2007	Self-report	Random sample Māori electoral roll	252	57.6%	Men	-	11.8	-	-
						20-64		10.7		
						Women				
						20-64				
2008/09 Adult Nutrition Survey (current study)	Oct 2008 to Oct 2009	Self-report	Multi-stage, stratified, probability proportionate to size sample design	4,721	61.0%	≥ 15	4.5 [‡]	7.0	8.1	4.9

† Crude rate; ‡ Includes other (non-Māori and non-Pacific) ethnic groups; * Age and sex standardised; ** Age-standardised to the WHO world population; § Age standardised; ^β Age standardised to the 2006 NZ population

Table 5. Reported prevalence of new diabetes for different regional and national studies in New Zealand, 1967–2009

Place	Study period	Diabetes test	Diagnostic criteria	Recruitment method	Sample size, or total recruited	Response rate (%)	Age group (years)	Prevalence <i>undiagnosed</i> diabetes (%)			
								European	Māori	Pacific	All ethnic groups
Rangiora community ¹⁴	April 1967	2 hr OGTT	Fasting blood sugar >110mg/100ml, or 1 hour > 200mg/100ml or 2 hour > 130mg/100ml or an increment of >40mg between fasting and 2hr value.		2670	93	>20	-	-	-	2.5
Single large multi-departmental workplace, Christchurch ¹⁰	Dec 1982 to Feb 1983	Random capillary blood glucose. If level ≥ 7.8 mmol/l, 2hr 75g OGTT	1980 WHO criteria	Workplace invitation	969	93	≥ 15	-	-	-	1.6
41 worksites in Auckland and 5 work sites in Tokoroa ¹⁵	May 1988 to April 1990	2 hr 75g OGTT	1985 WHO criteria	Workplace Invitation	5677	67	40-44	0.4	2.1	3.6	1.0
							45-49	1.0	7.3	1.0	1.4
							50-54	1.0	3.1	3.7	1.6
							≥ 55	1.0	6.6	6.8	2.3
							40-64 [‡]	0.8	4.6	3.6	
Dunedin general practice ⁹	Dec 1989 to June 1990	2hr 75g OGTT	1985 WHO criteria				Men	-	-	-	
							39-49				0
							50-69				3.3
							Women				
							39-49				0
50-69				0.8							
Large urban	Not stated	Random	Diabetes if fasting	Random	595	69.4	Men	-	-	-	

medical centre, Christchurch ¹³		plasma glucose and HbA1c. If glucose \geq 7.8 mmol/l or HbA1c >50 mmol/l, then 2hr 75g OGTT	glucose \geq 11.1 mmol/l at 2 hour post challenge, and at an intermediate time.	selection from practice age/sex register			65-69 70-74 75-79 80+ Women 65-69 70-74 75-79 80+					8.0 7.7 3.2 4.0 8.5 3.3 2.0 0
Three inner suburbs, South Auckland ¹⁹	1991 to 1994	Non-diabetic people with random glucose \geq 6.5 mmol/l within 2 hrs of a meal or \geq 6 mmol/l 2 hrs or more after a meal, then 2 hr 75g OGTT. Also, 20% screen negative people randomly selected for 2 hr 75g OGTT	1998 WHO criteria	Random selection all households with members aged 40-79 years	1899	69.4	40-59* 60-79*	3.3 2.7	10.6 7.9	13.7 9.1		
East Coast north of Gisborne ^{11,21}	May 2003 to Dec 2003	OGTT if negative self- report		Random selection local health provider age sex register	289	48.7	25+	-	3.6**	-		4.2
Auckland region ²⁰	Jan 2002 to Dec 2003	OGTT if negative self- report	1998 WHO criteria	1. Cluster sample, and 2. Random	3,770	1. 61.32. 65	35-74 [†]	1.8	3.8	4.0		-

				sample from electoral roll							
East Coast north of Gisborne ¹¹	May 2006 to Jan 2007	OGTT if negative self-report		Random selection local health provider age sex register	235	47.7	25+	-	-	-	3.0
Waikato District Health Board boundary, and the Ngati Tu Wharetoa tribal area ¹⁷		<i>Fasting</i> – finger-prick glucose. If glucose \geq 4.4mmol/l then 2hour 75g OGTT <i>Non-fasting</i> – random glucose and HbA1c	Screen negative if – fasting glucose \geq 5.3mmol/l, or random glucose \geq 5.3mmol/l, or HbA1c \geq 5.3%. Diabetes, IFG and IGT diagnosed using 1998 WHO criteria	Invitations from local GPs with media releases	4,269	-	Men 28-29 30-39 40-49 50-59 60-69 70-79 80+ \geq 28 Women 28-29 30-39 40-49 50-59 60-69 70-79 80+ \geq 28	-	2.3 2.3 7.7 8.4 11.2 12.4 0 6.5 2.2 2.4 3.8 5.0 8.5 7.0 0 4.2	-	-
2008/09 Adult Nutrition Survey (current study)	Oct 2008 to Oct 2009	HbA1c	American Diabetes Association criteria	Multi-stage, stratified, probability proportion-ate to size sample design	4,721	61.0	\geq 15	1.5	2.2	6.4	1.8

* Calculated by direct standardisation from the prevalence of diabetes within the screen-negative and screen-positive groups; ** Age-standardised to the WHO world population; † Age and sex standardised; ‡ Age standardised.

Table 6. Reported prevalence of *total* diabetes for different regional and national studies in New Zealand, 1967 – 2009

Place	Study period	Age group (years)	Prevalence total (<i>known and new</i>) diabetes (%)			
			European	Māori	Pacific	All ethnicities
Rangiora community ¹⁴	Apr 1967	>20	-	-	-	2.5
41 worksites in Auckland and 5 work sites in Tokoroa ¹⁵	May 1988 to Apr 1990	40-44	0.7	5.8	4.4	-
		45-49	1.7	11.3	5.8	-
		50-54	2.0	7.1	15.7	-
		55-64	3.4	17.1	12.2	-
Dunedin general practice ⁹	Dec 1989 to Jun 1990	Men	-	-	-	-
		39-49	-	-	-	0.8
		50-69	-	-	-	7.8
		Women	-	-	-	-
		39-49	-	-	-	1.9
		50-69	-	-	-	11.4
Large urban medical centre, Christchurch ¹³	Not stated	Men	-	-	-	-
		65-69	-	-	-	14.0
		70-74	-	-	-	28.2
		75-79	-	-	-	16.1
		80+	-	-	-	8.0
		Women	-	-	-	-
		65-69	-	-	-	17.0
		70-74	-	-	-	8.2
		75-79	-	-	-	11.2
		80+	-	-	-	13.8
Three inner suburbs, South Auckland ¹⁹	1991 to 1994	40-59	7.5	21.1	25.0	-
		60-79	11.2	22.8	29.1	-
East Coast north of Gisborne ^{11,21}	May 2003 to Dec 2003	25+	-	10.6**	-	12.6
Auckland region ²⁰	Jan 2002 to Dec 2003	35-74*	5.7	15.8	23.5	-
East Coast north of Gisborne ¹¹	May 2006 to Jan 2007	25+	-	-	-	10.6
2008/09 Adult Nutrition Survey (current study)	Oct 2008 to Oct 2009	15+	6.1	9.8	15.4	7.0

* Age and sex standardized; ** Age-standardised to the WHO world population.

Discussion

The 2008/09 NZANS provides for the first time reliable estimates of diabetes and prediabetes prevalence in New Zealand. Overall, the prevalence of diabetes was 7.0%, and prediabetes was 18.6%. The prevalence of diabetes was higher in men (8.3%) than women (5.8%), and was markedly higher among the obese group (14.2%) compared with that of normal weight (2.5%).

The highest prevalence of diabetes was observed among Pacific peoples, with rates among Māori in between that observed for Pacific and the NZEO groups. Rates increased with age with the highest prevalence observed for those aged 75 years and over.

The prevalence of prediabetes was high in all groups leading to an alarmingly high prevalence of a glucose metabolism disorder (diabetes or prediabetes) in working age groups. Almost 20% of those aged 35-44 years, more than 25% of those aged 45-54 years and almost 45% of those aged 55-64 years had a glucose metabolism disorder.

Diabetes prevalence surveys in New Zealand have used different methods and involved different population groups; direct comparisons are therefore not possible.^{3-5,8-21} However the data presented in Tables 4 to 6 together provide convincing evidence that the prevalence of diabetes has increased over time from the first measures in 1967 till today. This is consistent with observations world-wide.^{1,2}

The high prevalence of prediabetes observed in the 2008/09 NZANS strongly suggests that the prevalence of diabetes is likely to continue to increase for the foreseeable future. Estimates of the future burden of disease suggest that worldwide the number with diabetes will increase by 50% between 2011 and 2030; an annual growth of 2.7%.¹

The implications of increased diabetes-related morbidity, mortality and health care costs are considerable. In 2009 diabetes was the sixth leading cause of death for all New Zealanders (12.3 per 100,000 population) and the fourth leading cause of death for Māori (49.0 per 100,000 population).³² However this is an underestimate of the impact of diabetes since the condition is likely to have contributed to the other leading causes of death including ischaemic heart disease, cerebrovascular disease and some cancers.

In the present study, self-report of doctor-diagnosed diabetes was used to define diagnosed diabetes without confirmation from medical records. This approach is typically used in epidemiological studies, and has been shown to be a reasonably accurate measure of diagnosed diabetes compared with medical records.^{33,34}

Furthermore, the age-specific diagnosed diabetes rates for men in the 2008/09 NZANS were reasonably consistent with those from the VDR. For women over 55 years the age-specific rates derived from the VDR were higher than those reported in the 2008/09 NZANS and the NZ Health Survey suggesting under-reporting in the NZANS of diagnosed diabetes by women in this age category.

Overall the proportion of diagnosed to undiagnosed diabetes cases was better than the previously often quoted 1:1 ratio.³⁵ Improved diabetes detection was also observed among Māori aged 25 years and over in the 2003 Ngati and Healthy diabetes

prevalence survey (2:1 ratio)²¹ and among all three ethnic groups aged 35-74 years in the 2003 Auckland Diabetes Heart and Healthy Survey (2:1 ratio for Europeans, 3:1 for Māori and 5:1 for Pacific).²⁰ This suggests there has been good uptake of recent diabetes screening guidelines and that testing for diabetes has become more widespread. However, unlike both the NZEO and Māori groups for whom the ratio of diagnosed to undiagnosed diabetes cases was better than 3:1, among the Pacific population the ratio was almost 1:1 with 6.4% identified as having undiagnosed diabetes.

Moreover among both men and women aged <45 years in all ethnic groups, the ratio of diagnosed to undiagnosed diabetes cases was also 1:1, which indicates that those at risk are not being tested.

The NZ Society for the Study of Diabetes recommends screening obese children and young adults if there is a family history of early onset T2DM, or if they are Māori, Pacific or Indo-Asian. The high rate of prediabetes (11.6%) among those aged <45 years also indicates the need for more systematic screening in this age group, particularly as lifestyle changes can halt or delay the progression to T2DM.^{36,37}

We used the ADA criteria to define undiagnosed diabetes (HbA1c \geq 6.5% (48mmol/mol)).²⁵ Using HbA1c only to detect undiagnosed diabetes is likely to have missed some cases, which would have been identified on the basis of fasting plasma glucose criteria or following oral glucose tolerance tests (OGTT).

Where an OGTT has also been done as part of the survey testing, additional cases are identified. For example in the US, the prevalence of undiagnosed diabetes among those aged \geq 20 years was 2.25% using fasting plasma glucose, 1.58% using HbA1c, 4.52% using an OGTT and 5.41% if all three tests were used.³⁸

Similarly in the French Nutrition and Health Survey 2006-2007, the prevalence of undiagnosed diabetes for 18-74 year olds was 1.0% using fasting plasma glucose, 0.8% using HbA1c and 1.4% if both tests were used.³⁹ Therefore, it is likely that the prevalence of diabetes in the present study is underestimated.

For this study the use of the ADA prediabetes criteria allows comparisons with other countries, and highlights differences in prediabetes prevalence can be due to different cutoff points. For example, among US adults aged \geq 18 years the crude prevalence of prediabetes was 14.2% for the period 2005-2008,³⁸ whereas in France only 1.1% of adults aged 18-74 years were found to have prediabetes.³⁹

In the national French survey different criteria were used (HbA1c \geq 6% (42 mmol/l) and <6.5% (48 mmol/l)), but when both fasting plasma glucose (ADA criteria²⁵) and HbA1c were used the prevalence of prediabetes in France was 15.8%.³⁹ In the US the prevalence of prediabetes was 32.2% when the two tests were used.³⁸

The high rates of prediabetes, especially among those who are obese, is of particular concern since they herald a likelihood of continuing increases in rates of diabetes. Using the ADA HbA1c criteria for prediabetes, the 7.5 year probability of developing type 2 diabetes is 41.3% and the 10 year probability of a cardiovascular event is 13.3%.⁴⁰

The risks are even higher for those who are overweight or obese with concomitant high blood pressure or high cholesterol.⁴⁰ The identification of this at risk prediabetes

group whether by HbA1c, fasting plasma glucose or oral glucose tolerance test is important.

The implementation and long term commitment to preventive high risk and public health programmes provides the only hope of reversing the diabetes epidemic. Clinically and statistically significant delays in the progression from impaired glucose tolerance (prediabetes) to diabetes through lifestyle (dietary and physical activity) changes have been convincingly demonstrated in clinical trials.^{36, 37}

A local example, the Ngati and Healthy Prevent Diabetes project, which combined a high risk approach with a community wide approach, demonstrated a statistically significant reduction in the prevalence of insulin resistance from 38.2% to 25.6% among women aged 25-49 years over a 2-year period.¹¹ Women in this age group made more lifestyle changes than other community member groups.

The main strength of this study is that we were able to estimate the prevalence of undiagnosed diabetes and prediabetes in a nationally representative sample of New Zealand adults. The oversampling of both the Māori and Pacific populations enabled prevalence estimates to be calculated for these two ethnic groups, although small numbers in the younger age groups prevented the calculation of some age-specific rates.

While diabetes is common among Indo-Asian groups,¹ insufficient participants from this ethnic group prevented calculation of Indo-Asian-specific rates.

The relatively recent and now internationally accepted use of HbA1c as an alternative for the diagnosis of both diabetes and prediabetes has facilitated diabetes testing and screening on a large scale in the research and clinical setting without the requirement of a burdensome overnight 10-hour fast and 2-hour oral glucose tolerance test. However, as an oral glucose tolerance test was not done, it is conceivable that our reported rates of diabetes and prediabetes prevalence are underestimated. Although the prevalence of both type 1 diabetes and type 2 diabetes are increasing,^{41,42} we were unable to distinguish between these two main types of diabetes.

The response rate of 61% for the 2008/09 NZANS (44% for blood and urine samples) may be considered to be less than ideal, but for a national nutrition survey with a high respondent burden it is good. As the results were weighted so that they were representative of New Zealand's population, our estimates of diabetes and prediabetes prevalence can be considered to reasonably reliable.

Conclusions

The prevalence of diabetes in New Zealand is increasing. The high frequency of prediabetes suggests diabetes is likely to become more common, particularly in high risk groups. Implementation of effective evidence-based diabetes prevention strategies are required to reduce the increasing health and economic costs of the diabetes epidemic.

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The effectiveness of the Complete Health Improvement Program (CHIP) in Australasia for reducing selected chronic disease risk factors: a feasibility study

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Abstract

Aim To examine the effectiveness within the Australasian context of the Complete Health Improvement Program (CHIP) lifestyle intervention, which has been shown to produce meaningful reductions in selected chronic disease risk factors in the United States.

Methods Changes in body weight, blood pressure, blood lipid profile and fasting plasma glucose were assessed in 836 self-selected participants (age=55.9±12.7 yrs, 35% male/65% female) from 18 sites throughout New Zealand (N=731) and Australia (N=105).

Results In the 30 days of the program, significant overall reductions ($p < 0.001$) were recorded in the participants' body mass (-3.8%; 87.1±22.4 versus 83.9±21.5 kg), systolic blood pressure (-5.6%; 135±19 versus 127±17 mmHg), diastolic blood pressure (-4.6%; 80±12 versus 76±12 mmHg), total cholesterol (-14.7%; 5.17±1.08 versus 4.41±0.96 mmol/L), low-density lipoprotein cholesterol (-17.9%; 3.17±0.95 versus 2.60±0.83 mmol/L), triglycerides (-12.5%; 1.51±0.98 versus 1.32±0.71 mmol/L) and fasting plasma glucose (-5.6%; 5.55±1.49 versus 5.24±1.11 mmol/L). Participants at program entry with the highest classifications of total cholesterol, low-density lipoprotein, triglycerides and fasting plasma glucose experienced over 20% reductions in these measures in 30 days.

Conclusions Significant reductions in selected chronic disease risk factors were observed in 30 days using the CHIP intervention and the improvements were comparable to that observed in cohorts from the United States. The results of this feasibility study indicate that lifestyle interventions like CHIP may be useful for combating the burgeoning epidemic of chronic disease and further research is warranted.

Chronic diseases are the major cause of death and disability throughout Australasia and are a burden on sufferers, carers, communities and the population at large.¹ There is an increasing awareness that Lifestyle Medicine, which involves the application of environmental, behavioural and motivational principles to the management of lifestyle related health problems,² is efficacious for the primary, secondary and even tertiary prevention of chronic diseases.

The Complete Health Improvement Program (CHIP) is an intensive, community-based lifestyle intervention that originated in the United States and has demonstrated significant benefits for the management of cardiovascular disease,³⁻⁵ type 2 diabetes mellitus⁵ and depression.^{6,7}

The 30-day program, involving 16 group sessions, encourages participants to move towards a distinctive plant-based diet, become physical activity, abstain from substance use and practice stress management techniques.

The objective of the CHIP intervention is to educate and empower individuals towards intelligent self-care through enhanced understanding of the epidemiology and aetiology of many chronic diseases while providing the skills and support to enable positive behaviour change.³

The CHIP intervention has been delivered in a variety of community settings by both health professionals⁴ and non-health trained volunteers who were equipped with a comprehensive package for delivering the program.⁵ It is estimated that over 50,000 individuals have completed the program in the United States.⁸

The aim of this study was to examine the potential effectiveness of the CHIP intervention in the Australasian context for reducing selected risk factors for chronic disease.

Methods

The study examined the changes in selected chronic disease risk factors of 836 individuals (age = 55.9±12.7 yrs, 35% male/65% female) who chose to participate in one of 31 CHIP interventions presented in 18 locations throughout Australasia (731 participants from 14 sites in New Zealand and 105 participants from 4 Australian sites).

The programs ranged in size from 5 to 101 participants (mean group size = 26.3±23.0). Consent for the study was obtained from Avondale College of Higher Education Human Research Ethics Committee (Approval No. 20:10:07). Participants were encouraged to engage in the program in consultation with their personal health care provider.

The CHIP interventions were advertised in the local media (newspapers, radio) of the communities in which the programs were being offered and in some instances local medical practitioners recommended their patients to the program. Of the 836 participants who enrolled in the program, 790 (94%) completed the 30-day intervention after which they were encouraged to join a support group that met monthly.

Participants were deemed to have completed the program if they attended 13 of the 16 sessions and underwent both pre and post-intervention blood testing. As shown in Table 1, at program entry the participants were representative of an at-risk population with a mean BMI in the 'obese' category (31.2 kg/m²), borderline 'prediabetic' fasting plasma glucose (FPG) levels (5.55 mmol/L), and elevated systolic blood pressure (134.8 mmHg) and low-density lipoprotein (LDL) cholesterol levels (3.17 mmol/L).

The programs were facilitated by volunteer directors (age = 55.1±9.5, 5 males/13 females) who had an interest in positively influencing the health of members of their local community. The volunteer directors were not required to be health professionals, although 6 of the 18 were.

All directors underwent two days of training to develop group facilitation skills after which they were provided with a comprehensive CHIP resource package that included a curriculum guide for program delivery, 16 pre-recorded educational lectures presented by qualified experts, a cookbook and participant textbook and journal. The role of the volunteer director was to organise and facilitate the proceedings of the group sessions, not to educate. Even in the case that the director had medical training, the supplied resources were used and the program delivery was consistent.

The CHIP intervention involved 16 two-hour group sessions over 30 days. Each session typically involved viewing a one-hour pre-recorded lecture, a cooking demonstration, group discussion and a behaviour change challenge. Also incorporated into the program when local health experts could be sourced were shopping tours, nutrition workshops and guided exercise sessions. Participants paid a fee of approximately \$250 to cover the cost of venue hire, food samples distributed throughout the program, resources including reading materials and pedometer, and biomedical assessments.

Table 1. Mean changes in selected chronic diseases risk factors from baseline to post-intervention

Factor	N	Baseline Mean (SD)		Post-intervention Mean (SD)		Mean Change	% Change	t statistic	p value
Weight (kg)	790	87.1	(22.4)	83.9	(21.5)	-3.2	-3.8%	37.5	<0.001
BMI (kg/m ²)	718	31.2	(7.7)	30.0	(7.4)	-1.2	-3.8%	37.2	<0.001
SBP (mmHg)	787	134.8	(19.0)	127.4	(16.7)	-7.6	-5.6%	13.4	<0.001
DBP (mmHg)	787	80.0	(11.5)	76.3	(11.5)	-3.7	-4.6%	10.7	<0.001
TC (mmol/L)	779	5.17	(1.08)	4.41	(0.96)	-0.76	-14.7%	30.0	<0.001
LDL (mmol/L)	775	3.17	(0.95)	2.60	(0.83)	-0.57	-17.9%	26.1	<0.001
HDL (mmol/L)	779	1.32	(0.36)	1.21	(0.32)	-0.11	-8.3%	15.8	<0.001
TG (mmol/L)	778	1.51	(0.98)	1.32	(0.71)	-0.19	-12.5%	6.7	<0.001
FPG (mmol/L)	772	5.55	(1.49)	5.24	(1.11)	-0.31	-5.6%	7.6	<0.001

BMI – Body mass index; SBP – systolic blood pressure; DBP – diastolic blood pressure; TC – Total cholesterol; LDL – low density lipoprotein; HDL – high density lipoprotein; TG – triglycerides; FPG – fasting plasma glucose; SD – Standard deviation.

The CHIP intervention advocated daily exercise (30 minutes of moderate intensity or 10,000 steps) and included elements of positive psychology, but nutrition was the focus of the program. The intervention advocated a distinctive eating pattern as participants were encouraged to move towards a whole food, plant-based diet *ad libitum*, with emphasis on the consumption of whole-grains, legumes, fresh fruits and vegetables. This diet was recommended in order to achieve a daily target of fewer than 20% of calories from fat and less than 10 teaspoons of added sugar, 5,000 mg of salt (2000 mg of sodium) and 50 mg of cholesterol. Participants were also encouraged to consume 2 - 2.5 litres of water daily.³

At the beginning and end of the program the participants' height, weight, blood pressure and 12-hour fasting blood samples were taken. The blood samples were collected by trained phlebotomists and analysed by local pathology laboratories for total cholesterol (TC), low-density lipoprotein (LDL), high-density lipoprotein (HDL), triglycerides (TG) and fasting plasma glucose (FPG) levels.

Statistical analysis—The data were analysed using PASW™ Statistics (version 18) software. Data are expressed as mean ± standard deviation. Paired t-tests were used to assess changes in the biometric measures from baseline to post-intervention, both for the overall and stratified data. McNemar Chi-squared test was used to determine changes from program entry to post-intervention in the distribution of participants across the various risk factor categories. Cohen's d statistic was calculated to present effect size.

Results

The participants' mean changes from baseline to post-intervention are presented in Table 1. Significant reductions were recorded in all the biometrics with the most notable being in TC, LDL and TG. While HDL also decreased following the intervention, the TC to HDL ratio improved from 3.92:1 to 3.64:1 (p<0.001). Table 2 displays the stratified data using conventional risk factor categories. The National Cholesterol Education Program Adult Treatment Panel III classification system⁹ was used to categorise the participants for all risk factors except TC. The Framingham classification¹⁰ was used for the TC data as it includes five categories, compared to three in the ATP III classification, thus allowing a more detailed analysis of the effect of the intervention on the highest risk participants.

Table 2. Changes in chronic disease risk factor levels within 30 days according to initial risk factor classification

Risk Factor	N Baseline	N Post-intervention	Chi- squared* (p)	Baseline Mean (SD)	Post- intervention Mean (SD)	Mean Change	% Mean Change	p value	Cohen's d
BMI (kg/m²)									
18.5–24.9	137	168	78 (<0.001)	22.7 (1.6)	22.2 (1.6)	-0.6	-2.5%	<0.001	0.313
25–30	216	234		27.5 (1.4)	26.5 (1.4)	-1.0	-4.8%	<0.001	0.714
> 30	350	301		36.9 (7.0)	35.5 (6.9)	-1.4	-3.8%	<0.001	0.201
SBP (mmHg)									
<120	189	312	120 (<0.001)	112.9 (7.6)	114.2 (11.4)	1.3	1.2%	0.119	-0.134
120–139	314	297		130.2 (4.9)	125.1 (12.6)	-5.1	-3.9%	<0.001	0.533
140–160	226	151		148.9 (6.6)	136.5 (15.4)	-12.4	-8.3%	<0.001	1.047
>160	58	27		177.1 (12.7)	147.4 (16.5)	-29.7	-16.8%	<0.001	2.017
DBP (mmHg)									
<80	349	446	80 (<0.001)	70.1 (6.5)	70.2 (8.9)	0.1	0.1%	0.872	-0.013
80–89	277	258		82.9 (2.8)	78.2 (8.5)	-4.6	-5.5%	<0.001	0.743
90–100	133	63		93.5 (3.6)	84.2 (9.7)	-9.4	-10.1%	<0.001	1.271
>100	28	20		108.8 (5.3)	93.7 (10.9)	-15.1	-13.9%	<0.001	1.762
TC (mmol/L)									
< 4.00	93	268	407 (<0.001)	3.49 (0.40)	3.23 (0.55)	-0.26	-7.4%	<0.001	0.541
4.00–5.20	334	371		4.62 (0.37)	4.03 (0.59)	-0.59	-12.7%	<0.001	1.198
5.21–5.99	172	94		5.59 (0.21)	4.74 (0.63)	-0.85	-15.2%	<0.001	1.810
6.00–6.99	143	40		6.39 (0.29)	5.23 (0.74)	-1.16	-18.2%	<0.001	2.064
>7.00	37	6		7.62 (0.49)	6.01 (0.99)	-1.61	-21.1%	<0.001	2.061

LDL (mmol/L)											
<2.50	215	381	296 (<0.001)	2.06	(0.41)	1.80	(0.50)	-0.26	-12.6%	<0.001	0.569
2.50-2.99	140	171		2.79	(0.14)	2.39	(0.48)	-0.40	-14.3%	<0.001	1.131
3.00-4.00	271	181		3.49	(0.29)	2.84	(0.58)	-0.64	-18.3%	<0.001	1.418
>4.00	149	42		4.55	(0.45)	3.50	(0.72)	-1.05	-23.1%	<0.001	1.789
HDL (mmol/L)											
<1.00	147	201	96 (<0.001)	0.86	(0.10)	0.85	(0.12)	-0.01	-1.2%	0.546	0.091
1.00-1.55	439	470		1.26	(0.16)	1.17	(0.19)	-0.09	-7.1%	<0.001	0.512
>1.55	193	108		1.81	(0.23)	1.58	(0.29)	-0.24	-13.3%	<0.001	0.879
TG (mmol/L)											
<1.00	233	282	38 (<0.001)	0.75	(0.15)	0.82	(0.29)	0.08	10.7%	<0.001	-0.303
1.00-2.25	433	428		1.47	(0.33)	1.33	(0.48)	-0.15	-10.2%	<0.001	0.340
>2.25	112	68		3.21	(1.47)	2.31	(0.98)	-0.91	-28.3%	<0.001	0.720
FPG (mmol/L)											
<5.60	530	605	55 (<0.001)	4.91	(0.42)	4.87	(0.56)	-0.04	-0.8%	0.067	0.081
5.60-7.00	177	129		6.02	(0.39)	5.53	(0.55)	-0.48	-8.0%	<0.001	1.028
>7.00	65	38		9.47	(2.28)	7.42	(2.27)	-2.05	-21.6%	<0.001	0.901

BMI – Body mass index; SBP – systolic blood pressure; DBP – diastolic blood pressure; TC – Total cholesterol; LDL – low density lipoprotein; HDL – high density lipoprotein; TG – triglycerides; FPG – fasting plasma glucose; SD – Standard deviation. * McNemar Chi-squared test. SD – Standard deviation.

Participants who presented to the program with the highest risk factor classifications tended to experience the greatest improvements and the effect sizes were large.

Participants who entered the program with TC levels above 5.2 mmol/L experienced a mean reduction of 1.05 mmol/L which, according to the algorithm generated through meta-analysis by Gould and colleagues,¹¹ would result in a 19% decrease in the relative risk for all-cause mortality, a 26% reduced risk for coronary heart disease related mortality and a 31% reduced risk of a cardiac event.

As shown in Table 2, many of the participants who presented with the highest risk factor classifications at program entry had moved to lower risk factor classifications by the end of the intervention. Only 6 of the 39 individuals with TC levels above 7.0 mmol/L at program entry maintained these levels post-intervention. Of the 68 individuals with FPG levels indicative of diabetes at baseline, 30 (44%) reduced their scores below 7.0 mmol/L in the 30 days.

A comparison of the risk factor reductions observed in this study with those recently reported in a cohort of over 5,000 CHIP participants from the United States⁵ is presented in Table 3. Clearly, similar outcomes were observed in this study and the United States cohort.

Table 3. Comparison of the mean changes in selected chronic disease risk factors from baseline to post-intervention in the present study and CHIP participants from the United States

Factor	% Change	
	Australasian CHIP (N=787)	US CHIP* (N=5070)
BMI	-3.8%	-3.2%
SBP	-5.6%	-4.9%
DBP	-4.6%	-5.3%
TC	-14.7%	-11.0%
LDL	-17.9%	-13.0%
HDL	-8.3%	-8.6%
TG	-12.5%	-7.7%
FPG	-5.6%	-6.1%

BMI – Body mass index; SBP – systolic blood pressure; DBP – diastolic blood pressure; TC – Total cholesterol; LDL – low density lipoprotein; HDL – high density lipoprotein; TG – triglycerides; FPG – fasting plasma glucose.
* From Rankin et al.⁵

While the low numbers in some of the program groups did not allow statistical comparisons, there was considerable variability between the groups in the extent of change observed in the outcome measures.

Discussion

The findings of this Australasian study supports data from the United States, suggesting that the CHIP lifestyle intervention appears to produce meaningful reductions in selected chronic disease risk factors in the Australasian context. The

outcomes observed were comparable between both regions, with the greatest reductions among those with the greatest risk. However, caution in the interpretation of the findings is required because of a number of limitations.

Several confounders may explain the magnitude of the changes in the chronic disease risk factors observed in this feasibility study. Firstly, as the participants were self-selected, they likely entered the program with an elevated readiness for change and hence willingness to engage in the intervention.

In accordance with the transtheoretical model of behaviour change,¹² a key objective of the first few sessions of the CHIP intervention is to move participants from pre-contemplation to action. Yet the participants were probably beyond the pre-contemplation stage at program entry. It would be interesting to compare the outcomes observed in this study with participants who had not shown an initial interest in the program.

Secondly, in the absence of a control group, the extent to which regression to the mean explains the observed improvements cannot be determined. Consistent with regression to the mean is that the individuals with the most extreme baseline measures tended to experience the greatest improvements and hence inclination towards the norm. However, given the large size of the sample and that in some of the outcomes measures the high risk classifications moved 1.5 to 2 standard deviations, regression to the mean likely only explains a small component of the observed results.

Noteworthy, several studies of the CHIP intervention in the United States have demonstrated the effectiveness of the program using a randomised control design and the magnitude of change observed in the present study is similar to the treatment groups of these studies.⁴ Certainly, a randomised control trial is needed in the Australasian setting to extend upon the work done in this feasibility study.

The final potential confounder of the outcomes observed in this study is the Hawthorne effect. While the research team were not responsible for conducting all the interventions, the participant's behaviours and level of engagement with the program was undoubtedly influenced by the blood measures taken pre and post-intervention.

Given that the pre and post blood work is a standard component of the CHIP intervention, improvements achieved as a result of these accountability measures could be considered part of the intervention itself. However, further research is needed to elucidate the influence of the unique lifestyle recommendations of the CHIP intervention—namely its emphasis on a whole-food, plant-based eating pattern—from the motivational properties of the pre and post-intervention measurements made on the participants. Certainly, the inclusion of accountability measures is likely to be an important component of lifestyle interventions targeting chronic disease.

Notwithstanding the limitations in the research design, the results of this feasibility study are noteworthy given the size of the sample and the large effect sizes observed. Indeed, the results of this study indicate that the CHIP intervention shows promise for the management of chronic diseases in the Australasian context.

It is noteworthy that in only 30 days over 20% improvement was observed in the participants with the highest classifications of TC (21%), LDL (23%), TG (28%) and FPG (22%). The changes in TC and LDL compare favourably to those achieved by

pharmaceutical interventions involving statins¹¹ and far exceed the typical expectations of dietary interventions for lowering blood lipids.¹³

The large changes observed with the intervention is likely a result of the dietary recommendations of CHIP being more extreme than conventional guidelines. Despite its rigor, the participants anecdotally reported a high level of acceptability of the eating pattern, which was probably enhanced because the diet was not calorically restrictive and hence the participants were satiated.

In the United States, Barnard and colleagues¹⁴ reported similar levels of acceptability of a plant-based eating pattern to the more moderate diet recommended by the American Dietetic Association, although acceptability needs to be determined in the Australasian context.

As described in the results section, a decrease in TC and LDL would offer substantial cardio-protection and reduce the relative risk for all-cause mortality. However, while the observed decrease in TC and LDL are beneficial, the reduction in HDL appears counterproductive. Noteworthy, individuals who adopt a whole-food, plant-based eating pattern, which is free from exogenous cholesterol and low in saturated fat, typically have lower blood concentrations of all cholesterol subfractions, including HDL.¹⁵

Notwithstanding, these individuals do not have compromised cardiovascular health and are not at increased risk of type 2 diabetes mellitus.¹⁵ In fact, in the Lifestyle Heart Trial¹⁶ that prescribed a low-fat, plant-based diet, participants experienced regression of atherosclerotic plaque and a reduction in cardiac events despite a concomitant decrease in HDL levels.

The lowered HDL levels associated with a plant-based eating pattern may be explained by less need for reverse cholesterol transport. Importantly, despite the decrease in HDL observed in the present study, the TC to HDL ratio improved.

There were several anecdotal reports in the present study of participants' having their medications (e.g. hypertensive, hypercholesterolemia, hyperglycaemic) decreased or even ceased by their personal doctor during the course of the 30-day intervention. While this is a desirable outcome, a reduction in medication usage may have caused the results presented in this report to be understated. It is a limitation of this study that medication changes were not recorded and this should be included in future studies.

Given that many chronic diseases have lifestyle underpinnings,¹ there is a growing awareness that lifestyle interventions have merit at all levels of prevention. In terms of primary prevention, results of the 52 country INTERHEART study¹⁷ indicated that positive lifestyle practices, such as the consumption of fruits and vegetables, being physically active and avoiding the use of tobacco, can prevent up to 90% of myocardial infarctions.

With regards to secondary prevention, the Diabetes Prevention Program Research Group¹⁸ showed a 16-session lifestyle education program to be twice as effective as pharmaceuticals (metformin) for preventing at-risk people with pre-diabetes developing established diabetes. At the tertiary level, the potential for disease reversal is emerging as an area of interest in the field of lifestyle medicine.^{16,19-22}

Several studies have explored the potential of lifestyle interventions for chronic disease reversal and most have centred around a whole-food, plant-based diet high in fibre (>30 grams) and low in fat (<20%), cholesterol and refined sugar. Esselstyn¹⁹ showed regression of heart disease using a low-fat (<10%) plant-based diet alone, while the Lifestyle Heart Trial¹⁶ demonstrated cardiovascular disease reversal through plant-based nutrition combined with exercise, social support and stress management techniques.

Similarly, the role of lifestyle in potential reversal of type 2 diabetes has been known for over 30 years.^{20,21} Barnard and associates²² reported ~40% of people with type 2 diabetes treated with insulin could discontinue its use through participation in a 26-day residential program involving a near vegetarian, low-fat diet in conjunction with exercise.

In the present study, over 40% of participants who entered the program with FPG levels indicative of diabetes reduced their levels below this classification in 30 days. This observation is comparable to our recent report of over 5,000 CHIP participants from the United States.⁵

The results observed in this study of a free-living population are encouraging for a 30-day intervention, however, the question of sustainability remains. Maintenance of behaviour change following involvement in the CHIP intervention has been documented for up to 18 months in the United States²³ but a sustainability study in the Australasian context is needed.

Anecdotally there are numerous case reports of individuals involved in this project who experienced continued, and even profound, health improvements beyond their involvement in the CHIP intervention. However, achieving long-term compliance to interventions that aim to improve patient outcomes over time is a challenge. This is the case for both lifestyle or pharmaceutical interventions.^{24,25}

While the CHIP intervention incorporates elements to promote long-term health behaviour change—including education, social support, accountability measures, and a focus on one's environment and how to re-engineer it to support positive lifestyle choices—questions surrounding how to optimise engagement with lifestyle interventions need to be further explored.

A greater understanding of what makes lifestyle interventions most efficacious is required. For example, while improvements in participants' biometrics were recorded for all groups involved in this study, some groups appeared to achieve better outcomes than others. Given the intervention was essentially the same for all groups in terms of duration, intensiveness and content, the varying outcomes could conceivably be explained by factors relating to either the group participants and/or elements of the program that did vary between the groups.

Participant factors may include age, gender, social class, ethnicity, who they participated in the intervention with, baseline health status and the extent of engagement with the intervention and the lifestyle recommendations it advocated.

Other factors may include group size, social class, venue or setting, and characteristics relating to the facilitator such as their level of training and experience conducting lifestyle interventions. How these factors contribute to the success of a

lifestyle intervention remains to be elucidated. Further, it would be interesting to study the outcomes of a CHIP intervention conducted in a less intense manner, given that the current program involves 16 sessions over a one-month period.

A unique element of the study was the use of volunteers to administer a professionally generated lifestyle intervention. Only one third of the facilitators involved in this study had medical or health training, but regardless, all underwent two days of training that focused on the logistics of administering the program and providing them with group facilitation skills. It was mandated that the facilitators not provide lifestyle counsel as this was presented in the pre-recorded educational presentations provided as part of the CHIP resource.

Harnessing the energy of volunteers to facilitate lifestyle interventions, as employed in this study, represents a potentially powerful and cost-effective mode for administering lifestyle interventions. Many of the volunteer directors of the programs in this study had previously participated in a CHIP intervention and therefore had a strong investment and bond with the program. Indeed, passionate volunteers can be powerful agents of change and possess motivational properties to incite their peers to action.²⁶

Conclusions

The results of this feasibility study indicate that lifestyle medicine programs like the CHIP intervention show promise for the management of selected chronic diseases within the Australasian context. Further, volunteers can be valuable social capital in the combat of chronic diseases by facilitating well-designed and appropriately resourced lifestyle interventions.

A randomised control trial is warranted that investigates the effectiveness and sustainability of the lifestyle choices acquired during the CHIP intervention and the associated long-term reductions in chronic disease risk factors. A further investigation of the acceptability of the specific nutritional recommendations of the CHIP intervention in the Australasian setting is also required.

Finally, in order to optimise the outcomes achieved by lifestyle interventions such as the CHIP, research is required to elucidate how factors relating to the participant as well as the structure, content and facilitation of the program contribute to the success of the intervention.

Competing interests: Nil.

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Binge drinking among Māori secondary school students in New Zealand: associations with source, exposure and perceptions of alcohol use

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Abstract

Aim Describe factors associated with binge drinking among Māori secondary school students.

Method Analysis of Māori sample (n=1702) from the 2007 national youth health survey.

Results Among current drinkers, 31.5% reported binge drinking (5–9 drinks) and 30.4% reported heavy binge drinking (≥ 10 drinks) in a 4-hour session in the past four weeks. Compared with non-binge drinkers, binge drinkers more frequently reported 'drinking alcohol was okay for people their age' ($OR_{\text{binge}} = 1.9$; $OR_{\text{heavy binge}} = 2.4$), $p < 0.0001$), had friends that drank alcohol ($OR_{\text{binge}} = 2.4$; $OR_{\text{heavy binge}} = 4.0$, $p < 0.0001$), had sourced alcohol from friends ($OR_{\text{binge}} = 1.7$; $OR_{\text{heavy binge}} = 1.2$, $p = 0.002$) or from 'other adults' ($OR_{\text{binge}} = 1.6$; $OR_{\text{heavy binge}} = 1.7$; $p = 0.0004$) and buy their own alcohol ($OR_{\text{binge}} = 1.7$; $OR_{\text{heavy binge}} = 2.8$, $p < 0.0001$). Binge drinking was associated with poorer school performance, unsafe sex, unwanted sex, an injury, injuring someone else, motor vehicle crashes and 'doing things that could cause trouble'. Binge and heavy binge drinkers reported greater difficulty accessing drug and alcohol services ($OR_{\text{binge}} = 2.30$; $OR_{\text{heavy binge}} = 4.97$ $p < 0.0001$).

Conclusion Binge drinking is associated with a range of poor health and social outcomes for Māori youth. The associated poorer access to drug and alcohol services reveals an inequity requiring priority attention.

Alcohol is responsible for significant morbidity and mortality amongst young people.^{1–9} Although Māori drink less frequently than other ethnic groups,¹⁰ the usual quantity consumed per occasion is higher^{11,12} resulting in disproportionately higher levels of alcohol-related harm. Māori attending secondary school have the highest prevalence of 'ever trying alcohol' and binge and heavy binge drinking behaviours than any other ethnic group in New Zealand.¹³

Health behaviours and attitudes established during adolescence often carry over to adulthood.¹⁴ Therefore, addressing excessive episodic drinking patterns during adolescence will help to reduce excess morbidity and mortality, and also establish healthier lifetime drinking behaviours.¹⁵

This article investigates factors and outcomes associated with patterns of alcohol use among Māori youth in secondary schools throughout New Zealand in order to inform future research and interventions to reduce inequalities in alcohol-related harm.

The aims of these Māori specific analyses were to:

- Determine the prevalence of alcohol-related outcomes and sources of help for young Māori drinkers;
- Explore the influence of individual attitudes, peer and parental drinking behaviours on drinking patterns; and
- Identify the associations between source of alcohol and drinking patterns.

Methods

Study design—These analyses utilised data from Youth'07, a nationally representative self-administered anonymous health and well-being survey of 9,107 New Zealand secondary schools students conducted in the 2007 school year. The survey utilised a two-stage sampling design with 115 schools randomly selected to participate from the 389 eligible secondary schools in New Zealand.

In each participating school either 18% of eligible Year 9–13 students, or 30 students if the roll was less than 166, were randomly selected from the school roll and invited to participate. There was an 84% school response rate and 74% student response rate. The survey contained 622 questions about a wide range of health and wellbeing issues, including alcohol use. Students completed the questionnaire employing an anonymous multimedia computer-assisted interview (M-CASI) technology using handheld Internet tablets administered by a research team.¹⁶

The M-CASI technology aims to increase the reliability of the data and minimise concerns regarding social desirability of responses.^{17,18} A detailed description of the methodology for Youth'07 is available elsewhere.¹⁹

Definition of terms and measures—Age, gender and ethnicity were determined by self-report. Age was dichotomised into junior secondary school students (aged 12–14 years) and senior secondary school students (aged 15–18 years). Ethnicity was sought from students using the New Zealand census ethnicity question²⁰ and prioritised to one of five major ethnic groups: Māori, Pacific, Asian, Other ethnicity, New Zealand European. Only students who identified themselves as Māori are included in these analyses. Participants were assigned to a level of area deprivation by linking their residential mesh-block number to the 2006 New Zealand Deprivation Index²¹, categorised into three groups reflecting low deprivation (1–3), middle levels of deprivation (4–7), and high deprivation (8–10). The geographical areas where students resided were classified as 'urban' (more than 1,000 residents) or 'rural' (less than 1,000 residents).²²

Access to healthcare was measured with the following questions: "If you had problems or concerns due to alcohol or drug use, who would you go to, to get help?" (school guidance counsellor, friends, teachers, parents, school nurse, family doctor, drug and alcohol service, other, I wouldn't look for help). "In the last 12 months, has there been any time when you wanted or needed to see a doctor or nurse (or other healthcare worker) about your health, but weren't able to?" (yes, no). "In the last 12 months, have you had difficulty getting help for any of the following (help with stopping drug or alcohol use)" (yes, no).

Attitudes toward alcohol and exposure to alcohol use were measured with the following questions: "Which of the following do you think are ok for people your age to use regularly?" (alcohol e.g. beer, wine, spirits etc). "Which of the following do your friends use?" (alcohol e.g. beer, wine, spirits etc), "Which of the following do your parents or parent use in your home?" (alcohol e.g. beer, wine, spirits etc).

Sources of alcohol was measured using the following question: "When you drink alcohol, how do you usually get it?" (I buy it myself, friends give it to me, my brothers or sisters give it to me, my parents give it to me, I take it from home, another adult I know gives it to me, I get someone else to buy it for me, I pinch it, none of these).

Self-reported problems associated with drinking was measured with the following question: "How many times in the past year have you...(had friends or family tell you to cut down your alcohol drinking?, had your performance at school or work affected by your alcohol use?, had unsafe sex (no condom) after you had been drinking?, had unwanted sex after you had been drinking alcohol?, done things that could have got you into serious trouble (e.g. stealing)?, been injured after you had been

drinking?, been injured and required treatment by a doctor or nurse after drinking?, injured someone else after you had been drinking alcohol?, had a car crash after you had been drinking alcohol?)(yes, no).

Current drinkers are defined as students who stated they had consumed alcohol and did not indicate they no longer drank. The usual pattern of drinking was taken from the question, “How many alcoholic drinks do you usually have in one session, within 4 hours? (count one drink as one small glass of wine, one can or stubbie, one RTD)?” This was categorised into three groups: *Non-binge drinking* was defined as usually consuming 1-4 standard alcoholic drinks within one 4-hour drinking session. *Binge drinking/drinkers* was defined as usually consuming 5 to 9 alcoholic drinks within one 4-hour drinking session as defined within the ALAC monitor survey.²³ *Heavy binge drinking/drinkers* was defined as usually drinking 10 or more alcoholic drinks within one 4-hour drinking session.

Analysis—Frequencies and 95% confidence intervals were calculated to describe the distributions of pattern of drinking and the other alcohol variables of interest among Māori students by gender, age group, deprivation level, urban/rural location. Chi-squared tests (Rao-Scott) were used to test associations between pattern of drinking and self-reported harms and problems. Separate multinomial regression models (incorporating clustering and weighting) were used to investigate the associations between the patterns of drinking and the attitudes and exposure to alcohol use, source of alcohol and difficulty accessing services. These models included demographic and SES variables (NZDep, worrying about not having enough food and moving home frequently).

All students were included in the descriptive analysis of attitudes and exposure to alcohol use; however, only current drinkers were included in analyses of the associations with pattern of drinking. All analyses were conducted using the survey procedures, Surveyfreq and Surveylogistic (generalised logits link) in the SAS software v9.2.²⁴ This enabled us to use weighting to account for the unequal probabilities of selection of students within schools and adjust the variances to allow for clustering by school. The findings are interpreted within a kaupapa Māori^{25,26} and positive youth development.^{27,28} framework acknowledging the self-determination and the aspirations of Māori youth and their whānau/families.

Results

Demographic characteristics of Māori sample

There were 1702 Māori students in the Youth’07 sample accounting for 18.7% of the total secondary school sample (Table 1). Approximately one half of the participants were male (51.8%) with an equal distribution of junior (49.8%) and senior students (50.2%). Proportionately more Māori students lived in areas of mid (34.9%) and high (44.9%) socioeconomic deprivation. Most Māori students (83.4%) lived in urban/semi-urban settings (Table 1).

Table 1. Demographic features of Māori students in Youth ’07

Variables		Māori Male		Māori Female		Total Māori		
		n	%	N	%	N	%	
Total		882	51.8	820	48.2	1702	100	
Age (years)	Junior	443	50.2	405	49.4	848	49.8	
	Senior	439	49.8	415	50.6	854	50.2	
Urban		702	82.1	678	84.9	1380	83.4	
Rural		153	17.9	121	15.1	274	16.6	
Deprivation Index (NZDep2006)		Low	158	18.5	176	22.0	334	20.2
		Mid	308	36.0	269	33.7	577	34.9
		High	389	45.5	354	44.3	734	44.9

Table 2. Attitudes and exposure to alcohol use

Variables		Think it is okay to regularly use alcohol		Friends use alcohol		Parent/s use alcohol in your home	
		n /N	% (95% CI)	n /N	% (95% CI)	n /N	% (95% CI)
Total		688/1585	43.5 (40.5–46.5)	1233/1578	78.2 (75.2–81.2)	1027/1572	65.5 (62.3–68.6)
By Gender	Male	340/818	41.8 (38.3–45.3)	584/813	72.0 (67.9–76.0)	492/806	61.2 (57.2–65.2)
	Female	348/767	45.3 (41.6–49.0)	649/765	84.9 (81.9–88.0)	535/766	70.0 (65.6–74.4)
By Age	Junior	254/789	32.2 (29.2–35.3)	527/784	67.2 (62.7–71.7)	485/781	62.3 (58.3–66.2)
	Senior	434/796	54.6 (50.5–58.6)	706/794	89.0 (86.3–91.6)	242/791	68.6 (64.3–72.8)
By NZDep	Low	150/317	47.4 9 (42.0–52.9)	231/316	73.1 (68.1–78.1)	222/317	70.1 (65.1–75.2)
	Medium	249/549	45.4 (40.7–50.1)	439/548	80.3 (76.0–84.5)	380/543	70.1 (65.7–74.6)
	High	280/699	40.1 (35.9–44.4)	550/695	79.2 (75.7–82.7)	411/693	59.4 (55.4–63.4)
Urban & Rural	Urban	561/1305	43.0 (39.6–46.4)	1021/1302	78.6 (75.2–81.9)	834/1294	64.5 (61.0–68.1)
	Rural	118/260	45.8 (39.5–52.2)	199/257	77.3 (71.8–82.7)	179/259	69.4 (64.0–74.8)
Drinking status (among those who have ever drunk alcohol)	Not Current drinker	61/402	15.20 (11.36 –19.03)	207/400	51.62 (45.92–57.32)	174/401	43.51 (37.78–49.23)
	Current drinker	606/1106	54.80 (51.54–58.06)	983/1103	89.23 (87.42–91.03)	983/1103	75.19 (72.49–77.89)

Table 3. Alcohol use patterns by gender, age bracket, group, socioeconomic deprivation (NZDep) and urban/rural residence

Variables		Ever used alcohol		Non-binge (1–4 standard drinks within one 4-hour session)		Binge alcohol use (5 to 9 standard drinks within one 4-hour session)		Heavy binge alcohol use (usual pattern of drinking is 10 or more alcoholic drinks within one 4-hour drinking session)	
		n/N	% (95% CI)	n/N	% (95% CI)	n/N	% (95% CI)	n/N	% (95% CI)
Total		1290/1522	84.7 (82.1–87.3)	422/1106	38.1 (34.3–41.8)	348/1106	31.5 (28.4–34.7)	336/1106	30.4 (27.3–33.4)
By Gender	Male	646/777	83.3 (80.0–86.6)	187/549	33.9 (29.1–38.7)	170/549	31.0 (25.9–36.2)	192/549	35.0 (31.1–39.0)
	Female	644/745	86.3 (82.9–90.0)	235/557	42.1 (37.4–46.9)	178/557	32.1 (27.9–36.2)	144/557	25.8 (21.9–29.7)
By Age	Junior	579/750	77.2 (73.5–81.0)	245/466	52.7 (47.8–57.6)	120/466	25.8 (22.1–29.5)	101/466	21.5 (17.5–25.4)
	Senior	711/772	92.0 (89.5–94.5)	177/640	27.4 (23.3–31.5)	228/640	35.7 (31.7–39.7)	235/640	36.9 (32.9–40.8)
By NZDep	Low	266/311	85.5 (81.7–89.3)	109/234	46.6 (39.2–54.0)	70/234	29.9 (24.0–35.8)	55/234	23.5 (17.5–29.4)
	Medium	458/532	86.2 (82.9–89.5)	155/406	38.2 (32.9–43.4)	129/406	31.8 (26.8–36.7)	122/406	30.1 (25.3–34.9)
	High	553/661	83.5 (79.6–87.4)	155/453	34.0 (29.9–38.1)	143/453	31.8 (27.4–36.1)	155/453	34.3 (30.1–38.4)
Urban/rural	Urban	1052/1252	84.0 (81.1–87.0)	337/897	37.4 (33.5–41.4)	291/897	32.5 (29.1–36.0)	269/897	30.0 (26.8–33.2)
	Rural	225/252	89.2 (84.9–93.6)	82/196	41.8 (35.0–48.7)	51/196	26.0 (19.1–32.8)	63/196	32.2 (25.4–39.0)

Description of alcohol use and patterns among Māori students

Attitudes toward alcohol and exposure to alcohol use—43.5% of all Māori students perceived that it was okay for people their age to drink alcohol regularly with no differences found by gender, deprivation level and urban/rural location (Table 2).

Thinking it was okay to drink alcohol was more common among senior secondary school students (32.2% junior, 54.6% senior). Most students reported that their friends drank alcohol (78.2%), with females and senior students more frequently reporting their friends drank alcohol.

Parental alcohol use in the family home was reported by 65.5% of students, with female students more frequently reporting parental drinking (61.2% males, 70.0% females). Students who were ‘not current drinkers’ were less likely to think that alcohol was okay for people their age to use, and reported fewer friends or family members who drink alcohol.

Pattern of alcohol consumption—84.7% of all Māori students had ever used alcohol, and 73.3% reported that they were current drinkers. There were no differences by gender, deprivation level or urban/rural location, but senior students more frequently reported ‘ever having drunk alcohol’. Of all students who were current drinkers, almost one-third (31.5%) usually consumed 5–9 standard drinks within one 4-hour drinking session (binge drinking) and a further 30.4% usually consumed ten or more standard drinks on a single drinking occasion (heavy binge drinking). Binge drinkers were more frequently senior students and heavy binge drinkers were more likely to be male, senior students and those from areas of high deprivation than areas of low deprivation.

Table 4. Results from individual multinomial models controlling for age, gender and socioeconomic variables

Variables	Binge versus non binge	Heavy binge versus non binge	Overall probability of variable being associated
Attitudes and exposure			
Think it is OK for people your age to use to drink alcohol regularly	1.9 (1.4–2.5)	2.4 (1.8–3.3)	p<0.0001
Friends use alcohol	2.4 (1.4–4.1)	4.0 (2.1–7.5)	p<0.0001
Parents use alcohol in home	0.87 (0.6–1.2)	0.98 (0.7–1.3)	p=0.70
Access			
Had difficulty getting help with stopping drug or alcohol use in last 12 months	2.30 (1.22–4.34)	5.0 (2.71–9.13)	p<.0001
Unable to access health professional in last 12 months	1.38 (0.96–1.99)	1.65 (1.05–2.58)	p=0.09
Source			
Buy it myself	1.7 (1.1–2.6)	2.8 (1.8–4.3)	p<0.0001
Friends give it to me	1.7 (1.3–2.2)	1.2 (0.9–1.8)	p=0.002
Parents give it to me	0.7 (0.5–1.0)	0.8 (0.6–1.0)	p=0.08
Another adult gives it to me	1.6 (1.1–2.2)	1.7 (1.3–2.3)	p=0.0004

Multinomial regression models were used to investigate the association between attitudes, observed peer and parental drinking and the pattern of drinking whilst controlling for socioeconomic variables, age group and sex) (Table 4).

Thinking it was okay to drink alcohol ($OR_{\text{binge}} = 1.9$, $OR_{\text{heavy binge}} = 2.4$) and having friends who used alcohol ($OR_{\text{binge}} = 2.4$, $OR_{\text{heavy binge}} = 4.0$) were both associated with increased odds of binge drinking. Having parents who drank alcohol at home was not found to increase the odds of binge drinking

Source of alcohol—Students reported obtaining alcohol from a number of sources: friends (males 52.6%, females 62.4%), parents (males 50.2%, females 55.8%), got someone else to buy it for them (males 40.6%, females 46.6%), siblings (males 27.9%, females 33.6%), another adult they know (males 27.0%, females 28.0%), taken from home (males 15.9%, females 20.3%), stole it (males 8.4%, females 7.6%) or bought the alcohol themselves (males 17.9%, females 14.8%).

Senior students more frequently reported getting someone else to purchase alcohol for them (32.4%, 51.5%) or getting it from parents (45.5% juniors, 53.6% seniors) and junior students more frequently reported pinching alcohol (12.8% juniors, 4.6% seniors). Those students who bought their own alcohol were more likely to be senior students (6.0% 14 years and under, 23.9% 15-17 year olds). Only 14% (25/181) of those students who bought alcohol were aged 18 (legal age for purchasing alcohol) or older.

Multinomial regression models was used to investigate the association between students' major source of alcohol and the pattern of drinking whilst controlling for socioeconomic variables, age group and sex (Figure 1 and Table 4).

Students who bought their own alcohol were significantly more likely to be binge drinkers or heavy binge drinkers ($OR_{\text{binge}} = 1.7$, $OR_{\text{heavy binge}} = 2.8$). In addition, binge drinkers were significantly more likely to obtain alcohol from their friends ($OR_{\text{binge}} = 1.7$; $OR_{\text{heavy binge}} = 1.2$) and 'other adults' ($OR_{\text{binge}} = 1.6$, $OR_{\text{heavy binge}} = 1.7$). Furthermore, sourcing alcohol from parents was not associated with increased binge and heavy binge drinking.

Self-reported problems and harms associated with drinking alcohol in the last 12 months—The most commonly reported problems associated with alcohol use was: done things that could have got them into serious trouble (28.3%), being injured after drinking alcohol (27%), having unsafe sex (25.6%) and having their school or work affected by their alcohol use (14.1%). Other alcohol-related harms included injuring someone else after drinking alcohol (14.1%), requiring treatment for an injury by a doctor or a nurse, (4.9%) and involvement in a car crash (3.6%).

Figure 1. Associations between source of alcohol and pattern of drinking

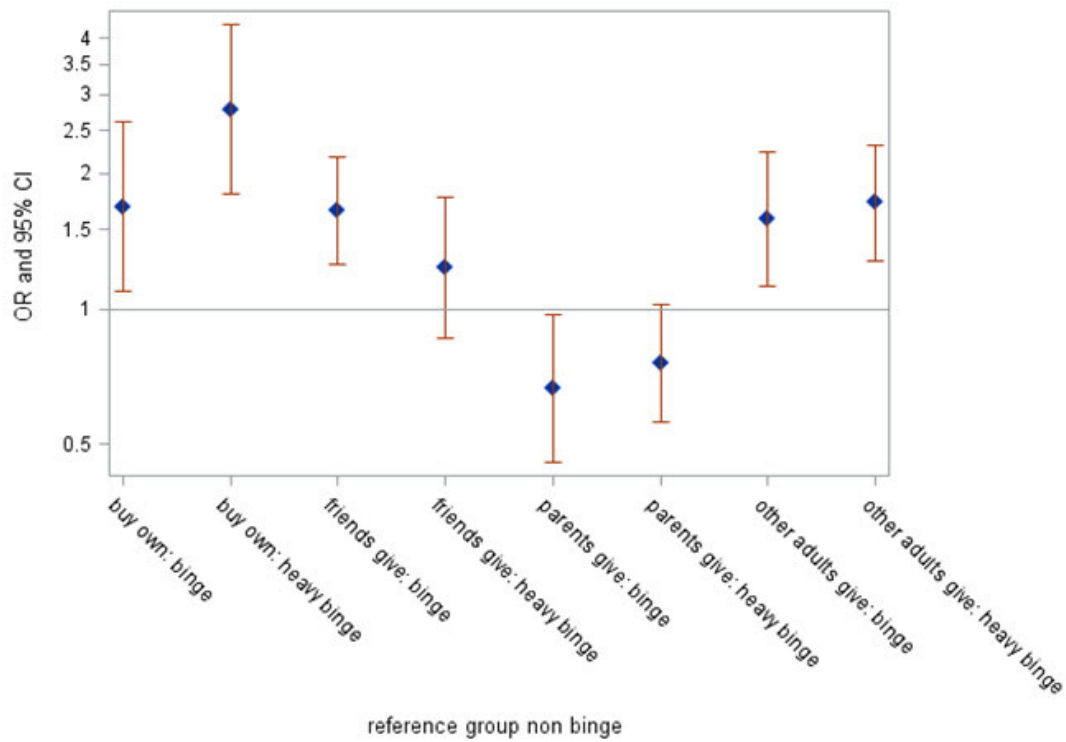
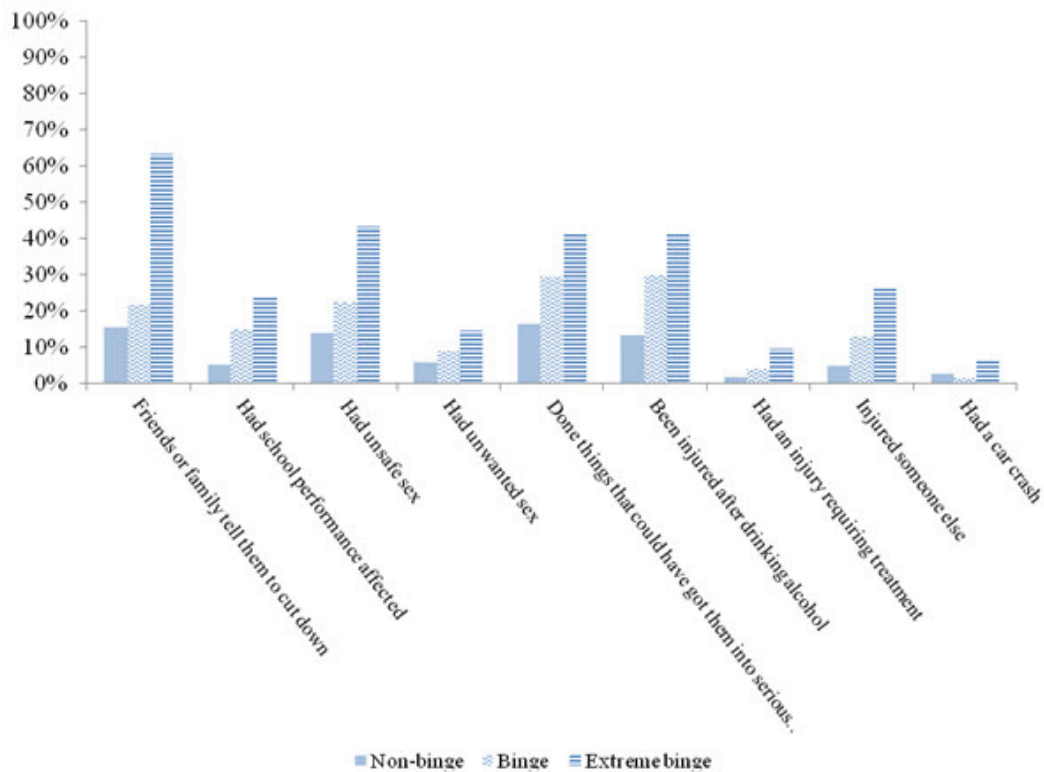


Figure 2. Problems associated with drinking by the pattern of drinking



Binge drinkers and heavy binge drinkers more frequently reported problems associated with alcohol use. Bivariate analyses showed strong associations between having problems with alcohol and pattern of drinking (Figure 2). Binge and heavy binge drinkers were significantly more likely to report that their parents and friends had told them to cut down their alcohol use ($\chi^2_{2df}=48.6$, $p<0.0001$), having their school work affected ($\chi^2_{2df}=54.1$, $p<0.0001$), having unsafe sex ($\chi^2_{2df}=92.5$, $p<0.0001$) or coercive or unwanted sex ($\chi^2_{2df}=15.2$, $p<0.0005$), done things that could have got them into serious trouble ($\chi^2_{2df}=65.5$, $p<0.0001$), being injured after drinking ($\chi^2_{2df}=69.7$, $p<0.0001$), requiring medical or nursing intervention after drinking ($\chi^2_{2df}=22.9$, $p<0.0001$), being injured or hurting someone else ($\chi^2_{2df}=75.0$, $p<0.0001$), or being involved in a car crash ($\chi^2_{2df}=12.6$, $p<0.0019$).

Accessing help and support for drug or alcohol use—Students were asked who they would go to for help if they had health problems or concerns about alcohol or drug use. Sixty-eight percent (68%) said they would seek help from friends, followed by parents (56%), school guidance counsellors (43%), family doctor (34%), school drug and alcohol service (31%), school nurse (21%), teachers (19%), and ‘other’ (21%). Nine percent said that they would not seek help from any person or service.

Six percent of all students (8 % of current drinkers) reported having difficulty accessing drug and alcohol services within the past year. Binge and heavy binge drinkers (after controlling for age, gender and socioeconomic factors) reported greater difficulty accessing drug and alcohol services within the past year (OR_{binge}=2.30, OR_{heavy binge}=4.97) (Table 4).

Twenty three percent of all students (25% of current drinkers) reported they had not been able to access a doctor, nurse or other healthcare worker in the last 12 months. After controlling for age, gender and socioeconomic factors in a multinomial regression model, there was no overall difference in the reporting of difficulty in accessing a healthcare worker in the past 12 months) (Table 4).

Discussion

The results of this large scale, representative national youth health survey of Māori students attending secondary schools throughout New Zealand provides evidence of high levels of binge and heavy binge drinking behaviours.

Several factors were found to be strongly associated with binge drinking, including the perception that ‘drinking alcohol is okay for people my age’, having friends who drink alcohol, obtaining alcohol from friends and ‘other adults’ and being able to purchase one’s own alcohol.

Our findings are consistent with previous literature suggesting that peers are a significant influence on drinking patterns in young people.²⁹ However, as Leung et al³⁰ note further longitudinal studies are required to disentangle whether young people start drinking after affiliating with alcohol-using peers, or intentionally seek out peers to drink with once they have initiated drinking. Despite the unknown direction of this pathway, efforts are needed to address the significant risks associated with the supply of alcohol from friends and other adults.

The recent retention of the legal purchase age of 18 years means that the new provision within the Alcohol Reform Bill to restrict the supply of alcohol to minors and increase the supervision of its consumption will become increasingly important as a mechanism to reduce the social supply of alcohol to young people, particularly Māori.

Contrary to several studies in other countries,³¹⁻³⁴ our study found that witnessing parents drinking alcohol in the home environment did not appear to increase the odds of Māori students' binge drinking behaviours. In addition, although over half of the Māori students in this study sourced alcohol from their parents, this was also not found to be associated with an increased risk for binge drinking. This is in contrast to previous New Zealand research that has found that parents who bought alcohol for their youth-aged children tended to supply excessive quantities and were often unaware of the impact of alcohol on young people.³⁵ Perhaps the presence of other protective factors (parental monitoring, relationships and communication)³⁶ may have reduced the influence of the parental factors in this study, or perhaps peers play a more significant role for young Māori.

Further research is required to explore this finding to inform both peer and parent-based programmes. The finding that over a quarter of binge drinking youth sourced alcohol from another adult they knew is intriguing but could not be characterised further in this study. In order to address this issue, future research needs to explore who these other adults may be, the contexts involved, and if parents are aware of these sources of access.

There is increasing evidence that there is no safe level of drinking for young people,³⁷ yet most young Māori aged 13-18 years in secondary schools are actively engaged in some form of alcohol use. Commercial access to alcohol was also strongly associated with heavy drinking patterns. Therefore, regulation of the sale and supply of alcohol must ensure it protects young people from harm and enables them to reach their full potential in adulthood.

A number of strong, evidence-based measures are available to reduce alcohol harm to young people.³⁸ The strongest levers in alcohol harm reduction for young people are price, legal purchase age, restrictions on marketing and advertising and zero tolerance for blood alcohol for young drivers.^{7,39-46} To be effective in reducing harm to young people, alcohol reform in New Zealand must include the evidence-based measures listed above, and be assessed for their impact on reducing inequalities.

Currently, there is scant literature on what works to reduce alcohol-related harm for young Māori drinkers. It is now almost ten years since Kypri⁷ noted in this journal the lack of evidence on policies and interventions that benefit Māori. This must be urgently addressed.

Students who reported binge and heavy binge drinking behaviours were more likely to report problems associated with alcohol use. This is consistent with extant literature on alcohol related harm.⁴⁷ Large proportions of Māori in our study reported problems and harms associated with binge drinking, yet also reported poor access to healthcare when required. Given that friends and family were the preferred source of help for drinking problems, it is important that effective strengths-based peer and whānau/family-based interventions for Māori youth are explored.

Evidence-based programmes that support families to improve communication and interactions regarding substance use⁴⁸⁻⁵³ and those that use culturally focused skills training⁴¹ should be examined closely to determine their appropriateness for Māori. Any such strategies need to be consistent with the Māori concepts of Whānaungatanga and manaakitanga or the valuing of inter-relationships and connections as these are central to Māori wellbeing.^{27,54,55}

Finally, we would recommend the complex social environments that influence Maori youth drinking, require strong public health strategies that are strengths-based and are grounded in a Māori positive youth development approach.^{27,56-58} These approaches^{27,28} focus on developing the capacities and strengths of young Māori, while surrounding them with positive opportunities for development and safe environments.⁵⁹⁻⁶²

A focus on individual responsibility and a deficits focus to alcohol harm reduction fails to consider that 'drinking alcohol is a social, as well as an individual act'⁵⁵ — particularly since New Zealand society often reinforces excessive alcohol use as a desirable social norm.^{27,56-58}

Limitations and further research

This study identified factors associated with binge related behaviours for Māori youth. As with any cross-sectional research no causal inferences can be made. This is a representative study, randomly sampling schools and students throughout New Zealand, however these data represent Māori youth who attend secondary school on the day of the survey, and therefore cannot be generalised to all Māori youth.

Those who do not attend secondary school (e.g. Alternative Education) or truant youth may be more vulnerable to alcohol related harm.⁶³ In addition, the role of other factors such as identity, cultural engagement and exposure to ethnic discrimination were not analysed in order to examine how they may moderate and mediate the observed associations.

Qualitative research is also required to further explore Māori young people's perceptions of binge drinking behaviours and factors that they perceive are helpful in assisting them to reduce excessive drinking behaviours. Finally, when considering major sources of alcohol, we are unsure who the 'other adults' might be and whether parents were aware that 'other adults' were a significant source of alcohol for their minors. Exploring the role of 'other adults' as a source of alcohol for young people alcohol is required.

Conclusions

Māori secondary students in New Zealand that observe their friends using alcohol, perceive alcohol is okay for people their age to use, are able to buy alcohol themselves or access it through friends or 'other adults' are vulnerable to excessive alcohol use and associated problems.

Strong legislative strategies are required to regulate the sale and supply of alcohol for youth to minimise their alcohol-related harms. In addition, social campaigns that address the 'drinking culture' in New Zealand are required to reduce the exposure and social acceptability of heavy binge drinking for Māori youth.

Māori youth reported poorer access to drug and alcohol services therefore we recommend that interventions and services are strengths-based, community focused, culturally relevant, actively engage families and schools to support equitable health outcomes for young Māori.

Competing interests: Nil.

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A new podiatry service for patients with arthritis

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Abstract

Aim The aims of this study were to identify the impact of a new podiatric rheumatology service on reducing foot pain, impairment and disability in patients with foot problems associated with rheumatic disease, and to report on patient satisfaction with the service.

Method A retrospective study of 245 patients with rheumatic disease at Counties Manukau DHB was conducted. Foot pain, impairment and disability were measured using a self-reporting patient outcome measure, the Foot Function Index. A range of podiatric interventions were reported. A self-administered, postal patient satisfaction questionnaire was sent to 148 patients.

Results Over two-thirds of patients were observed with hallux valgus (bunions). The results demonstrate a significant reduction in foot pain ($p < 0.001$) from initial visit to second visit (18% reduction in pain). A significant decrease in foot disability ($p = 0.04$) was found from initial visit to second visit. No significant differences were seen with foot impairment ($p = 0.78$). A variety of intervention measures were used with 24% of patients being prescribed foot orthoses and 28% of patients given footwear advice. The patient satisfaction survey found 84% of patients reported they were satisfied with the new service and 80% of patients reported that the service helped with their foot problems.

Conclusion The current service meets the needs of patients who suffer from rheumatological foot conditions such as rheumatoid arthritis and gout. The need for good foot education, provision of foot orthoses and advice on footwear are crucial to reduce the burden on patients with rheumatological foot conditions.

The role of the podiatrist in the rheumatology team is becoming recognised as a vital component in the integrated care given to patients by the multidisciplinary team.¹ Increasingly, consultants and their teams are requesting specialist foot care services and it is suggested that the podiatrist is a key practitioner in the management of patients with musculoskeletal disease.²

It has been recommended that patients should understand the role and have access to a podiatrist.³ Podiatrists have a prominent role to play in symptom relief and in improving the quality of life because, for patients with inflammatory arthritis, the involvement of the feet, even to a mild degree, is a significant marker for impaired mobility, functional incapacity and negative psychosocial impact.^{3,4}

Despite evidence for the need of podiatry services, podiatry is frequently an underused and under-resourced service and in many areas in New Zealand there is no specialist podiatry service.

In support of specialist foot care, a new podiatric rheumatology service was established following an evidence-based approach highlighting the need for improved access to podiatry care for rheumatology patients in New Zealand.^{1,2} In two subsequent publications, we have highlighted that patients with RA⁴ and gout^{5,6} have an increased need for a range of basic foot care services. Further, there is evidence that early intervention for existing or potential foot problems can improve long-term outcomes.⁷

The aims of this study were to identify the impact of a new podiatric rheumatology service on reducing foot pain, impairment and disability in patients with foot problems associated with rheumatic disease, and to report on patient satisfaction with the service.

Methodology

Study design—The retrospective study design was based on adult patients with a history of documented foot problems associated with rheumatic diseases. Patients were eligible if they had a referral made by rheumatologists, rheumatology nurse specialists and podiatrists within Middlemore Hospital and/or Manukau Super Clinic. Northern X Regional Ethics Committee approved the study [NTX/11/EXP/156].

All referrals were graded by one of the podiatrists (KE) and sent to the Rheumatology Scheduler for an appointment. Two experienced podiatrists were employed with a mean clinical experience of 15 years. Specialist training in dealing with the rheumatic foot was undertaken by the podiatrists.

Foot pain and disability—Disease impact was measured using the Foot Function Index (FFI).⁸ The FFI is a measure of foot pain and its impact on mobility and activity limitation. The FFI has been validated for patients with RA and used to evaluate the effectiveness of foot orthoses and footwear for those with rheumatoid arthritis.^{9,10} It is a self-administered questionnaire consisting of 23 items grouped in three domains: foot pain (nine items), disability (nine items) and functional limitation (five items). All items are rated using 100mm visual analogue scales, and higher scores indicate greater pain, disability and limitation of activity and thus poorer foot health.⁷

Podiatric intervention—A number of interventions were made available including advice relating to footwear and foot care, reduction of callus and corns, conservative treatment of nail conditions, debridement of gouty tophi, management of foot ulcers, prescription of foot orthoses and provision of exercise programmes. Foot orthoses covers a range of devices including: pre-fabricated, simple insoles and shoe inserts/modifications. Referral to other services was also reported.

Patient satisfaction—Furthermore, a self-administered postal survey was carried out to evaluate patient satisfaction of the current podiatric service. This covered the period from July 2010 to March 2012. Age, ethnicity, gender, disease duration and type of rheumatological condition were recorded. 148 questionnaires were sent out.

Data analysis—Data were analysed using SPSS v20.0 software. Gender, rheumatic disease and podiatric interventions were described as percentages. All other demographic characteristics were described as the mean (SD). Paired-tests were undertaken to look at significant differences of foot pain and disability between baseline measurements and follow-up visit.

Results

Participant demographics and disease characteristics

A total of 245 referred patients were assessed and treated over the 18 month time-period. The mean (SD) age of 55.7 (13.3) years old was recorded with 69% being women. 155 patients were managed for a further treatment, 76 for a third visit and a further 55 patients for subsequent visits.

The median duration of RA disease was 15 (IQR: 7.3–25) years which suggests a well-established disease with levels of functional disability. A range of rheumatic diseases were seen including: RA (52%), osteoarthritis (19%), gout (14%); psoriatic arthritis (8%); scleroderma (4%); ankylosing spondylitis (2%) and Sjögren’s syndrome (1%).

Foot pain and disability

Over two-thirds of patients were observed with hallux valgus (bunions). Over 90% of patients in the study presented with symptomatic callus under the plantar surface of the foot and/or on the toes. The results demonstrate (Table 1) a significant reduction in foot pain ($p<0.001$) from initial visit to second visit (18% reduction in pain). A significant decrease (Table 1) in foot disability ($p=0.04$) was found from initial visit to second visit (23% reduction in disability). No significant differences were seen with foot impairment ($p=0.78$) but we did find a 10% decrease in foot impairment.

Table 1. Foot pain and disability scores

Outcome	Mean (SD)
Foot Function Index [Pain]: Visit 1	29.7 (12.0)
Foot Function Index [Pain]: Visit 2	24.3 (12.7)
Foot Function Index [Disability]: Visit 1	41.2 (24.2)
Foot Function Index [Disability]: Visit 2	31.9 (26.3)
Foot Function Index [Impairment]: Visit 1	4.2 (5.5)
Foot Function Index [Impairment]: Visit 2	3.8 (5.8)

Podiatry interventions

A range of interventions were used including treatment of nail deformities, callus reduction, ulcer management (as well as debridement gouty tophi), clinical padding, foot orthoses, footwear advice, foot health education and provision of exercise programmes (Table 2). A further 69 patients were referred to other services.

Table 2. Podiatric interventions

Variable	Value
Conservative treatment of nail conditions, n (%)	23 (8%)
Callus reduction, n (%)	28 (10%)
Ulcer debridement, n (%)	12 (4%)
Clinical padding, n (%)	15 (5%)
Foot orthoses, n (%)	69 (24%)
Footwear advice, n (%)	82 (29%)
Foot health education, n (%)	13 (5%)
Exercise, n (%)	44 (15%)
Referral for bespoke footwear, n (%)	1 (0.3%)
Discharged, n (%)	4 (1%)

Foot orthoses—The range of foot orthoses included simple insoles, pre-fabricated contoured foot orthoses and modifications to foot orthoses.

Referrals—Referral to other health care professionals included: radiography, vascular consultant, orthopaedic, physiotherapy, district nurses, dietician and occupational therapy (Table 3). Further investigations included blood tests and referral back to the rheumatology nurse and rheumatologist.

Table 3. Referrals to other services

Variable	Value
Orthotic centre, n (%)	20 (29%)
Physiotherapy, n (%)	18 (26%)
Medical imaging, n (%)	7 (10%)
District nurse, n (%)	5 (7%)
Blood tests, n (%)	4 (6%)
Orthopaedics, n (%)	4 (6%)
GP, n (%)	4 (6%)
Rheumatologists, n (%)	3 (4%)
Occupational therapy, n (%)	2 (3%)
Dietician, n (%)	2 (3%)

Patient satisfaction survey of the podiatric rheumatology service—From 148 questionnaires sent out there were 62 responses (response rate: 42%). The mean (SD) age was 59.9 (11.5) years old and most respondents were female (86%). Over 74% of respondents were European, 13% Maori, 5% Pacific Islanders and 8% Asian. We found that the majority of patients reported that the podiatry service was very useful (63%) and helped with their foot problems (80%). Overall, the patients reported that they were satisfied with the podiatry service (84%).

Discussion

General findings and supporting literature—The purpose of this study was to undertake a clinical evaluation of the podiatric rheumatology services in Counties Manukau. Overall, this study demonstrates that, in this particular outpatient clinic, poor foot health and foot pain is highly prevalent in patients with rheumatic diseases. Most patients with RA had foot involvement ranging from callus, corns and lesser toe deformities.

Foot problems are known to occur in other rheumatic diseases such as gout.^{5,6} Patients with chronic gout suffer with high levels of foot pain associated with poor footwear, and find difficulties in purchasing adequate footwear.⁶ Our findings also demonstrated severe pain, impairment and limited activity suggesting most patients suffer with long-term disability from a range of chronic rheumatological conditions.

The current study found significant reductions in pain and impairment over 18 months. Furthermore, the results demonstrated significant differences from baseline to next visit with the podiatric interventions. Improved clinical results with podiatric intervention for patients with RA are clearly demonstrated. The data indicates that the

clinical needs of patients were addressed by the podiatric intervention of footwear advice, education, callus reduction and prescription of foot orthoses.

It is known from previous work that there is a link between levels of usage and patient satisfaction with footwear¹¹⁻¹⁵ and podiatric interventions, and therefore we can assume that the levels of podiatric interventions were associated with greater satisfaction.

The prescription of foot orthoses and footwear for patients with rheumatological foot conditions has been previously reported.^{14,15} We found that the majority of referrals were to the Orthotic Centre for prescribed therapeutic footwear.

The use of therapeutic footwear for reducing pain and improving mobility in those with established foot deformity has been documented.¹² Specialist prescription footwear should be available for patients who cannot fit into appropriate retail footwear and, in this area, podiatrists and orthotists should collaborate to achieve the optimal clinical outcome.

Those patients with severe symptoms and/or complications were referred back to the rheumatologist or other members of the MDT in order to protect them from the severe consequences of foot infection and ulceration.¹⁶ Advice was also obtained from the patient's rheumatologist on the management of infected ingrown nails or if there was a need for nail surgery; particularly if the patient's medical management was with biologic therapy.

Scalpel debridement was always carried out with caution, the pressure areas offloaded and, when necessary, a referral made to the orthopaedic team. Scalpel debridement is a quick and simple intervention to perform for painful plantar callosities in rheumatoid arthritis but is poorly researched.^{18,19}

Woodburn¹⁸ demonstrated that reduction of plantar callus with scalpel debridement in rheumatoid arthritis reduces forefoot pain (up to 48%) but for a very limited time (7 days). Reduction in callus resulted in an increase in forefoot pressures and suggests that reduction of callus over prominent metatarsal heads may lead to tissue damage. This would be of particular concern in patients with reduced tissue viability (long term steroid therapy, vasculitis, concurrent peripheral vascular disease) and/or neuropathy.¹⁷

While debridement alone particularly over prominent metatarsal heads or any other prominent joint can lead to tissue damage, many of the patients present with hemorrhagic callus over these joints, indicating incipient ulceration. We believe debridement alone is insufficient treatment. However, debridement, offloading and cushioned dressings can provide short-term relief of symptoms and prevent ulceration until the patient is seen by the orthopaedic team.

Williams and Bowden²⁰ reported that rheumatology teams and podiatry services should collaborate and aim to improve the foot health service to patients with disabling foot problems. The results from the current study indicate that for any future rheumatology service in New Zealand podiatrists should be part of the multidisciplinary rheumatology team and have received specialist training in this area to reduce the burden of rheumatic foot problems.

The results from the patient satisfaction survey showed that patients were highly satisfied with the care provided by the rheumatology specialist podiatrist. The quality of care from the patients' perspective is increasingly considered an important component of comprehensive chronic disease management.²¹

Patients who are satisfied with health care are more likely to be involved in medical decision making, to be compliant with treatment strategies and are less likely to experience adverse health outcomes.²² Previous studies have reported on patient satisfaction of a rheumatology service,^{23,24} but this is the first to be undertaken specially relating to a podiatric rheumatology service in New Zealand.

Future developments—Self-management programmes of foot problems have been advocated for patients with rheumatic diseases. A recent study undertaken in the UK using a self-management foot care programme for 30 patients with RA demonstrated that just over 50% of patients were physically able to undertake some aspects of self-managed foot care, including conservative management of nails, callus filing and daily hygiene and inspection.²⁵

We plan to provide patients with information and education using the recently reformatted Arthritis NZ 'Care of Feet'²⁶ pamphlet to enable patients to recognise the signs of foot problems and to understand what to do if variations occur.

Anecdotal evidence from this study suggests that the use of the structured, self-administered questionnaires were difficult to complete. This was due to a number of patients being unable to comprehend the wording of the questions or because English was not their first language.

We are currently piloting the use of face-scales as an alternative method. These are often used in paediatric services and research to evaluate foot pain, impairment and disability. We also found that a number of patients with chronic tophaceous gout and coexisting diabetes had previously been managed on a regular basis by the Podiatry Diabetic Foot Ulcer Team at the Manukau Super Clinic. However, several of these patients had to be discharged back into the community, missing out on regular podiatry care.

Over the past 18 months, some of these patients have been re-referred by the rheumatology podiatry service. Further, the characteristics of foot ulceration associated with chronic gout are an area for future study.

A multidisciplinary team (MDT) approach (incorporating various healthcare professions such as specialist nurses, physiotherapists, occupational therapists and podiatrists) is considered essential for the management of patients with rheumatoid arthritis.²⁷

Future developments should include further integration of specialist podiatrists into the MDT with the emphasis on minimising the effects of the disease and managing the patient's foot health needs. This integrated approach utilises the skills and knowledge of all MDT members, and fosters good interprofessional working practices, with the patient being the focus throughout the assessment and management of their needs.

Conclusion

The current work has highlighted that the burden of foot pain and disability in patients with rheumatic diseases can be reduced with a range of foot care interventions provided by specialist podiatrists.

A baseline foot examination at first presentation has identified patients with existing or imminent needs. Further, this foot examination provides a comparison for evaluating any changes either due to disease progression or the outcomes of foot health interventions.

Future developments may incorporate self-educational foot health programmes and collaborating formally with other health care professionals such as orthotists and orthopaedic surgeons, with the aim of improving even further the foot health outcomes.

Overall, this research has demonstrated that the current service has impacted on the foot health of these patients positively and that it meets the perceived need of the patients attending the service.

Competing interests: Nil.

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Accuracy of visual acuity testing in New Zealand primary health care

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Abstract

Aim To determine if visual acuity is tested reliably in primary health care in New Zealand.

Methods Fifteen to 26 'eyes' from seven participants were tested in the Eye Department of Dunedin Hospital under standardised conditions; and across 17 centres in nine general practices and the Emergency Department of Dunedin Hospital for comparison. Variables including lighting and distance were measured; chart type and centre conditions were recorded.

Results Eleven centres (65%) produced visual acuity scores that were inconsistent with the Eye Department, where 10 (59%) of them produced worse visual acuity scores and one centre (6%) produced better visual acuity score. Ten centres (59%) did not meet New Zealand Transport Agency standards of adequate illumination of greater than 500 lux. Ten centres (59%) failed to have their charts at the specified distance.

Conclusion There were inconsistencies in visual acuity testing in primary health care in Dunedin, New Zealand which may be related to the overall poor compliance with lighting and distance standards. These factors are potentially easily modifiable and their change should lead to improvements in visual acuity measurements.

The general practitioner (GP) or emergency doctor is often the first port of entry into the health system in countries with a primary healthcare model such as New Zealand. The Emergency Department of Dunedin Hospital (ED) was considered a primary healthcare facility for the purposes of this study.

It is estimated that 10% of all patients present to their GP with eye-related conditions,¹ and 2% of 'Accident and Emergency' admittance involves the eyes.² Furthermore, vision has medicolegal implications in areas such as suitability for surgery, for example in cataracts,^{3,4} and is frequently a reason for withdrawal of driving licences often decreasing independence and quality of life of the person concerned.

Visual acuity is also used as a marker of fitness by the Civil Aviation Authority, Maritime New Zealand, Police, Defence Force and Immigration New Zealand. A reliable and repeatable visual acuity score is increasingly important as vision declines with age and the proportion of older drivers increases.⁵

Visual acuity is a measure of the accuracy of form vision, the ability to discriminate spatially separated visual stimuli.³ Visual acuity measurement is a general screening tool taught in medical school and used by doctors and allied health professionals.¹

It can be affected by many variables, including examiner technique, patient variables or physical variables such as lighting, glare, chart type or distance from which the test is conducted.⁶⁻⁸ The International Organisation for Standardisation (ISO) provides standardised conditions for visual acuity testing,⁹ and the New Zealand Transport Agency (NZTA) provides recommendations for driver testing.¹⁰

Visual acuity measurement has been shown to be 99% reliable under standardised conditions,¹¹ and repeatable in a large eye clinic.¹² However, there are to our knowledge, very few studies that have investigated the reliability of visual acuity scores outside a controlled environment in a primary healthcare sector.^{7,8}

These studies have limited generalisability to a New Zealand primary healthcare setting as they differ temporally and geographically and are deficient in relating the effect of various variables to visual acuity scores. Hence, this study aimed to determine the accuracy of visual acuity testing in New Zealand primary health care.

Methods

Study design—This was a comparative study which used 5 patients from the Dunedin Hospital Eye Department between the ages of 67 and 88, the elderly participants; and two students from University of Otago aged 21, the young participants. Both eyes of all 7 participants were tested for visual acuity in the Eye Department of Dunedin Hospital, general practices and ED. Ethical approval was granted from the Lower South Regional Ethics Committee. Informed consent was obtained from all of the participants, general practices and ED.

Study sites—All general practices in Dunedin that were members of Southern Primary Health Organisation (SPHO) were contacted, apart from two which were excluded for not falling within the reasonable driving radius of 30 minutes from Dunedin Hospital. Twenty-eight general practices were contacted between 14 November 2011 and 13 December 2011. Eleven general practices showed interest and nine practices were able to participate within the given time frame of 6th and 20th December 2011. (Five practices were not followed up due to time constraints.) Along with ED there were 10 localities that participated. There were in total 17 visual acuity testing centres as some of the localities had multiple centres. Six general practices and ED had one centre, one general practice had two centres, one three centres and one five centres.

Study protocol—The five elderly participants had their right and left eyes tested with and without visual aids, equating to a total of 20 ‘eyes’. The two young participants had their right and left eyes tested: without visual aids, with visual aids plus cataract simulating goggles, and with visual aids; totalling 12 ‘eyes’. (The cataracts simulating goggles were made using sheets of lightly frosted plastic according to standard Eye Department methodology.)

Six of these ‘eyes’ were not tested as they were 6/60 (logMAR 1.0) or below in visual acuity score when tested in the Eye Department. This made a total of 26 ‘eyes’ available for testing. However, due to participant non-attendance, between 15 and 26 ‘eyes’ were tested in each centre, with 10 ‘eyes’ being tested in all centres. (Table 1).

Table 1. Number of ‘eyes’ tested in each centre

Centre No.	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
No. of ‘Eyes’	16	18	15	15	24	24	20	24	24	20	22	24	26	22	22	20	26

The visual acuity of the seven participants (all 26 ‘eyes’) was first tested in the Eye Department of Dunedin Hospital under standardised conditions (logMAR chart at 4 m with 1700 cd/m² luminance).

Participants were then tested in as many of the 17 study centres as their personal commitments allowed. The participant order of testing was randomised and the examiners followed a masked protocol, so they were not aware of the participants’ visual acuity score.

All participants were tested in order of best to worst combination of vision to minimise chart memorisation. Over the 17 centres and the Eye Department there were 12 different types of charts used, and one type of chart was repeated no more than three times.

Information bias was minimised by asking the examiner (doctor or nurse) to administer the test as they would for any other patient. Visual acuity scores were converted to logMAR units in order to achieve standardisation.¹³ (Snellen charts were approximated to logMAR units.)

Where the participant could not read the top line of the chart at the required distance, this was later converted to logMAR score of 1.0. The logMAR score was adjusted by 0.02 for each letter either read or missed on a particular line.¹²

Lighting and distance variables were measured; chart variables and centre conditions were recorded. Illumination (lux) of externally illuminated charts was measured using an illuminance meter (DSE Model Q-1400 Lux Meter) with the cosine corrected probe that was suspended directly over the 4/8 (6/12) portion of the chart (the critical value for passing class 1 or 6 driver licence as stated by NZTA)¹⁰, in contact with the chart and facing external light source(s).

Luminance of internally illuminated charts was measured using the same lux meter at the 4/8 (6/12) chart position, with the probe placed in contact with the chart but facing inwards. Lux readings were converted to absolute luminance (cd/m^2) values by using a cross calibration conversion factor, which was acquired by use of calibrated luminance probe (using precision light measurement equipment, RTI Electronics (Sweden) 'Piranha' system with light probes LUX 80LX-110404 and MON80MO-110404, calibrated at RTI (Sweden) June 2011), and the lux meter measuring luminance of a range of internally illuminated screens under laboratory conditions at Dunedin Hospital (Medical Physics).

Care was taken to avoid shadowing of the probe during the measurements. The source of light was noted, including the use of window light. The marked distance of the chart was recorded to the nearest 0.1 m using an 8 m tape, as well as the type of chart used (Snellen or logMAR) and the location of the centre.

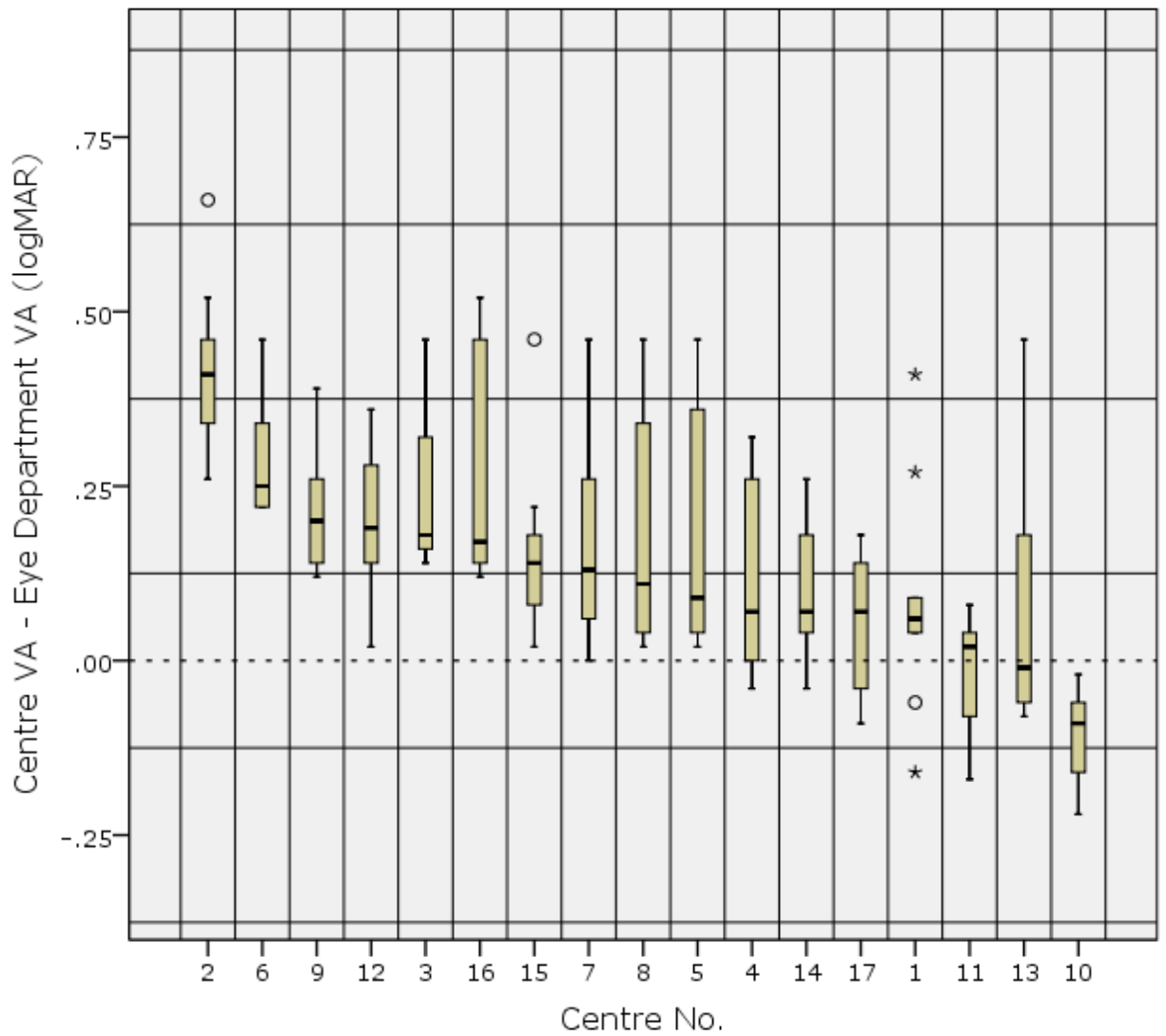
Box and Whisker plot (non-parametric descriptive graph) was used to find inconsistencies in visual acuity scores between the Eye Department and the centres. Statistical analysis was carried out using statistical software, IBM SPSS.

Results

Eleven of the 17 centres (65%) produced visual acuity scores that were inconsistent with the Eye Department. Visual acuity scores were determined to be inconsistent when the interquartile ranges of the centres did not overlap with the no difference value of 0.00 in analyses with 'eyes' attending all centres and all 'eyes'.

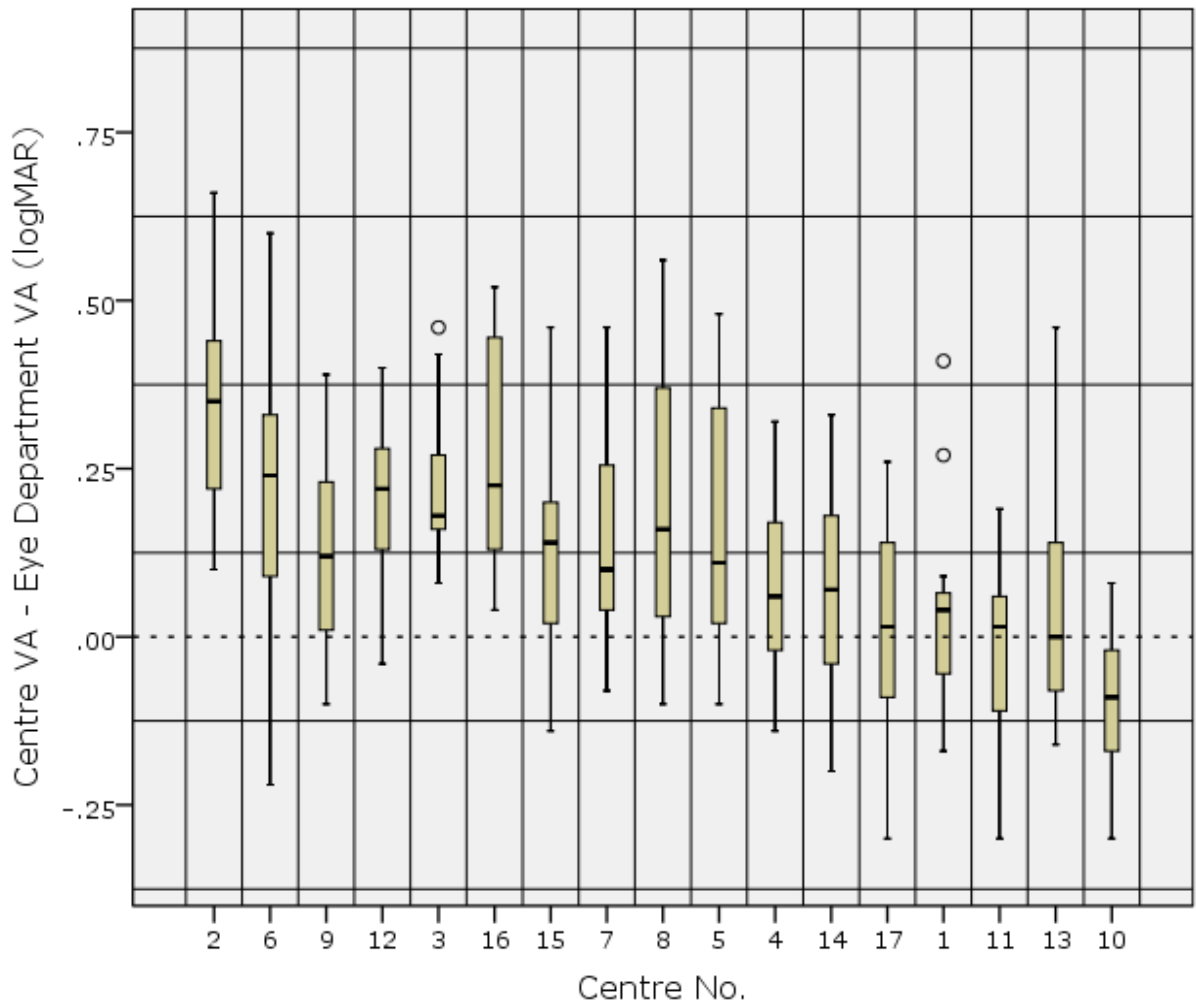
Ten of these 11 centres or 59% of the 17 centres produced worse visual acuity scores and one centre (10) or 6% of the 17 centres produced better scores than the Eye Department. (Figure 1a & 1b)

Figure 1a. Box and whisker plot for ‘eyes’ attending all centres



Footnote: VA = visual acuity. Centre VA of each ‘eye’ was subtracted by the Eye Department VA. There are 10 ‘eyes’ in each box and whisker. Outliers are shown as circle (1.5 × interquartile range) and asterisk (3.0 × interquartile range).

Figure 1b. Box and whisker plot for all ‘eyes’



Footnote: VA = visual acuity. Centre VA of each ‘eye’ was subtracted by the Eye Department VA. There are 15–26 ‘eyes’ in each box and whisker. Outliers are shown as circle ($1.5 \times$ interquartile range).

Visual acuity scores for centres 1 and 14 were inconsistent with the Eye Department’s when analysed for ‘eyes’ attending all centres, however, when all ‘eyes’ were included in the analysis, they were consistent. All other centres were uniformly consistent or inconsistent in both analyses.

Light intensity ranged from 160 to 2500 lux (mean of 630 lux) for externally illuminated charts and from 1800 to 4400 cd/m^2 (mean of 3100 cd/m^2) for internally illuminated charts. Ten of the 17 centres (59%) did not meet NZTA standards of greater than 500 lux illumination of the chart.¹⁰ Internally illuminated charts were 15 to 37 times of the recommended lighting of 500 lux equivalence. (This was determined by multiplying luminance scores by $\pi \times 1/\text{albedo}$, where the charts were assumed to have lambertian surface and the albedo was assumed to be 0.75.) Two of

17 centres had internally illuminated charts with luminance well in excess of the recommended 80 to 320 cd/m² by the ISO.⁹

The distance ratio (marked distance patient instructed to stand behind divided by specified distance on chart) ranged from 0.8 to 1.5 with a mean of 1.0, where 10 of the 17 centres (59%) had patient distances that were not accurately marked. The centre was considered to be accurately marked if the distance was within +/- 5% of the specified distance. (All centres used direct chart testing.) The Eye Department chart had a distance ratio of 1.0.

Tables 2a and 2b show light intensity and distance ratio respectively in ascending order. A pattern emerged with clustering of centres with worse visual acuity scores around low light intensity. Ordinary room ceiling light was the sole light source of most of these centres, where charts were often not uniformly lit. There was no obvious pattern with distance ratio or with centres stratified by chart type. (Table 2c) Nine of 17 centres (53%) did not draw their window blinds when conducting the test, providing a potential source of glare.

Furthermore, three centres tested visual acuity in a hallway with multiple doorways. Three of five nurses and two of 12 doctors indicated they had not received sufficient formal training in measuring visual acuity. Three of the 17 centres had 3 m charts. None of the participants complained of deteriorating vision or visual problems over the course of the study.

Tables 2a,b,c. Physical variables in centres

Table 2a. Lighting

Centre ^a		Light Intensity (lux)	Light Source
5	-	160	Room Light
7	-	160	Room Light
3	-	180	Room Light
12	-	180	Room Light
16	-	190	Room Light
9	-	220	Room Light
6	-	225	Room Light
2	-	300	Room Light
8	-	320	Room Light
14		450	Spot Light ^c
15	-	530	Spot Light ^c
4		880	Room Light
13		990	Room Light
10	+	1800 ^d	Back Light ^c
11		2200	Spot Light ^c
17		2500	Spot Light ^c
1		4400 ^d	Back Light ^c

Table 2b. Distance

Centre ^a		Distance Ratio ^b
10	+	.8
6	-	.9
13		.9
17		.9
12	-	.9
11		.9
8	-	1.0
1		1.0
5	-	1.0
7	-	1.0
9	-	1.0
14		1.0
4		1.0
15	-	1.1
16	-	1.1
3	-	1.3
2	-	1.5

Table 2c. Chart type

Centre ^a		Chart Type
1		Snellen
11		Snellen
12	-	Snellen
16	-	Snellen
17		Snellen
2	-	LogMAR
3	-	LogMAR
4		LogMAR
5	-	LogMAR
6	-	LogMAR
7	-	LogMAR
8	-	LogMAR
9	-	LogMAR
10	+	LogMAR
13		LogMAR
14		LogMAR
15	-	LogMAR

Footnote: VA = visual acuity.

2a. Centres with worse VA marked with -, better VA marked with + and no difference left blank.

2b. Distance Ratio = Marked distance / Distance specified on chart.

2c. Light source is in addition to ordinary room ceiling light. d. Luminance in cd/m².

Discussion

Visual acuity scores in 65% of the centres were inconsistent, with all but 'one' having worse mean visual acuity scores than the Eye Department. Light intensity and distance were highly variable. This is consistent with previous studies.^{7,8} Moreover 59% failed to meet NZTA's standards in light intensity,¹⁰ and 59% failed to have an accurately marked distance for the charts.

Centres were determined to produce inconsistent visual acuity scores with the Eye Department, only when this was shown in both figure 1a and figure 1b. This ensured a conservative method of analysis was used, where all 'eyes' were used in support of the 'eyes' that attended all centres.

One explanation for variation in light intensity is the absence of recommended lighting in the 'Royal New Zealand College of General Practitioners' (RNZCGP) standards.¹⁴

Visual acuity increases with respect to chart luminance, reaching a plateau over the range of 100 to 10,000 cd/m².¹⁵ The recommended value by the ISO and the light intensity of the internally illuminated charts, including the Eye Department, fall within this range. Externally illuminated charts are likely to have a similar relationship.

Space limitation of rooms, misreading recommended distances and inaccurate measurements offer explanations for failure to meet specified chart distances. Centre 10's better visual acuity scores than the Eye Department is probably due to the chart being placed too close to the participant.

Testing done at a distance other than what the chart is calibrated for will yield inaccurate visual acuity scores, since chart calibration specifies a set distance.³ (Centre 10 had similar lighting, glare and chart type to the Eye Department.) ISO recommends a minimum viewing distance of 4 m.⁹

Glare has a negligible effect on visual acuity for high contrast charts for patients with no lens opacities.¹⁶ High contrast charts are normally used for visual acuity measurement.^{4,6,17} However, glare can affect the contrast of optotypes (figures or letters) on the chart,⁸ in varying amounts affecting chart consistency.

Optotypes in the chart should be of uniform contrast throughout.⁹ Patients with cataracts are less tolerant to charts that do not meet the recommended high contrast ratio, where glare may significantly alter their visual acuity score.⁴ Therefore, glare should be minimised by taking adequate measures to have non-reflective charts and closing window blinds.

Although no obvious pattern existed with chart type in this study, logMAR charts have been shown to be more accurate than the traditional Snellen charts.^{13,17} Neuronal processing is a known component of visual acuity,⁶ and should be considered into testing where there could be distractions, such as in hallways of busy practices.

Furthermore, despite the small sample size, the proportion of medical professionals indicating not receiving sufficient formal training in measuring visual acuity was of particular concern, as 98% of general practitioners in New Zealand rate the ability of a graduating medical student to perform visual acuity measurements as being important.¹

The study had the following limitations:

- The small number of participants, multiple measurements from the same eye, coupled with the non-attendance in each centre meant that we were unable to generalise the results of the study to New Zealand, draw further conclusions

such as the reliability of result in each centre, nor directly relate visual acuity scores to variables.

- Potential selection bias as only 11 of 23 practices (48%) that were followed up were willing to participate.
- It is possible that examiners produced better visual acuity scores than otherwise because the test was in a study situation.
- The use of different examiners may have added to the inconsistencies, although this has shown to have minimal influence on previous studies using standard measurement procedures.^{11,12}

Based on these biases, the proportion of centres that produced worse visual acuity scores in comparison to the Eye Department may have been falsely lowered in this study. In spite of these limitations, however, it was considered that this study provided substantial evidence for a multi-region based study.

The proportion of the population over 65 is increasing in New Zealand; vision declines with age and loss of mobility and independence is a frequent cause for depression in the elderly.⁵

Inaccurate visual acuity measurement can result in poor grading of suitability for surgery such as cataracts. The influence of the primary or emergency doctor on driver licensing and vision screening is most important; and if doubts exist regarding a patient's vision, he or she is often referred to an optometrist or an ophthalmologist.

Therefore, it is important that visual acuity is measured accurately to prevent unnecessary referrals, to maintain independence in the elderly, for occupational eligibility and for immigration.

Conclusion:

As a preliminary study, we conclude that there are inconsistencies in visual acuity testing in primary health care in Dunedin, New Zealand; which may be related to variable lighting conditions and failure to conform to calibration distances as stated on the charts. These factors are potentially easily modifiable and their change should lead to improvements in visual acuity measurements.

Recommendations:

- Specified chart distance should be adhered to by providing a clear mark on the floor for patient positioning. Charts with a specified viewing distance of 4m or more are recommended.⁹
- An externally illuminated chart should be illuminated to at least 500 lux,¹⁰ while an internally illuminated chart should have a luminance between 80 cd/m² and 320 cd/m².⁹
- Blinds should be drawn on windows and non-reflective charts used to minimise glare.
- Internally illuminated charts are recommended as they provide the best source of lighting; alternatively a spotlight positioned appropriately and taking care to minimise glare can usually meet recommended lighting standards.

- LogMAR charts are recommended over Snellen charts.^{13,17} High contrast charts should be used,^{4,6,17} where optotypes do not differ noticeably in contrast and contour.⁹
- RNZCGP Cornerstone standards should specify lighting standards.

Competing interests: Nil.

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Inflammatory myopathies—a review of newly diagnosed patients (2004–2008) in the Counties Manukau region

Rajiv Gupta, Peter Gow

Abstract

Aims To estimate incidence, review clinical characteristics and management of newly diagnosed patients with idiopathic inflammatory myositis in Counties Manukau and to compare the findings with other reported series in New Zealand and overseas.

Methods A case note study of computer generated data of patients having a diagnosis of inflammatory myopathy from January 2004 to December 2008 were included in this retrospective review.

Results Twelve patients were newly diagnosed in the 5-year period. Polymyositis (PM) was diagnosed in 58%, dermatomyositis (DM) in 33% and inclusion body myositis (IBM) in 8%. Amyopathic dermatomyositis (ADM) and malignancy associated dermatomyositis were diagnosed in one patient each. There was slight preponderance of men in dermatomyositis and women in polymyositis. Muscle biopsy was performed in 75% and electromyography was reported in 58%. High-dose prednisone was administered to 83%, and 50% required other immunosuppressives, such as methotrexate (33%) and azathioprine (16%). Overall therapeutic response was good (75%) with 2 deaths (17%), one each in dermatomyositis and polymyositis. Regular follow-up was maintained in 92%. Nasopharyngeal carcinoma was diagnosed in a patient with polymyositis during follow up.

Conclusions Idiopathic inflammatory myositis is a challenging group of heterogeneous disorders. This study highlights the need to review current criteria to include subcategories, such as amyopathic dermatomyositis, and the importance of long term surveillance to detect occult malignancy.

Idiopathic inflammatory myositis (IIM) is a group of rare musculoskeletal disorders affecting fewer than 10 individuals per million per year.¹ They can be difficult to diagnose accurately due to variable presentation causing confusion with other muscle diseases such as viral myositis, toxic myositis and dystrophies.²

A recent international workshop on clinical trials in inflammatory myositis highlighted the deficiency of the currently accepted classification by Bohan and Peter.^{3–5} It may require inclusion of subcategories, such as amyopathic dermatomyositis and necrotising myositis and various other forms of myositis associated with newly identified myositis-specific and myositis-associated autoantibodies (anti-ARS, anti-SRP, anti-MI-2).⁶

Currently, the treatment of idiopathic inflammatory myositis is largely empirical as there are only few randomised prospective clinical trials to provide definite guidelines for their management.^{7,8} Corticosteroids remain the mainstay of treatment but

additional immunosuppressives and/or immunomodulators are often required to improve disease response.^{9,10}

We report the clinical characteristics and management of newly diagnosed patients with inflammatory myopathies in Counties Manukau, New Zealand, in a 5-year period between 2004 and 2008.

Methods

Computer generated data of both inpatients and outpatients having a diagnosis of inflammatory myopathy (according to ICD -10 codes) in a 5-year period were included in this retrospective audit. Patients who failed to fulfil diagnostic criteria of Peter and Bohan or had other documented cause of myositis, were excluded from the study. The clinical notes were individually scrutinised for various parameters, including the demographics of the patients, clinical presentation, laboratory investigations, diagnostic procedures and the assessment of management. The latter included commencement of drug therapy, clinical and/or biochemical response, duration of follow up, complications during therapy and prognosis. Due to their unavailability, myositis specific antibodies and myositis-associated antibodies were not done.

Results

Twelve patients out of 175 screened were diagnosed with idiopathic inflammatory myopathies in the 5-year period (PM 7, DM 4 and one IBM). The mean age of 62 years in DM was higher in comparison to 49 years in PM, with slight female preponderance in PM (male: female 3:4) and significantly more men being affected with DM (male:female 3:1). Only one woman, aged 66 years, was diagnosed with IBM in this period. Caucasians constituted less than half of this cohort (5), the rest being from other ethnicities (Table1).

Table1. Age, sex and ethnicity

Demographics	PM	DM	IBM	Total	%
Mean age	7(58.3)	4(33.3)	1(8.3)	12	
(Range)	49	62	66		
Gender					
Male	3	3	0	6	50
Female	4	1	1	6	50
Ethnicity					
Caucasians	3	1	1	5	41
Pacific	2	0	0	2	17
Chinese	1	1	0	2	17
Others	1	2	0	3	25

The onset of symptoms before presentation varied from one month to 12 months in both PM and DM, with an average of 4.4 months for each. IBM was diagnosed 18 months after the appearance of symptoms in the single patient with this condition. Most (10) had gradual onset of the disease, with one patient having acute onset (≤ 4 weeks), and another having an episodic presentation.

Most of the patients had characteristic clinical and biochemical features, prompting a diagnosis of inflammatory myositis, which included dermatomyositis, polymyositis and inclusion body myositis (Table 2).

In the dermatomyositis group, one patient had possible (amyopathic) DM with skin rash, elevated creatinine kinase but without muscle weakness, one associated with colonic malignancy, and one each had a diagnosis of probable and definite dermatomyositis. In the PM group, pulmonary fibrosis and nasopharyngeal carcinoma were noted to develop during the follow up period.

Table 2. Clinical features at presentation.

Clinical presentation	PM	DM	IBM	Total	%
Myalgia	7	3	0	10	83
Weakness	7	3	1	11	92
Skin involvement	0	4	0	4	100
Dysphagia	1	1	0	2	17
Medical associations	1 (statin) 1 (HF/AF)	1 colon cancer			

PM=Polymyositis; DM=Dermatomyositis, IBM=Inclusion Body Myositis, HF/AF=Heart failure/Atrial fibrillation.

Inflammatory markers were elevated in 6 patients, with more frequent elevation in ESR (6) than CRP (3). Creatine kinase (CK) was elevated in 11 out of 12, with variable rise. CK was normal in the single patient with inclusion body myositis. Anti nuclear antibodies (ANA and ENA) were positive in three patient, two with DM and one with PM. The latter patient also had anti Jo-1 antibodies, elevated anti RO52 (122) and pulmonary fibrosis. MRI revealed myositis in one patient. EMG and muscle biopsies were positive in 7 and 9 of the 12 patients, respectively (Table 3).

Prednisone was used in 10 out of 12 patients. Immunosuppressives were used as steroid- sparing agents in 6 patients, methotrexate being the most common, followed by azathioprine (Table 4). Pulse therapy, in addition to oral prednisone, was administered in only one patient with polymyositis. No patient was treated with cyclophosphamide, intravenous immunoglobulins (IVIG) or cyclosporine (Table 4).

Table 3. Summary of investigations

Investigations		PM (7)	DM(4)	IBM (1)	Total (%)
ESR	↑	3	3		6 (50)
CRP	↑	2	1		3 (25)
CK	↑	7	3	1	11 (92)
LDH	↑	2	0	0	2 (17)
ANA	↑	1	2		3 (25)
	Normal	5	1		6 (50)
ENA	Positive	1	2		3 (25)
Anti Jo	↑	1	0		1 (8)
Muscle specific antibodies		0	0	0	
MRI		1	0	0	1 (8)
EMG	Abnormal	6	1	0	7 (58)
	Not done	1	3	1	4 (42)
Muscle biopsy	Abnormal	7	1	1	9 (75)
	Not done	0	3		
Skin biopsy		0	2	0	2 (17)

↑- increased, Positive /Abnormal – consistent with inflammatory myopathy.

ESR=erythrocyte sedimentation rate, CRP=C reactive protein, CK=creatinine kinase, LDH=lactate dehydrogenase, ANA=antinuclear antibodies, ENA=extranuclear antibodies. MRI=magnetic resonance imaging, EMG=electromyography.

Table 4. Therapy and outcomes

Management	PM (7)	DM(4)	IBM (1)	Total (%)
Prednisone	6	3	1	10 (83)
Immunosuppressives	4	2	0	6 (50)
	MTX-2 AZA-2	MTX-2, HCQ-1		
Follow up (m)	7	3	1	11 (92)
Range	1 to 54	1 to 42	60	
Mean	27.3	17.2	60	25.3
Disease complications	Pulmonary fibrosis-1			1 (8)
Drug-induced complications	4 (steroid)	1 TB & death		5 (41)
Response	6	3	0	9 (75)
Refractory	1		1	2 (17)
Outcome				
Good	4	2		6 (50)
Death	1	1		2 (17)
Cancer (nasopharyngeal)	1			1 (8)
Progressive			1	1 (8)

The therapeutic response, based on improvement in the clinical manifestations (primarily proximal muscle weakness), biochemical parameters and functions, was good in the majority of the patients (71% in PM, and 66% in DM), with concurrent physiotherapy and regular follow up. Regular follow up was maintained throughout

this period, apart from two patients who died and one lost to follow up, contributing to good outcomes.

One patient with polymyositis who had refractory disease subsequently developed nasopharyngeal carcinoma 5 years after the diagnosis. Two patients with PM died in the study period, including one within 3 months of diagnosis due to tuberculosis. One patient had disease-related complications (pulmonary fibrosis) and five had treatment-related complications with steroid adverse effects in four patients and one who died secondary to widespread tuberculosis (Table 4).

Discussion

The reported overall incidence of IIM ranges from 2-10 new cases per million persons per year in various populations.¹¹ Our study showed an incidence of 5.1 per million per year (based on an estimated population of 464000 in 2007). This was comparable with other reported series, both regionally and internationally.

Twenty-nine patients with polymyositis and dermatomyositis were seen at the three main hospitals in Auckland between 1967 and 1977,¹² with a similar incidence in the North Canterbury over a ten year period and recently in Middle Eastern region over a 14 year interval.^{13,14}

The average number of presentation per year were slightly less in Counties Manukau as compared to North Canterbury (2.4 vs 3.75). Estimates of incidence and prevalence are hampered by the varying diagnostic and classification criteria, and selection bias.¹⁵

The higher frequency of DM in men in our series, was different from that reported in literature, with DM more common in women.^{11,15} The mean age for PM patients was lower than those with DM, as compared to the patients in North Canterbury.¹³

All patients with polymyositis fulfilled the criteria of Bohan and Peter. Among the patients who did not meet the entry criteria for the study, there was one patient each with a diagnosis of 'amyopathic' dermatomyositis, 'possible', and 'probable' dermatomyositis. This highlights the need for having an internationally accepted and revised classification of IIM.^{15,16}

These issues are currently under intensive debate and efforts are being taken by various expert groups (the European Neuromuscular Centre and The International Myositis Assessment and Clinical Studies Group) to establish a uniformly accepted classification and diagnostic criteria.³ Moreover, the classification by Bohan and Peter suggested more than 3 decades ago needs updating as it does not include subcategories, like clinically amyopathic dermatomyositis, inclusion body myositis and autoimmune necrotising myopathy.

Anti-Jo antibodies are reported to be present in 15–20% of the cases, but, in this cohort, it was positive in only one of the patients who had pulmonary fibrosis (8%). This patient also had elevated anti-RO52, a myositis associated antibody reported to be present in 37 % of myositis which correlate well with anti ARS, predicting the course of interstitial lung disease.⁶ Moreover, these and other newly identified autoantibodies in IIM have been shown to be useful in making an accurate diagnosis and choice of therapy.

Myositis is well known to be associated with malignancy, especially dermatomyositis, though its association is less clear with PM.¹⁷ In our series, dermatomyositis preceded the diagnosis of colon cancer in one patient, whereas nasopharyngeal carcinoma developed after 5 years in a patient with polymyositis. This highlights the importance of ongoing surveillance and regular follow up in such patients. Recent research emphasize the importance of measuring specific cancer associated myositis autoantibodies (anti p155), which has a high negative predictive value, especially in dermatomyositis.¹⁸

The clinical presentation, laboratory findings and management (table 3 and 4) are concordant with other studies except for the fewer patients with documented EMG (58%) and muscle biopsy (75%) in this cohort.¹¹ Therapy and outcomes of management were comparable with other studies, with 50% showing good response to therapy and 17 % (one patient each with polymyositis and inclusion body myositis) who had refractory disease.^{14,19} Seventeen percent mortality is relatively low in comparison to other reports.^{13,14}

The low mortality could be explained by short follow up period (mean 25.3 months), early diagnosis and aggressive therapy, few patients with malignancy associated myositis, and better management of disease/drug- related complications. None of our patients required other drug therapies (IVIg, cyclophosphamide, cyclosporine or Rituximab), which are primarily indicated for severe and refractory disease.⁷

In summary, our study data is consistent with other reported series. Idiopathic inflammatory myositis is a group of uncommon heterogenous disorders, in which a review of the current criteria is required for inclusion of other subcategories, such as amyopathic dermatomyositis. Long term follow up and regular surveillance are prudent to detect occult malignancy, particularly in patients with amyopathic dermatomyositis or dermatomyositis.

Competing interests: Nil.

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Testing times: do new prenatal tests signal the end of Down syndrome?

Robert Cole, Gareth Jones

Abstract

Since 2010, prenatal screening for Down syndrome (DS) has been offered to all pregnant women in New Zealand. The programme has been criticised by several groups, on claims that screening is eugenic and discriminatory towards those with DS. Recently, tests have been developed that may one day prove more efficient than current screening methods. They are an example of 'Non-Invasive Prenatal Diagnosis' (NIPD), which enables diagnosis earlier in pregnancy with less risk of complications. If the current programme raises objections, what threats does this new and seemingly more attractive technology pose to the DS community?

We argue that NIPD is simply an extension of current screening methods, raising similar ethical concerns. Presently, the programme shows little evidence of 'eugenics', demonstrated by moderate uptake rates and varying attitudes towards disability. We do not regard the offer of screening to be threatening, as women choose whether or not to be screened depending on their own personal circumstances. One day, prenatal testing may result in fewer people with DS; but past and present trends indicate these individuals will continue to be supported, irrespective of 'group size'. Care and respect for the disabled will remain essential, regardless of a woman's decision over her pregnancy.

In October 2011 a new prenatal test for Down syndrome (DS) was unveiled in the United States.¹ The test analyses proportions of fetal DNA in maternal plasma to give a risk estimate of DS in the fetus. This is an example of 'Non-Invasive Prenatal Diagnosis' (NIPD), which can also detect Trisomies 13 and 18.² Though not specific enough at present, with improvement NIPD could make current prenatal screening and diagnostic methods for DS unnecessary. However, groups such as 'savingdowns.com' believe that New Zealand's current screening process is 'eugenic', discriminating against those with DS by 'the prevention of their births'.³

Further concerns are raised over these new tests, since NIPD has the potential to be used in more pregnancies than with existing screening. Therefore it is timely to ask: what would NIPD mean for the DS community? Furthermore, what does testing, both now and in the future, say about our attitudes towards DS, and disability as a whole?

Since 1968 women have been selectively offered prenatal diagnosis for DS on the basis of advanced maternal age.⁴ This 'opportunistic' method of screening was declared 'unsafe and should not continue', as unnecessary numbers of invasive diagnostic procedures were harming some pregnancies.⁵

In 2010 'quality improvements' were introduced which resulted in New Zealand's current screening programme, which tests for DS and other congenital conditions. The method of screening differs, depending on gestational age.

The majority of screening tests are taken before 14 weeks of pregnancy, via the 'First Trimester Combined Screening' pathway. Here, information on the mother (such as maternal age and smoking status) is combined with a maternal blood test of two serum markers and the results of a Nuchal Translucency (NT) test on ultrasound.

After 14 weeks' gestation 'Second Trimester Maternal Serum Screening' is offered, which utilises information on the mother and the results of four serum markers, without a NT scan. Both of these screening pathways give a numerical risk estimate of congenital malformation in the fetus, which is then conveyed to the patient as either 'low' or 'increased' risk. Throughout the process women are to be reminded that the screening is voluntary, that they can opt out at any time, and that partners and family can be involved in decision-making.⁶

Currently, just over half of all pregnancies receive DS screening, though it is offered to all pregnant women.⁷ Thus there is considerable room for uptake to increase, which may well result from the use of a quick, safe test such as NIPD. The 'quality improvements' of 2010 were developed to provide equality of access and safety for mother and fetus, and NIPD could prove to further fulfil these aims.

Non-invasive prenatal diagnosis

In the case of an 'increased risk' result, two diagnostic techniques can be used to confirm DS (and other genetic abnormalities) in the developing fetus. Chorionic villus sampling (CVS) is used earlier than 14 weeks of gestation, and amniocentesis is used after this time. Both procedures carry with them a spontaneous abortion risk of around 1%.⁴

With current screening most pregnancies subjected to CVS or amniocentesis do not actually have a DS fetus,⁸ and as a result, fetuses are lost as a consequence of these diagnostic procedures. The primary advantage of NIPD is that there is no risk of spontaneous abortion, because diagnosis is based on only a blood sample.

Currently, NIPD has the potential to be used from 10 weeks of pregnancy,² similar to CVS and earlier than amniocentesis.⁹ In the future, NIPD could be used from seven to nine weeks, as fetal DNA is found in maternal blood at a very early gestational age.¹⁰ Results could be obtained more quickly, as current invasive procedures have turnaround times of 1 to 3 weeks.⁶

At present diagnostic results are rarely received before 12 weeks, and often after 17 weeks of gestation,¹¹ leading to late terminations which can be traumatic and (at times) dangerous. An earlier diagnosis would allow women more time for decision-making, and the option of an earlier, safer termination with less emotional and mental repercussions.

It has been reported that women express interest in NIPD, primarily due to the absence of risk of spontaneous abortion.¹² Some women find diagnosis helpful to prepare for the birth of a child with DS,¹³ and NIPD would be preferable to amniocentesis/CVS as there are no major complications.

NIPD may prove more attractive for district health boards, by reducing the number of costly invasive procedures at specialist care centres. For these reasons, NIPD is likely to be used in more pregnancies than current diagnostic procedures at some stage in the

future.¹⁴ Should costs drop and clinical efficacy be proven, NIPD could eventually make current screening methods redundant as well.

But what would NIPD mean for the DS community? Increased uptake of tests will result in increased detection of DS, and probably more terminations. The number of DS births may, as a result, drop. However, it is unlikely that DS will disappear. Abnormalities escape detection using even the most rigorous diagnostic techniques, and there will always be women who do not wish to undergo testing.

But as more pregnancies are tested, will DS become a 'rare' disorder? In time, perhaps. However, the life expectancy of those with DS is increasing, and is likely to soon approximate that of the non-DS population.¹⁵ This will mask, at least temporarily, any effect of NIPD on the prevalence of DS. Hence, even with a rapid increase in the uptake of NIPD, it is unlikely that the numbers of those with DS will change markedly in the near future.

NIPD and current DS screening tests both provide comparable information, and enable similar choices for pregnant women. Because of this, the ethical issues likely to be raised by NIPD will be analogous to those associated with current screening. 'Savingdowns.com', an anti-screening group, argues that a nationwide DS screening programme is simply a money saving exercise, initiated by a government which views individuals with DS as nothing more than a drain on society.³

'Savingdowns.com' claims that the current screening costs '\$75,000 per [terminated] child with Down syndrome'. However, such wording misrepresents what screening provides to the majority of women; namely, reassurance in the case of a 'low risk' result. To evaluate DS screening on the basis of cost-effectiveness is to compare DS to other screened, treatable diseases, such as breast and cervical cancer.

In these cases, a 'cure' is the overall aim. There is no 'cure' for a DS pregnancy, indeed this is not the aim of the test. The test's purpose is to give women information on the pregnancy, not prevent a DS birth. The value of this knowledge to women is impossible to quantify, because the choices it makes possible would otherwise be unavailable. For these reasons cost-effectiveness should not be used to assess DS screening,⁵ despite claims that it is the overall aim.¹⁶

Is screening for Down syndrome 'eugenic'?

Anti-screening groups frequently label the current screening 'eugenic' in nature, making distinct comparisons to the killing of the disabled in Nazi Germany in the 1930s–40s.

This clouds the debate around the ethics involved, since current screening is voluntary, not state-enforced. Patients are given a choice whether or not to be screened, and how to respond to the resulting information.

DS screening does not serve to systematically erase the congenitally disabled from the population; it provides information for patients about their pregnancy.

This is not the start of a 'slippery slope' to Third Reich genocidal acts; as shown below, support and advocacy for the disabled has never been greater. Nevertheless, it is important to ensure that women are never coerced into accepting screening or subsequent termination, and a decision is made which is appropriate for them.

If all pregnant women were persuaded to be screened and unable to make voluntary decisions then eugenic overtones would indeed be present. Such persuasion does not need to be administered by the state; if severe pressure was exerted by health practitioners a form of 'institutionalised eugenics' could still eventuate. Such a practice would require all women, or a large proportion of them, to be coerced in the same way. However, with only 55% of pregnancies utilising DS screening,⁷ this does not appear to be the case.

Thus to argue that the screening programme is 'eugenic' seems inaccurate, as nearly half of all pregnant women are declining the offer of screening. This points to the success of fair, supported choices free from state or medical coercion.

Accusations of 'institutionalised eugenics' are better directed at termination of pregnancy, as up to 90% of women who receive a positive result from CVS/amniocentesis proceed with termination.¹⁷ This number seems high until placed in context: those unlikely to consider termination on the grounds of DS often decline diagnostic testing.

If there remains a suspicion of 'institutionalised eugenics', it is unlikely that any health workforce could unilaterally enforce such a decision on an entire population, for several reasons. Those in the health workforce are far from homogeneous, and have varying perceptions of disability. No longer do patients leave their choices purely in the hands of doctors, while a patient's right to refuse medical treatment is well-recognised and enshrined in law.

Lastly, non-directive counselling has been shown to be beneficial in allowing women to make fair, independent decisions for screening decisions,^{18,19} and is offered to women both before and after diagnostic testing.⁶

There is evidence that a minority of practitioners may attempt to pressure women towards termination of a DS fetus; however, a similar number urges continuation of an affected pregnancy.²⁰

Hence, to argue that the current programme is eugenic is an over-generalisation, even though some health professionals may be unduly persuasive in offering termination. This fault is not implicit within the screening process per se; it points to a flaw in the education of health professionals, where a proportion are inadequately informed about the quality of life of those with DS.¹¹

While DS results in varying levels of intellectual disability, those affected report a consistently high satisfaction with their lives. The vast majority of people with DS feel that they are capable and have self worth, and love their families and friends.²¹

Children with DS are frequently described as being more content, caring and loving than non-DS children. It is incumbent upon obstetricians, GPs and midwives to ensure that information such as this is conveyed to women involved in the screening pathway. With tests such as NIPD likely to be used earlier and more frequently in pregnancy, this becomes increasingly important.

Supporting individuals with Down syndrome

But does the mere offering of a test not subtly imply that DS is undesirable, a 'disease' best avoided? We argue that this is not necessarily the case, although we

recognise that children with DS require more care and support than non-DS children.²² This support is often required throughout life as in most cases a person with DS cannot live fully independently.²³

For some families, raising a child with DS will be immensely difficult, and it is for this reason that we allow the option of termination. This is similar to the option of termination for other serious congenital disabilities, a situation that has prevailed since the 1978 amendment of the Crimes Act in New Zealand.

Society offers a choice, not a routine procedure; it is the woman, not the state, who makes this judgement. The assumption is that women make this choice in regard to their own life circumstances, and not merely because a screening process is offered within the first 20 weeks of pregnancy.

As a consequence of the availability of NIPD and any further tests that may be developed in the future, the numbers of those with DS may fall. However, there is no indication that society will cease to value these groups, even though they number less than in the past.

Disabled persons gained recognition and respect throughout the 20th century, regardless of their group size. Awareness and services for the disabled have grown dramatically²⁴ and funding is set to increase for the near future.²⁵

Some argue that a reduction in number of those with a disability like DS will reduce the standing, recognition and support of such individuals in society.²⁶ This is unsubstantiated, since there is little evidence that society neglects to treat rare disorders because there are few with the condition. For instance, we do not value and support those with DS more than those with Fragile X syndrome, on the grounds that DS is more prevalent. There is no evidence that our care for those with spina bifida is inferior to that of 10 years ago because the incidence of spina bifida has decreased.^{27,28}

We value and treat individuals as persons, supporting them in regards to their needs, not the number who shares their disorder. Along similar lines we will continue to support those with DS, and this is in no way jeopardised by women's decisions for their own pregnancy.

We concurrently offer prenatal screening and value the disabled by upholding several values in society.

First, we value an ethic that stresses the importance of 'doing the most good'. On these grounds we accept that in some cases, the perceived disadvantages resulting from a DS pregnancy (to child and family) may outweigh the perceived good from the child's life.

Second, we value reproductive liberty, the ability to make individual decisions over one's pregnancy. Others, such as the state, are limited in their control of this right. Alongside these we uphold dignity, respect and justice, realising that those who are disabled demand equal respect as citizens, thereby deserving support from society.²⁹

Inevitably, these values must be held in some tension; but as long as they are recognised as important, we will make sure one (e.g. reproductive liberty) never fully undermines another (e.g. respect for the disabled). From this, we can argue for two

compatible viewpoints - that screening is justified, and that the disabled will continue to receive support and respect from society.

The advent of less invasive tests such as NIPD places increasing demands upon our ethical awareness. While NIPD does not automatically lessen the value society places on disabilities such as DS, technological efficiency must never be our sole consideration in the use of such tests. It must be balanced by serious regard for continued, and, if necessary, increasing support for children and adults with these conditions.

It should be noted that NIPD is still in its infancy, with technological advances permitting detection of other conditions, reducing cost, and improving specificity all required before NIPD is likely to be offered as standard care.³⁰

Regardless, should NIPD or tests like it one day replace current screening methods, unwavering advocacy for those with disability will remain of paramount importance.

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Tutu toxicity: three case reports of *Coriaria arborea* ingestion, review of literature and recommendations for management

Sally F Belcher, Tom R Morton

Abstract

We describe three cases of tutu berry (*Coriaria arborea*) ingestion resulting in tonic-clonic seizures in two individuals and mild symptoms in the third. Tutu poisoning in humans appears to be a rare occurrence; the last reported case in the medical literature being over 40 years ago.

We review the literature on tutu poisoning and recommend extending the period of observation for poisoned individuals from 8 hours to 12 hours or longer. We also recommend that prophylactic benzodiazepine use should be considered in those with mild to moderate symptoms of poisoning.

Background

There are about 30 species of Coriariaceae found around the world including southern Europe, eastern Asia, south and central America, and New Zealand.¹ The six species native to New Zealand and the Chatham islands (*Coriaria angustissima*, *C. arborea*, *C. lurida*, *C. plumosa*, *C. pteridoides* and *C. sarmentosa*) are all known by the name tutu¹ and are mostly deciduous shrubs found in grassland. There is variety in appearance and distribution as seen with *Coriaria arborea*, or “tree tutu”, which may become an evergreen tree growing to 6 metres in height and being found in coastal and montaine forest.²

The primary toxin, tutin, was discovered in 1870³ and is found in all varieties of tutu^{1,2}. It is a picrotoxin-like toxin which acts as an antagonist at amino acid receptors within the CNS, especially the medullary, cortical, respiratory, vasomotor, and autonomic centers⁴. In cultured neurons tutin causes significant suppression of GABA type A receptors⁵, resulting clinically in anxiety and seizures.

Tutu has gained notoriety for its poisonous nature and is responsible for the greatest percentage of stock poisoning by plants in New Zealand.¹ Indeed the first two sheep brought to New Zealand by Captain James Cook in 1773 both died after eating the plant, just a few days after their release in Queen Charlotte Sound.^{3,6}

A number of settlers, particularly children who were fond of the palatable berries, also died^{2,7}. Fatal poisoning has also been reported in elephants, including one from a travelling circus who ate roadside tutu shrubs in 1957.^{2,3,7,8}

The whole plant is toxic except for the soft, black or purple fleshy petals (known as berries).² Maori were certainly aware that the berries contained highly toxic seeds and that careful separation was needed to avoid poisoning.² The strained juice though was

valued and used for medicinal purposes, to flavour bland foods, and to brew a sweet wine.^{1,2}

Alfred Saunders, the first settler to step ashore in Nelson from the *Fifeshire* in 1842, described his initial encounter with the local Maori:

“But we soon gave the Maoris another and more real cause for uneasiness by our eagerness to taste their nice-looking tutu berries. They knocked them out of our hands as we lifted them to our lips. They took a handful of the seeds, and turned up their eyes with an expression of horror. They squeezed out some juice through a suspicious looking cloth, and offered us a drink, which was really delicious, at the same time holding the seeds in one hand and fencing us off with the other, which we understood to mean that we must not eat or touch the seeds. We thought that their actions were most likely based on some superstitious reason. We little knew, as we left them, how much real anxiety we had given them, or that we owed our lives to their extreme vigilance.”⁹

Today, whilst livestock poisoning continues to occur, little is heard of direct tutu poisoning in people.² Indirect poisoning though has occurred sporadically in recent times through the consumption of toxic honey.² Bees that collect honeydew exudates from vine hopper insects (*Scolypopa* sp.) that have fed on the sap of *Coriaria arborea*¹⁰ can produce honey containing tutin and its toxic derivative hyenanchin (a hydroxytutin) (J. Fountain, personal communication, November 5, 2012).

In 1905 Dr Austin described his treatment of a family who, following ingestion of wild honey, suffered prolonged vomiting, then sustained convulsions over a five hour period followed by several days of delirium in the mother's case.¹¹ Since 1889 there have been 141 reported cases of illness from ingesting toxic honey and four cases of death in New Zealand, with a further 30 cases of children becoming ill after honey was sent to a school in England.¹⁰

The last known cases occurred in 2008, when 22 people fell ill after eating honey from the Coromandel Peninsula which was found to have high levels of tutin.^{10,12} This resulted in the Food Safety Authority calling for tougher controls on the honey supply.¹⁰

Clinical manifestations

Symptoms of tutu poisoning described in case reports from the turn of the twentieth century include vomiting, giddiness, delirium, great excitement, convulsions and coma, ending in death.³ For those who survived, long-term ill health and severe memory impairment has been described.³

Our literature review found the last case report was published in 1972 and described an elderly Maori lady who, following taking a tea made from the leaves, suffered confusion and vomiting.¹³ The New Zealand Poisons Centre describes the onset of nausea and vomiting after a characteristic delay of 3–6 hours, followed by tremor then repeated tonic-clonic seizures and finally respiratory compromise (see Table 1).

When death occurs (usually due to respiratory arrest) it is within 24 hours, and in non-fatal cases, duration of symptoms is dose-dependent and may last 24 hours to 5 days.

Table 1. Severity of Coriariaceae poisoning (acute effects by organ system)

Organ System	Mild Symptoms	Moderate Symptoms	Severe Symptoms
<i>Neurological</i>		Drowsiness, dizziness, tremor, anxiety, excitement, confusion	Generalised weakness, amnesia, incoordination, stupor, tonic-clonic seizures, coma
<i>Gastrointestinal</i>	Dry mouth, nausea	Vomiting, diarrhoea, frequent defecation	
<i>Musculoskeletal</i>		Tremor	Muscle spasm, convulsions
<i>Respiratory</i>		Tachypnoea	Dyspnoea, pulmonary congestion, chest pain, rhonchi on auscultation, respiratory arrest
<i>Cardiovascular</i>		Tachycardia	
<i>Genitourinary</i>		Urinary frequency	
<i>Ocular</i>			Blurred vision
<i>Dermatologic</i>			Jaundice-like appearance

Source: Table compiled from information provided by TOXINZ.⁴

Treatment

The New Zealand Poisons Centre currently an eight-hour period of observation in a facility with advanced life support capability, a bedside ECG and serum electrolytes. There is no specific antidote, gastric decontamination and enhanced elimination are not recommended, and serum levels are not of clinical use. Treatment is then supportive and based on severity of symptoms (see Table 1). A benzodiazepine is considered the mainstay of treatment, especially in those with neurological symptoms⁴.

Case reports

In April 2012, three patients presented to Nelson Hospital Emergency Department (ED) on the advice of local garden centre staff who were concerned they had eaten tutu berries. The trio had been camping in the Kahurangi National Park for some weeks and were tramping beside the Cobb Reservoir near Takaka. They came across “wild berries” which they described as small and black/red in colour.

Cautious tasting revealed a sweet fruit “like a blueberry”, and assuming this meant they were edible, the trio went on to consume “hundreds” of berries each. At around two hours post ingestion (2-HPI) the trio developed nausea, but they attributed this to their travelling on an unsealed road in the back of a truck. However the nausea did not abate after the journey ended and so, worried the berries were to blame, they went to a garden centre.

Patient A had fortuitously taken a photograph of the berries (see below) which staff identified as tutu and advised urgent medical attention.

Photograph 1. Taken by patient A (confirmed to be *Coriaria arborea* by Dr John Steel of the Botany Department, University of Otago)



Photograph 2. *Coriaria arborea* berries



Photograph 3. *Coriaria arborea* leaves



Source: Reproduced with permission of photographer, Wayne Bennett.

The trio arrived at ED at 5-HPI and were seen by a triage nurse. Patient A was a 26 year old male, European tourist with no past medical history of note, taking no medications and no reported allergies; patient B was a healthy 21-year-old female, American tourist; patient C was a healthy 20-year-old female, New Zealand European, with a family history of epilepsy (father).

Patients A and B both complained of moderate nausea, whilst patient C said her nausea was mild. Patient A additionally complained of fatigue and a dry mouth and was noted to be febrile (37.8°C) and “excitable”. Shortly before being seen by medical staff, at 5:30-HPI, patient A had a tonic-clonic seizure which lasted two minutes and spontaneously resolved followed by a post-ictal period of 20 minutes.

There was no deterioration prior to the seizure which was sudden and unexpected. He remained tachycardic (110 bpm) but neurological exam was unremarkable with no tremor and normal reflexes. He was given diazepam 10 mg PO and the decision was made to admit for overnight observation.

During this time patient B developed an elevated heart rate (100 bpm) and became highly anxious, then at 6:30-HPI had a 2-minute, self-resolving tonic-clonic seizure. Following a brief post-ictal period, she too was given diazepam 10 mg PO.

The decision was made to admit all three for observation and to treat patient C prophylactically with diazepam. Patient C at this point had vital signs within normal range and reported feeling entirely well. Following transfer to the intensive care unit (ICU), at 9-HPI, patient A had a second 2-minute tonic-clonic seizure and received a second dose of diazepam. Patients B and C were given a further prophylactic dose of diazepam 5 mg PO.

Overnight observation in ICU was hitherto unremarkable for all three patients. At 20-HPI patients A and B were reviewed and found to have poor recollection of the events (felt to be expected post-seizures), but otherwise had normal vital signs and examination. Patient C continued to be entirely stable. Bloods were taken in ED at 6-HPI, and repeated next day at 20-HPI. Patient A initially had a minimally elevated INR (1.3) which normalised the next day (normal LFTs both samples) and a slight drop in haemoglobin from 147 to 127. Patient B and C's bloods were unremarkable for both samples.

All three patients had normal serial ECGs. The trio were discharged at 22-HPI with no follow-up arranged. Attempts to contact all three patients after 1 week and 1 month were unsuccessful.

Discussion

The tutu plant has gained an infamous reputation due to its devastating effects on New Zealand stock and a number of deaths amongst early settlers. However, in recent decades there has been a paucity of published case reports detailing human poisoning.

Tutu poisoning as described by previous authors and the National Poisons Centre (NPC) begins with mild symptoms including nausea and dry mouth, progresses to moderate symptoms including tachycardia, vomiting and excitability, then severe symptoms, predominantly seizures, develop before death from respiratory arrest.

We present three cases, two of whom developed symptoms with a similar progressive pattern. Patient A, at 5-HPI had moderate symptoms (ongoing nausea, pyrexia and excitability) then at 5:30-HPI developed severe symptoms (first seizure) which continued until 9-HPI (second seizure). Patient B evolved likewise but her seizures occurred at 6:30-HPI, the onset of which, like patient A, was rapid and without warning.

The NPC recommends hospital monitoring for 8 hours post ingestion then, if asymptomatic, the patient can be discharged. However nausea may occur only after a delay of 6 hours, and, given the lack of recent literature, a physician may be falsely reassured their patient has escaped with mild symptoms only. Based on our experience, the development of severe symptoms was abrupt and unheralded and, with no reliable way of determining which patients will progress as such, disposition must be addressed cautiously.

We would recommend extending the observation period for asymptomatic patients to 12 hours, and for those with mild symptoms to 12 hours or longer. This may lead to admissions of patients who remain asymptomatic; however this must outweigh the consequences of developing potentially life-threatening symptoms post discharge.

In our series, patient C had only mild symptoms but given the progressive course of patients A and B was treated with diazepam. We are therefore unable to conclude whether her symptoms simply self-resolved, or were prevented from worsening. However, we suggest that strong consideration is given to prophylactically treating suspected cases of tutu poisoning with benzodiazepines, even if the patient is only mildly symptomatic.

Table 2. Suggested management of Coriariaceae poisoning based on presenting symptoms

All cases of ingestion	Monitored in facility with advanced life support capability Monitoring to include ECG and routine bloods
Asymptomatic	Hospital monitoring for 12 hours Discharge - if remains asymptomatic with normal ECG
Mild symptoms	Hospital monitoring for 12-24 hours Consider prophylactic benzodiazepines
Moderate symptoms	Hospital monitoring for 12-24 hours Consider admission to HDU/ICU Consider prophylactic benzodiazepines
Severe symptoms	Emergency stabilisation (ABC) Benzodiazepines for seizure management

Source: Information modified from TOXINZ current recommendations.⁴

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Dabigatran overdose secondary to acute kidney injury and amiodarone use

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Abstract

Oral direct thrombin inhibitors have improved treatment of non-valvular atrial fibrillation. Safety concerns have been raised since there is no antidote for treatment of secondary haemorrhages and the absence of widely validated test to monitor drug levels. We present a case of dabigatran overdose in an 82-year-old female who was treated with a seemingly appropriate dose.

Oral direct thrombin inhibitors and factor Xa inhibitors have improved treatment of non-valvular atrial fibrillation. However, safety concerns have been raised since there is no antidote to these new agents for reversal of anticoagulation as well as a widely available test to monitor drug levels compared to warfarin.

Case report

We present the case of an 82-year-old female with a history of non-valvular atrial fibrillation who was switched from warfarin to dabigatran 1 week prior to presentation, and who presented with evidence of dabigatran overdose.

The patient presented to the emergency department with complaints of generalised weakness, dizziness and decreased appetite for 2 days, along with nausea and an episode of vomiting the day of presentation from suspected gastroenteritis.

The patient denied melaena, hematochezia, haematemesis, haemoptysis, epistaxis, or any other signs of bleeding. Apart from atrial fibrillation, the patient had a history of chronic systolic congestive heart failure with implantation of an automatic implantable cardioverter-defibrillator, coronary artery disease, hypertension and hyperlipidaemia.

She had been treated with dabigatran 150 mg twice daily, carvedilol, simvastatin, furosemide and amiodarone. One week prior to presentation the patient was started on dabigatran. The decision to start dabigatran was made because of the patient's preference to avoid routine international normalized ratio (INR) evaluation.

Vital signs on admission were stable and physical examination unremarkable. She weighed 79.5 kilograms (kg). A complete blood count was significant only for thrombocytopenia (platelets 95,000/mcl) which had been stable for several years.

Haemoglobin was 12.2 g/dl. The peripheral blood smear showed occasional schistocytes. Chemistry revealed only elevated creatinine of 1.78 mg/dl from a baseline of 1 mg/dl 1 week before and blood urea nitrogen (BUN) of 40 mg/dl from a baseline 20 mg/dl. INR was 7.25 and partial thromboplastin time (PTT) 135 seconds.

The patient denied warfarin use, and that was confirmed with the patient's home health assistant and family.

Fibrinogen level was below 80 mg/dl, D-Dimer level was less than 150 ng/dL and thrombin time was more than 120 seconds, consistent with excessive anticoagulation secondary to dabigatran. The actual dabigatran plasma concentration was not measured.

Dabigatran was held with resolution of coagulopathy over the next 4 days. Subsequently, dabigatran was started at a lower dose of 75mg twice daily after the patient's creatinine had returned to 1.20 mg/dl and BUN of 26 mg/dl with intravenous fluid hydration and improved oral intake. The patient's INR was 1.49 and PTT 40.8 seconds after restarting dabigatran.

Discussion

We present the case of dabigatran overdose in a patient who was treated with a seemingly appropriate dose. Dabigatran, a direct thrombin inhibitor, is not metabolized by cytochrome P450.¹ It produces a more immediate anticoagulation compared to warfarin and does not require bridging with heparin and coagulation monitoring.^{2,3}

Dabigatran is excreted mainly (80%) by the kidneys² and has glycoprotein P efflux transporter mediated interactions with medications such as verapamil and amiodarone.¹ A dose of 150 mg dabigatran was started twice daily in our patient, which has been linked to a higher risk of major bleeding in patients greater than 75 years of age.

Our patient developed acute kidney injury as manifested by an increase in baseline creatinine by more than 1.5 times baseline, possibly due to dehydration, after a brief gastrointestinal illness. This was compounded by the concomitant intake of amiodarone, a p-glycoprotein inhibitor that can increase the peak plasma concentration. This led to both a decreased clearance and increased plasma concentration of dabigatran as manifested by the increase in thrombin time.

The maximum effect of dabigatran on clotting parameters occurs at the same time as maximal plasma concentrations-2 hours after the last dose.⁴ At high plasma concentrations, dabigatran can prolong the prothrombin time in a linear fashion, while the effect of dabigatran on the partial thromboplastin time is curvilinear, with flattening of this response curve at higher concentrations.³

The median peak partial thromboplastin time is approximately two-fold that of control and the trough which occurs 12 hours after the last dose, is 1.5 fold of that of control⁴. The thrombin clotting time (TT) and ecarin clotting time (ECT) are the most sensitive clotting assays for the detection of the action of dabigatran in the blood; a prolongation of either indicates its anticoagulant properties, but contrary to warfarin and INR both tests lack standardization from institution to institution.⁵

The TT assay is most useful as a sensitive method for determining if any dabigatran is present. At therapeutic concentrations, dabigatran prolongs TT in a linear fashion, but at concentrations greater than 600 ng/ml, the test frequently exceeds the maximum measurement time of coagulometers.⁴ The ECT and the Hemoclot® Thrombin Inhibitor assay (Hyphen BioMed, Neuville-sur-Oise, France), a sensitive diluted TT assay, may provide greater clinical precision and a more accurate measure of dabigatran activity than the TT,⁵ however they are not widely available.

There is currently no antidote for dabigatran so overdose must be managed individually. Recent recommendations for management of significant hemorrhage in patients on dabigatran include discontinuation of the medication, mechanical haemostasis, transfusion of factor VII or prothrombin complex concentrates, vigorous diuresis in order to optimise dabigatran clearance and in severe cases, haemodialysis³. The transfusion of fresh frozen plasma does not reverse the anticoagulant effect of dabigatran, but a monoclonal neutralizing antibody is under development.³

There is limited experience on the use of dabigatran. Our case adds to that experience by demonstrating that dehydration along with increased plasma concentrations can alter dabigatran's pharmacokinetic profile and change the bleeding tendency.

Concurrent intake of other medications such as amiodarone can also affect dabigatran's peak plasma concentration. This may present a challenge for patients such as the elderly and the critically ill. To our knowledge there are five more cases of dabigatran overdose reported in elderly patients with impaired renal function.⁶⁻⁸ All five, contrast to our case, had bleeding complications.

Competing interests: There is no potential conflict of interest for any author and this case report was presented as a poster at the American College of Physicians National 2012 Meeting in New Orleans LA, USA.

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Tramadol intoxication and tongue laceration

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Clinical

An unconscious 20-year-old male was brought to the Emergency Room 3 hours after ingesting 3 grams of tramadol with the intention of committing suicide.

On intra-oral examination, the patient had multiple lacerations on the right lateral surface of his tongue (Figure 1).

Figure 1. Lateral tongue lacerations (photo taken after the patient regained consciousness)



What is the significance of this finding?

Answer

Tramadol toxicity can cause nausea, vomiting, agitation, tachycardia, hypertension, central nervous system depression, respiratory depression, and seizures.¹⁻³

Tongue laceration(s) in a tramadol-intoxicated unconscious patient with no witnesses to occurrence of the seizure(s) is a clinical indicator of generalized tonic-clonic seizure.

Of course, its absence does not exclude a seizure diagnosis. It has been shown that lateral tongue biting has a specificity of 100% for the diagnosis of generalized tonic-clonic seizures.^{4,5}

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Colchicine poisoning: defusing the ticking time bomb

We would once again like to shine the spotlight on the danger of colchicine in overdose/poisoning and propose a possible solution to decrease its potential morbidity and mortality in these situations. We agree with our rheumatology colleagues that when taken correctly, it is very beneficial to patients who suffer from gout; however, due to some prescribing practices, it may be a proverbial “ticking time bomb” in many households.

A recent case at our hospital highlights this. An adolescent patient (91.5 kg) spontaneously “took a handful” of her father’s colchicine in a self-harm attempt after an argument. She presented with nausea, vomiting and profuse diarrhoea the following day. From the recently filled bottle of 0.5 mg tablets, 43 out of the 100 tablets were missing, meaning she ingested a maximum of 0.24 mg/kg, which would be consistent with her signs and symptoms.

Good supportive care, the only treatment available for colchicine poisoning, was provided, and she recovered and was discharged 6 days later without sequelae. Sadly, at the time of writing this letter, another patient within our DHB with a known colchicine overdose of 1.4 mg/kg died. Mortality approaches 100% for ingestions of >0.8 mg/kg and when considering the small size of the tablet (Figure 1), it is easy to see how a “handful” of tablets in a spontaneous overdose can be significant.¹

Figure 1. 0.5 mg tablet on a 10 cent coin



Colchicine is currently listed as a “stat” medication with PHARMAC, meaning that if a 3-month supply of 0.5 mg of colchicine twice daily for prevention of gout is prescribed, 180 0.5 mg tablets could be dispensed in a single bottle.² If this amount is ingested by someone weighing 72 kg or less, it is almost always 100% fatal, even with the best medical care available.¹ We understand that the majority of patients use colchicine in acute gout flares rather than prophylaxis, however even then some providers, rightfully taking into consideration the cost of medications to patients, commonly prescribe 100-plus tablets.

We agree with Dr Dalbeth and her colleagues that stating “colchicine...must be used with extreme care” may be alarmist and may discourage practitioners from prescribing a very useful drug in the treatment of gout and that is not the intent of this letter.³ It is not the “quality” that is in question, rather the “quantity” of the tablets being dispensed at one time that we are wanting to draw attention to.

We are urging prescribers to write for a maximum quantity per dispensing of 30 0.5 mg tablets with repeats sufficient for a 3 months’ supply. This would decrease the quantity per bottle, limiting morbidity and mortality in accidental poisonings as seen with children, and spontaneous intentional overdoses (especially the adolescent) in which the patient just grabs the first bottle of medication they have access to.

We are sensitive to the cost and convenience aspects to patients. The \$5 copayment will cover the entire 3 months’ supply regardless of the number of repeats.

The prescriber only needs to:

- State they only want up to 30 tabs dispensed at once.
- Include refills/repeats sufficient for up to 3 months’ supply.

While it may be slightly inconvenient for patients to pick up a new bottle every two weeks, we feel it is a worthwhile compromise to remove a potential harm from the medicine cabinet.

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Time is right for Human Factors in Healthcare

Once a year New Zealand's healthcare professionals get an opportunity to reflect on the impact that human error has on patients nationwide. This period corresponds with the release of the annual report on serious and sentinel events by the Health Quality & Safety Commission, which sometimes occurs at the end of the year,¹ sometimes at the beginning of the new year.²

Last year was somewhat exceptional in that there was a lot of information reflecting on the contemporary status of New Zealand's healthcare system. For example, in April news outlets reported that New Zealand ranked among the six worst industrialised nations in regards to healthcare preventable deaths,³ and a couple of New Zealand Medical Journal's (*NZMJ*) articles highlighted, among other things, that 14% of surgeons were resistant to use procedures for preventing wrong site operations,⁴ and that New Zealand's healthcare should move towards a more public and transparent reporting system.⁵ In fact, such information trend seems to be continuing into 2013, with a January *NZMJ* publication signalling that about 30% of patients may be suffering from medication-related harm in our District Health Boards yearly.⁶

This letter wants to contribute to the topic by reflecting on my own recent experience with healthcare Human Factors and medical error.

Back in 2011 I was invited to address healthcare professionals on Human Factors, at Tairāwhiti District Health and Gisborne Hospital. In preparing for the occasion I discovered with certain dismay that Human Factors is not part of the training curriculum of nurses at Massey University nor could I identify any particular course which could do a similar thing for other health professionals at the University of Otago. The question is, should it be?

Here is where my recent experience comes in. My wife delivered a baby not long ago at MidCentral Health Palmerston North Hospital. During the long labour, her midwife eventually had to get something to eat. While she was gone, my wife needed a new saline drip as well as to go to the toilet, so we called for assistance. The nurse who attended the call (perhaps a nurse student, or was she a fully trained midwife?) was with us for barely a few minutes and still managed to commit four errors in such short period of time.

Firstly, she tried to turn off the electronic fetal monitoring machine but was confused, being perhaps unfamiliar with it, and rather than turning it off she lowered the sound output to a barely audible level; she eventually found the off function, though.

Secondly, after removing the transducers from my wife's belly, she proceeded to disconnect the empty saline drip rather than simply let my wife go to the toilet, considering that she would be taking her I.V. stand with her anyway. However, she did not follow the connecting tube from the saline bag to the cannula, as expected, and ended up disconnecting the oxytocin drip instead; upon realising her mistake, she quickly reconnected the oxytocin tube and pulled the saline tube.

Thirdly, once my wife was back from the toilet and into her bed and reconnected to the machine, the nurse left with the empty saline drip never to come back with a replacement. About 10 minutes later, when the midwife returned, she discovered the last error: that the nurse did not actually disconnect the saline drip properly but simply pulled the tube from the cannula leaving the back stop valve opened.

Those were not the only mistakes happening that day, though, as the midwife also made two mistakes during the long waiting period. At one time she changed the saline drip but forgot to open the valve at the chamber to restore the flow, until queried about it. At another time she changed the oxytocin drip and forgot to re-start the flow until the machine sounded the corresponding alarm.

There are no hard feelings about this experience, though: human error is a commonplace occurrence, not less so in healthcare. What is appalling is that so many mistakes happened in such short period of time doing tasks that were routine, as the errors that occurred could have been prevented easily—for example, with a bit more of awareness about the chances of error occurrence and better procedures.

What I think most appalling is that Human Factors is not taught as part of the training curriculum of healthcare professionals, so that errors as those exemplified above are timely captured or prevented altogether.

I didn't observe any mistakes in the operating theatre, and we left with a healthy baby on Christmas Day. But I was worried all the time I was there. I now wonder if the time is due for Human Factors to find its rightly place in New Zealand's healthcare curriculum and practice.

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Pelvic magnetic resonance imaging for rectal cancer in a provincial centre: time to follow the standards?

Preoperative magnetic resonance imaging (MRI) directed multidisciplinary team discussion of rectal cancer significantly reduces positive circumferential resection margin (CRM) rates.¹ In 2010, the Central Cancer Network (CCN) in the lower North Island adopted the United Kingdom Royal College of Radiologists cancer medical imaging guidelines *Recommendations for Cross Sectional Imaging in Cancer Management*.²

They recommend eight key findings when reporting pelvic MRI for locoregional staging of rectal cancer; tumour site, height from anal verge, infiltrating border morphology, extramural venous invasion, depth of extramural spread, nodal stage, organ invasion and distance to CRM.

The accuracy of MRI staging in rectal cancer outside of expert centres is unknown. Hawke's Bay Hospital (a provincial centre) is part of the CCN and its medical oncology services are provided by MidCentral District Health Board. Patients from the Hawke's Bay with potentially curable rectal cancer are considered for neoadjuvant treatment following discussion by the MidCentral Gastrointestinal Multidisciplinary Team (GIMDT).

An audit was performed to assess the compliance frequency of Hawke's Bay Hospital pelvic MRI reports for rectal cancer staging with CCN guidelines and to compare their accuracy of staging with reports from the GIMDT. Patients presenting with rectal cancer between January 2009 and December 2011 were identified from a prospective database. Those who underwent pelvic MRI staging at Hawke's Bay Hospital and subsequently underwent a rectal resection were included in the study. MRI reports were analysed for their inclusion of the CCN key findings as well as comparability with staging reports issued from the GIMDT.

Eighty-one patients with rectal cancer were identified, of which 32 were excluded leaving a study size of 49 patients. The inclusion of CCN key findings was poor, with no patients having all eight findings included in their MRI report. The reporting frequency of the key findings as recommended by the CCN is outlined in Table 2. T and N stage was accurate with the GIMDT report in 79% of cases. As a result of a difference in reporting, there was a change of treatment recommendation for seven (14%) patients.

Table 1 – Inclusion of CCN key findings in Hawke’s Bay pelvic MRI reports for rectal cancer staging

CCN Key Findings	Inclusion in MRI Report
Site of Tumour	33 (67%)
Height from anal verge	21 (43%)
Morphology Infiltrating border	8 (16%)
Extramural venous invasion	6 (12%)
Depth extramural spread	6 (12%)
Nodal Stage	48 (98%)
Organ Invasion	48 (98%)
Distance to CRM	24 (49%)

Hawke’s Bay reports were also compared with the subsequent histopathology of the rectal resection specimen. However as 74% of patients underwent potentially downstaging neoadjuvant treatment with long course chemoradiotherapy or radiotherapy alone, any comment about histopathological correlation with reporting would be limited.

The reporting of pelvic MRI for rectal cancer in the Hawke’s Bay does not comply with CCN recommendations for the guidance of neoadjuvant treatment decisions. Improving the quality of pelvic MRI reporting, particularly with respect to predicted CRM and maximum depth of extramural invasion, may allow identification of those patients who are at low risk of local recurrence and could be treated with primary surgery or short course radiotherapy alone.

Measures to improve the quality of pelvic MRI reporting ought to be instituted. Pelvic MRI should be reported by a smaller number of the radiologists with sub-specialty interest in pelvic MRI. Reporting using a proforma including the CCN key findings should be considered. Dual reporting may also increase the quality of information provided from the Pelvic MRI, as others have identified that the number of MRI interpreters affects how well MRI predicts CRM involvement.^{3,4} Double reporting by GIMDT radiologists should continue for the foreseeable future.

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Prolonged use of a reminder sticker results in sustained improvement in documentation of resuscitation status

In a previous study reported in this *Journal*,¹ we showed that the short-term use of a reminder sticker at the time of the post-acute medical ward round was associated with a statistically significant improvement in rates of documentation of resuscitation status and appropriate prescription of venous thromboembolism (VTE) prophylaxis.

We aimed to investigate the effects of the prolonged use of this sticker.

The adult general medical service at Auckland City Hospital consists of four ward-based teams (Red, Black, Gold and White). The prolonged use of the sticker was audited on the Red and Black teams.

The sticker contained contact details of the medical team and reminders about documenting resuscitation status, prescribing VTE prophylaxis and retaining or removing intravenous (IV) cannulae.

Prior to the reintroduction of the sticker across all general medical teams the charts of 100 consecutive patients admitted Monday to Friday under both Red and Black teams were reviewed in the afternoon following the post-acute ward round.

Both teams were blinded to this review. The charts were audited for documentation of resuscitation status and the appropriate prescription of VTE prophylaxis (the VTE prophylaxis guideline for medical patients in the Auckland City Hospital RMO Handbook was used to adjudicate this).

We did not audit whether IV cannulae were necessary or unnecessary as we had previously shown that the use of a reminder sticker could improve the removal of unnecessary IV cannulae.²

At the time of the reintroduction of the sticker all medical teams received education highlighting the importance of completing the sticker. They were asked to complete the sticker and place it in the patient's clinical notes at the time of the post-acute ward round. They were aware that sticker use would be audited at some time in the future but were not informed when this would occur. The sticker was then not formally discussed until after the intervention period audit.

The nursing staff responsible for general medical patients were asked to remove a patient's IV cannula if the sticker requested this.

The sticker was reintroduced in June 2011. Four months later the charts of 100 consecutive patients admitted under both Red and Black teams were again audited as above and the same information was collected. The charts were also audited for presence and completeness of the sticker. The patients whose sticker stated "please remove intravenous cannula" were reviewed for the presence or absence of an IV cannula. Both teams were again blinded to this review.

The two tailed Fisher's exact test was used to calculate univariate p values.

Ethical approval was granted by the Northern X Regional Ethics Committee.

Documentation of resuscitation status for the Red and Black team patients improved from 82.5% in the pre-intervention period to 95.5% in the intervention period ($p < 0.0001$).

The prescription of appropriate VTE prophylaxis for the patients that were audited was not significantly different with rates of 29% in the pre-intervention period and 14% in the intervention period ($p = 0.08$).

During the intervention period audit the sticker was present in 155 (78%) of the audited charts and was complete on 119 (77%) occasions. Of the 36 stickers that were incomplete, the following sections were not completed; resuscitation status, VTE prophylaxis and IV cannula removal ($n = 16$), VTE prophylaxis and IV cannula removal ($n = 7$), IV cannula removal ($n = 5$), VTE prophylaxis ($n = 4$), resuscitation status ($n = 3$) and resuscitation status and VTE prophylaxis ($n = 1$).

The sticker asked for the removal of an IV cannula in 32 (16%) patients. When reviewed, a median of 4.5 hours after the sticker had been placed, this cannula remained *in situ* in 15 (47%) patients.

The reintroduction of the reminder sticker was associated with a statistically significant improvement in the rate of documentation of resuscitation status after a prolonged duration of sticker use. There was no change in the appropriate prescription of VTE prophylaxis. The sticker may have resulted in the removal of a number of unnecessary IV cannulae and has the potential to result in the removal of further unnecessary IV cannulae if nursing staff respond to the sticker request more often.

We have shown a prolonged benefit of the sticker in terms of documentation of resuscitation status. Our initial concerns about sticker fatigue were unfounded at least for this aspect of the sticker.

During the period of the sticker use there was a trend towards a reduction in the number of audited patients who received appropriate VTE prophylaxis as adjudicated by the current ADHB RMO handbook guideline. This guideline is now felt by many physicians to be over inclusive. This impression may have been emphasized by a review article, published at the time of the intervention period audit, which found that heparin VTE prophylaxis in medical patients resulted in little or no net benefit.³ It could be argued that the sticker provided benefit by ensuring that the potential use of VTE prophylaxis was considered on the post acute ward round in 127/200 (64%) of audited patients.

IV cannulae remained *in situ* in 47% of patients whose sticker had asked for this to be removed despite a median of 4.5 hours between sticker placement and our review. The removal of unnecessary IV cannulae is reliant on nursing staff reading the patients notes, seeing the sticker and following the request. This adds an extra step to the process of IV cannulae removal which potentially reduces the proportion of unnecessary IV cannulae removed.

Options to potentially increase the removal of unnecessary IV cannula further include additional nursing education about the sticker, for the medical team to remove IV cannulae themselves once the sticker has raised this issue or for a more direct form of communication between the medical team and the nurse caring for the patient.

There were a number of potential limitations to this audit. Changes in personnel on the Red and Black teams between the pre-intervention and intervention periods were unavoidable due to regular rotation of registrars and house officers. Obviously the reminder sticker is only useful if it is used; regular reminders about its use and a plentiful supply of stickers are required.

Given the success of this audit we plan to continue with the use of the sticker. We have updated the sticker by adding a reminder to discuss smoking cessation with patients who smoke. Each rotation of registrars and house officers onto the general medical service will receive education about the use of the sticker as part of their orientation.

We have shown that, at least for documentation of resuscitation status, the reminder sticker has a prolonged benefit.

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Medical Ethics Up-to-Date

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1. If called by night to attend a stranger at a distance, dress quickly and go, never stopping to ask who wants you, or if the bill will ever be paid, lest you be counted inhuman.
2. Never ask how many doctors are in attendance in a case, or how many kinds of patent medicines a patient is taking. Such curiosity on the part of the doctor is vulgar.
3. Never insult a stranger by asking for credentials, nor a patient by asking for money-pounds and shillings are the vernacular of bankers, lawyers, tradesmen, and "workers."
4. Never send in a bill; patients will think you are hard up, but pay your bills promptly. Send a cheque, it looks better.
5. In writing a prescription write illegibly. It does not matter. The druggist will put in "something just as good."
6. Be sure to mention the fact of your being overworked, and also cholecystitis, appendectomy, opsonic index, operative work, toxaemia, words which impress the laity. Your wife must tell her friends how busy you are.
7. When going by a patient's home slip in socially and tell her of some interesting case, or of some operation you have just performed, and incidentally mention how busy you are.
8. Never be friendly with any other doctor. It's unethical. If you think another doctor makes a guinea more a month than you do, cut him dead.
9. If another doctor's name is mentioned in your presence compress your lips, and the patient will understand that your hypertrophied good principles keep you from telling that your principles are so high you can't reach them.
10. If called in after another doctor has been treating a case of meningitis, make your diagnosis "inflammation of the brain," and be sure to say how much better it would have been had you been called in earlier.
11. It is understood that you would not interfere with gestation, but it is well to tell of the large sums of money you have been offered and refused.
12. If the other fellow does not think as you do it proves his inferior intellect.
13. Jealousy and envy are the tributes paid to superiority.
14. Do not expect the "glad eye" when you give the "cold shoulder."
15. We have not enough "skin specialists" in the profession to offset the "wasters" in the laity.

16. Try not to have views. They are distressing—especially to others. If you must think, do it as quietly as possible.
17. Pretend that you are more skilful and proficient than others, and people will soon take you at your own estimation, especially if you can raise a small band of touts and clarjuers.
18. Endeavour to like each other, but if you can't—don't.

Do general health checks in adults reduce morbidity and mortality?

It is assumed that general health checks would be effective in reducing morbidity and mortality but these benefits have not hitherto been demonstrated. This Cochrane systematic review and meta-analysis examines the evidence from 14 randomised trials (182,880 participants) which compared outcomes in those who had health checks with those without such checks.

The result suggest that general health checks in adults do not reduce morbidity or mortality from disease, neither overall nor for cardiovascular or cancer causes. There was an increase in new diagnoses but the researchers suggest that these represent overdiagnosis and overtreatment. Geriatric trials were not included in this study.

BMJ 2012;34:e7191.

Comparison of plain vertebral X-ray and dual energy X-ray absorptiometry for the identification of older women for fracture prevention in primary care

Osteoporosis has been defined in relation to bone mineral density (BMD) measured by dual-energy X-ray absorptiometry (DXA) when the BMD at the spine, hip or wrist is found on DXA to be 2.5 or more standard deviations (SD) below the reference mean for young adults (T-score of -2.5 or less).

This Australian study compares the merits of plain vertebral X-ray and BMD in the identification of osteoporosis and risk of fracture in women older than 70 years. The researchers report on a cohort of nearly 2000 women with a median age of 76 years, not known to have proven osteoporosis, who underwent DXA scanning and thoracolumbar X-rays. The conclusions of their study were that the use of either DXA or X-ray will identify approximately two-thirds of women aged 70 years and over who would be eligible for fracture prevention. The use of X-ray would identify a marginally larger number of women at a lower financial cost but involve substantially greater radiation exposure.

Internal Medicine Journal 2013;43:36–45.

Performance data on surgeons in the UK National Health System (NHS)

League tables showing the results of individual surgeons working in the NHS in England are to be published within two years. Surgical standards in England came under scrutiny in 2001 when high death rates were noted at the Bristol Royal Infirmary. This involved poor outcomes seen in babies undergoing heart surgery. The issue was highlighted again in 2012 when a gynaecologist in Cornwall was suspended over issues of his clinical competence.

The performance data publication is intended to drive improvements in standards by forcing surgeons to deal with performance problems in an open and transparent manner.

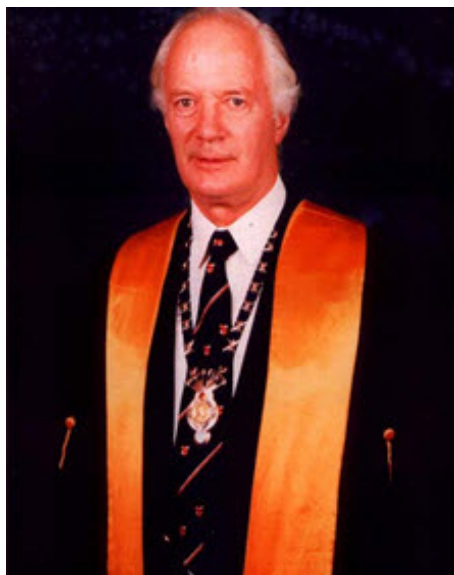
The British Medical Association speculates that surgeons might be deterred from taking on complex and high risk procedures if they were judged solely by “simplistic league tables.”

BMJ 2012;345:e8377.

David Charles Warnock

4 September 1927 – 10 October 2012

David Warnock died on 10 October 2012, aged 85.



This was only 3 months after the diagnosis of adenocarcinoma of the oesophagus.

Despite treatment, his hoped-for ‘window-of-opportunity’ did not eventuate to enable him to enjoy his recent move with his wife Tess to a tranquil villa setting in a retirement village, and to complete his planned change in governance for the Palmerston North Medical Museum since renamed the David Warnock Medical Museum, which he had expanded enormously over 33 years.

David was born on 4 September 1927 in the Dannevirke Hospital, New Zealand. David’s parents lived in nearby Woodville and he had four brothers and one sister.

Two of his older brothers were killed in World War II and another brother was injured in the War. These events were pivotal on David’s subsequent decision to study medicine.

David was initially educated in Woodville and, for a short while, attended Palmerston North Boys’ High School. He and his school-friend Jack Tait decided they would join Radio Corp in Wellington. On being told their studies there were equivalent to BSc papers, they decided they should go to Victoria University. Subsequently, Jack became a Physicist at Christchurch Hospital and David, competing with demobbed army personnel from the war, was accepted at Otago University Medical School where he graduated in 1958.

He served as a House Surgeon in the Wellington Hospital in 1958 and 1959 and then became the Eye Registrar in the same hospital in 1960 to 1961. In 1963, as was common in those days, David travelled to England by ship to further his ophthalmic career. While studying for his Fellowship exam, David’s ailing father died, followed unexpectedly 3 months later by the death of his mother.

Later in 1963, in England, David met up again with his old friend Fred Hollows, later Professor Fred Hollows OA, FRANZCO. He had known Fred in Palmerston North Boys’ High School. Fred preceded him as the Eye Registrar at the Wellington Hospital. Fred told him he was concluding his training position in the Cardiff Eye Department in order to undertake Eye research with the UK Medical Research Council and suggested that David apply for his vacated post. David was duly appointed, and worked in the Cardiff Eye Department from 1963 to 1966. During that

time, David gained the Fellowship of the Royal College of Surgeons of Edinburgh in 1965.

Having completed his ophthalmic training programme, David then returned to Wellington as a visiting consultant to the Wellington Public Hospital and in private practice in 1966. He remained there for a couple of years and in 1968 he was appointed as a visiting consultant Ophthalmologist at the Palmerston North Hospital. In 1969 he obtained the FRACS and the FRACO in 1980. He practised in both the Public Hospital and also the private ophthalmic setting for many years until retirement from the Public Hospital in 1992, and from private practice in 1995.

During David's ophthalmic practice years he was always very personable. He was well respected by his medical colleagues, nursing and associated staff and of course, his patients. He had the pleasure of working alongside his wife, Tess. Tess was appointed as the Orthoptist at the Palmerston North Hospital, and they also worked together in private practice.

David was very active in all hospital teaching, clinical, and administrative meetings, and was a foundation member for several years on the Educational and Qualification Committee of the Ophthalmological Society of New Zealand. David was an Executive member for several years and then the President of the Ophthalmological Society of New Zealand in 1989–1990. Locally, he partook actively in the affairs of the NZ Medical Association.

So called professional retirement did not dampen David's enthusiasm for enjoying life and if anything, he had a busier schedule after retirement, as he had more freedom to pursue his many interests.

David was a member of the The Royal Society, Massey Geological Society, the Manawatu Tramping Club, the Manawatu Officers Club, Manawatu Probus Club and was an active participant of the Manawatu Woodworkers' Guild. He enjoyed wood turning, wood and stone carving and art-painting.

David enjoyed New Zealand's outdoor activities and had tramped most of the National walkways. He had spent 6 weeks in the Himalayas, and climbed the famous Annapurna Crossing. He kayaked the Wanganui River and recently cycled the Middlemarch Rail Trail.

He was a keen scuba-diver, exploring many sunken wrecks and sea-fishing reserves both in New Zealand and overseas. He had a great love of sailing, he built a Mirror dingy and sailed his trailer-sailer on Lake Taupo. He also enjoyed ocean sailing and took the opportunity to sail on the modern-day Endeavour. He also completed a full course of flying instruction for a private pilot's licence.

Back in his days in Cardiff in the 1960s, David became interested in antiques, started his own collection and including 16th Century apothecary jars. This interest continued on his return to New Zealand. Following the death in 1979, of Dr. Neil Little, ENT Surgeon in Palmerston North and at Neil's request, in 1980 David commenced the curatorship of Neil Little's large collection of antique hearing aids and ENT Instruments. David subsequently expanded this collection into the largest collection of medical antiquities in New Zealand. In 2001 he formed a Charitable Trust to govern the Museum with David as the inaugural chairman until his death.

Foreseeing the vast museum project ahead, David undertook University study and was awarded a Diploma in Museum Studies from Massey University. One of the most important tasks was to be the correct systematic cataloguing of the medical collection and this he accomplished. His work was acknowledged by the Manawatu Branch of the Royal Society in 2009 by awarding David with a 'Certificate of Excellence'.

Over the years he initiated and coordinated many items that were serviceable or reduplicated to be consigned to needy destinations around the Pacific and Nepal etc. For example instruments, a dental chair and older-style hospital beds went to the Solomon Islands. Items were loaned for many film projects, to local amateur theatrical groups and conferences. He opened the museum to schools and various other visitors and dignitaries.

David will always be remembered as a very caring and competent Ophthalmologist who always had the best interests of his patients at heart. He was a very convivial colleague and friend. As a dedicated family man, he supported Tess with her interests and he treasured observing and partaking in a wide range of indoor and outdoor activities with his daughter and grand-children. All his friends across a wide range of interest groups will have very fond memories of their association with David and he will be sadly missed. We all extend our sincerest condolences to his wife Tess, daughter Cate and her family.

Various events and opportunities over the years no doubt influenced David's path through life. He was adaptable, had a wide range of talents and interests, had a delightful personality and sense of humour and he has left us with a significant Medical Museum—the David Warnock Medical Museum.

'In the end, it's not the years in your life that count. It's the life in your years'. *Abraham Lincoln*

Dr Philip Boulton (FRANZCO) wrote this obituary; he is a 40-year friend and colleague.

Beryl Overton Howie

7 November 1924 – 1 December 2012; QSO, DSc Otago, MB ChB, FRCS, FRACS, FRCOG, FRACOG

Born in Invercargill, educated at Epsom Girls' Grammar School, Beryl Howie gained a national University Scholarship, and completed her Medical Intermediate at Auckland in 1944. While at Otago Medical School she boarded at St Margaret's College, becoming student president in 1947.



Following Sixth Year at Christchurch and graduating in 1949, she served as House Surgeon at Timaru before gaining a post at the Radcliffe Infirmary, Oxford, England, where she trained for her MRCOG. Under Sir John Stallworthy, it was a rigorous apprenticeship. In 1959 Beryl was appointed to lead the Department of Obstetrics and Gynaecology at the Christian Medical College (CMC), Ludhiana, Punjab, India. Three years later, promoted to full Professor within the University of Punjab, (becoming the first New Zealand woman medical graduate to hold a full medical chair), she played a key role in upgrading CMC from licentiate to MBBS standard.

Founded in 1894 by Dame Edith Brown, CMC was the first medical college for women in Asia; after 1953 men were also able to enter. Along the way she learnt to speak Hindustani and Punjabi. She was funded initially by overseas groups supporting CMC and from 1963 by the Presbyterian Church of Aotearoa-New Zealand.

In 22 years at CMC Beryl delivered thousands of babies, taught undergraduates to provide quality care with limited resources, trained 90 to postgraduate diploma level and trained 29 to become doctorates in medicine. She modelled for her registrars, residents and students the compassion and competence needed to manage the difficult deliveries, all-too-often obstructed, seen daily in Indian hospitals. The training she gave meant her trainees coped well wherever they worked in India. Her concern went out into the community, and she involved the indigenous midwives, the dais.

Cervical cancer was common and treated with radium, and she was expert in the repair of vaginal fistula. From staff quarters to labour ward at the Old Brown Hospital was 5 minutes by bicycle at any time of night—in those days it was a busy street of Old Ludhiana, congested with rickshaws and bicycles, cows and cars. According to former student Dr TK Cherian (Head of Reproductive and Foetal Medicine, St Stephen's Hospital, New Delhi) "I remember her evening rounds in the ward when patients could even share their personal problems and get solace in her loving words and soft touch. The magnitude of love and respect her patients had for Dr Howie was

evident the night she boarded the train from Ludhiana station—the platform was an ocean of her patients and their families.”

The New Zealand High Commissioner presented her with the Queens Service Order before she left India to return to New Zealand in 1981. Her textbook *High-Risk Obstetrics, a practical handbook*, was published by Macmillan in 1985. From 1984-89 was Medical Advisor to Interserve, an international Christian network working across Asia, until she officially retired to Auckland in 1990. In 1989 she received an honorary Fellowship of the Royal Australasian College of Obstetricians and Gynaecologists. Named after her at CMC is a new six-storied block housing the Obstetrics and Gynaecology Department, for which churches and other community groups in New Zealand raised the funds, matched by a grant from the New Zealand Government.

In her youth Beryl was an enthusiastic hockey player, and also learnt the cello. In retirement she resumed her interest in music, especially chamber music. She continued to actively support people serving overseas, as well as student groups. Beryl also had a great love of children, whether nieces, nephews, or the children of friends, and followed with warm interest babies she had delivered. That interest extended to the students she had taught, and to their children also. She charmed our children with her humour and with enthusiastic involvement in their interests.

Some 200 came to her memorial service in Ludhiana. Former students, residents and registrars sent messages vividly recalling the competence, compassion and determination she modelled. These qualities and her commitment to people in need were based on her lifelong Christian faith, which was deep, personal and attractive.

She is survived by her brother, John Howie, and her nephews and niece, Hamish, Jock and Melissa.

This obituary was written by former colleagues David and Rosemary Troughton and Murray Laugesen of Christchurch.