



CONTENTS

This Issue in the Journal

- 4 A summary of the original articles featured in this issue

Editorials

- 6 Managing the skin cancer surge
Jeremy W Simcock
- 8 BCG and bladder cancer
Peter J Gilling

Original Articles

- 10 The Voice of Experience: Results from Cancer Control New Zealand's first national cancer care survey
Inga O'Brien, Emma Britton, Diana Sarfati, Wayne Naylor, Barry Borman, Lis Ellison-Loschmann, Andrew Simpson, Craig Tambllyn, Chris Atkinson
- 20 Predictors of physical activity and quality of life in New Zealand prostate cancer survivors undergoing androgen-deprivation therapy
Justin W L Keogh, Daniel Shepherd, Christian U Krügeloh, Clare Ryan, Jonathan Masters, Greg Shepherd, Rod MacLeod
- 30 Robot-assisted laparoscopic prostatectomy: a 2010 update
James B Duthie, Joanna E Pickford, Peter J Gilling
- 35 Immunohistochemical testing for colon cancer—what do New Zealand surgeons know?
Simon J Harper, Alison R McEwen, Elizabeth R Dennett
- 41 A comparative analysis of cardiovascular disease risk profiles of five Pacific ethnic groups assessed in New Zealand primary care practice: PREDICT CVD-13
Corina Grey, Sue Wells, Tania Riddell, Romana Pylypchuk, Roger Marshall, Paul Drury, Raina Elley, Shanthi Ameratunga, Dudley Gentles, Stephanie Erick-Peleti, Fiona Bell, Andrew Kerr, Rod Jackson
- 53 Pathology referrals for skin lesions—are we giving the pathologist sufficient clinical information?
Marius Rademaker, Murray Thorburn

Review Article

- 59 Non-melanoma skin cancers in New Zealand—a neglected problem
Nicholas D L Brougham, Elizabeth R Dennett, Swee T Tan

Viewpoint

- 66 Changes to the eligibility to bill on Medicare in Australia: a threat to New Zealand's medical workforce?
Katie Elkin

Clinical Correspondence

- 72 BCG sepsis following inadvertent intravenous BCG administration for the treatment of bladder cancer can be effectively cured with anti-tuberculosis medications
Ziya Akbulut, Abdullah E Canda, Ali F Atmaca, Haci I Cimen, Canan Hasanoglu, Mevlana D Balbay
- 78 Isolated melanoma metastasis to stomach with possible regressed primary lesion: the importance of pursuing solitary melanoma metastases
James D McKay, Alf Deacon
- 80 A case of a testosterone-secreting oncocytic adrenocortical carcinoma
Munanga Mwandila, Hayley Waller, Victoria Stott, Philippa Mercer
- 83 Medical image. Varicella-zoster virus pneumonia
Hsi-Che Shen, Tsu-Tuan Wu, Sheng-Hsiang Lin

100 Years Ago in the NZMJ

- 85 Obituary: Thomas Hocken

Proceedings

- 87 Proceedings of the Waikato Clinical School Biannual Research Seminar, Wednesday 13 October 2010

Methuselah

- 96 Selected excerpts from Methuselah

Letters

- 98 Sample, send, screen, survive—simple. Rotary Club-subsidised community trial points way to simple screen for bowel cancer risk
Derek Anderson, Frik de Beer
- 99 Screening for diabetes during and after an acute myocardial infarction: when and how?
Lakshminarayanan Varadhan, David Barton

- 102 Sugar consumption in New Zealand—with Thornley and McRobbie
response
Winsome Parnell, Jane Dodd, Donnell Alexander
- 105 Ultrasound-guided nerve blocks are costly
Geoffrey Horne
- 106 Pelvic floor exercises
David H B Speary

Obituaries

- 107 Graham Collingwood (Mont) Liggins
- 109 Melvin Athol Brieseman



This Issue in the Journal

The Voice of Experience: Results from Cancer Control New Zealand's first national cancer care survey

Inga O'Brien, Emma Britton, Diana Sarfati, Wayne Naylor, Barry Borman, Lis Ellison-Loschmann, Andrew Simpson, Craig Tamblin, Chris Atkinson

A first ever survey of the experiences of people seeking outpatient cancer treatment in the New Zealand health system reveals very high satisfaction levels, balanced by plenty of room for improvement. The results of the 2009 Cancer Care Survey show 97% of survey respondents were satisfied with publicly funded outpatient cancer care. Most people were very positive about their care coordination and the level of privacy, dignity and respect provided by healthcare professionals. Areas for improvement include provision of emotional support and information, and consideration of the patient's circumstances in planning treatment.

Predictors of physical activity and quality of life in New Zealand prostate cancer survivors undergoing androgen-deprivation therapy

Justin W L Keogh, Daniel Shepherd, Christian U Krägeloh, Clare Ryan, Jonathan Masters, Greg Shepherd, Rod MacLeod

Men with prostate cancer had lower levels of quality of life than those without the cancer. Although less than 50% of the cancer survivors were categorised as physically active, those who were active had greater quality of life than those who were less active. Increased levels of physical activity may therefore help increase the cancer survivors' quality of life; and this may be achieved by focusing on the survivors' attitudes to exercise as well as the extent to which they believe they control their exercise behaviour.

Robot-assisted laparoscopic prostatectomy: a 2010 update

James B Duthie, Joanna E Pickford, Peter J Gilling

RALP is a technique which has replaced Open Prostatectomy in most major institutions in the US and Europe. The learning curve is short for skilled open surgeons and satisfactory outcomes can be achieved after a relatively small number of cases. This procedure has been successfully introduced into Australasia in recent times with results which are similar to large international series.

Immunohistochemical testing for colon cancer—what do New Zealand surgeons know?

Simon J Harper, Alison R McEwen, Elizabeth R Dennett

8–12% of colorectal cancers are associated with genetic syndromes, i.e. are hereditary. The most common of these is Lynch Syndrome for which there are clinical criteria (Bethesda) that can be used to identify colorectal cancer patients who may have this syndrome. If identified testing can be undertaken to confirm the diagnosis. A pathologist who screens the cancer specimens with special stains initially undertakes the testing. Treating surgeons need to know these clinical criteria in order to request appropriate testing from pathologists. The aim of this study was to assess the knowledge of New Zealand surgeons about the Bethesda criteria.

A comparative analysis of cardiovascular disease risk profiles of five Pacific ethnic groups assessed in New Zealand primary care practice: PREDICT CVD-13

Corina Grey, Sue Wells, Tania Riddell, Romana Pylypchuk, Roger Marshall, Paul Drury, Raina Elley, Shanthi Ameratunga, Dudley Gentles, Stephanie Erick-Peleti, Fionna Bell, Andrew Kerr, Rod Jackson

Few studies have compared the cardiovascular risk factor profiles of different Pacific ethnic groups in New Zealand. The PREDICT study is the largest to date measuring cardiovascular disease risk factors in Pacific peoples (11,642 aged 35–74 years). We found differences in various cardiovascular disease risk factors, including smoking, cholesterol and blood pressure levels, between five Pacific ethnic groups (. Despite these differences in individual risk factors, the estimated risk of experiencing a cardiovascular event varied very little between Pacific groups.

Pathology referrals for skin lesions—are we giving the pathologist sufficient clinical information?

Marius Rademaker, Murray Thorburn

This audit shows that in over a third of histology requests, important clinical information was not included. Sixty percent of confirmed malignant lesions had not been identified on the request form as being malignant, including 87% of melanomas, 55% of basal cell carcinomas (BCCs) and 43% of squamous cell carcinomas (SCCs). The specimen was inadequate to make a diagnosis in 6.7% of cases. Finally, 40% of lesions suspected of being a melanoma were sampled by punch biopsy, which is considered suboptimal. Clearly there is room for improvement.



Managing the skin cancer surge

Jeremy W Simcock

Non-melanoma skin cancer (NMSC) is not a one-off event for a small number of individuals. Rather it is repeated problem affecting a large proportion of the population through the second half of their lives. Brougham and colleagues' review¹ of the possible incidence of NMSC in New Zealand suggests that the problem is both large and increasing. However we really do not know for certain as there is no reliable data on our true incidence of NMSC, let alone how the incidence is changing over time.

We have good information for the small but important sub-group of renal transplant recipients. In comparison with similar populations in other countries, patients in New Zealand have high rates of both NMSC incidence and mortality.² For the New Zealand population as a whole, 122 deaths were attributed to NMSC in 2007 (twice that of cervical cancer).

Extrapolation of overseas data suggests that the age-specific incidence rates of NMSC are increasing relentlessly. For example, the Danish rates have more than doubled in the last 30 years,³ and Scottish rates are estimated to be increasing 1.4–5.1% year-on-year.⁴ This combined with a burgeoning population of fair-skinned over-60s explains the large increase in absolute numbers of patients presenting with NMSCs. More recent literature suggests that age-specific incidence rates may not be increasing across the board. The Australian paper which provides the best information from our region⁵ states that rates have stabilised in under 60 year-olds. Similarly, a recent Canadian study⁶ has shown rates stabilising for NMSCs occurring on the head and neck, but not for other sites.

In the absence of local epidemiological information, we are left to observe how individual patients with NMSC are faring. Treatment services in secondary care are struggling to meet 6-month targets despite a typical clinical priority of 6 weeks. Some services have introduced barriers to referral such as the need for histological confirmation of malignancy as described by Rademaker and Thorburn.⁷ This results in an unnecessary procedure for the patient and histopathology costs to the funder. For a cancer that has an estimated annual treatment cost of NZ\$22–50 million per year,¹ the question must be asked as to whether we can afford to continue implement such inefficient and expensive barriers to patient care.

In contrast to most cancers which are primarily managed by a well circumscribed group of hospital-based specialists, NMSC treatment is provided across primary, secondary and tertiary care. Although this broad-based involvement is identified as a barrier to data collection,¹ it provides the potential solution to manage what may well be the single biggest increase in demand in cancer services.

The challenge is to coordinate treatment services so that most NMSC is treated locally at the time of diagnosis and that those cases that are more complex receive expeditious specialist treatment. NMSC does not fit current hospital-based cancer

service models well and is more appropriately managed in primary care as a persistent health problem. Identifying and treating NMSC early is both beneficial to the patient and more cost effective.

Now is the time for those involved in skin cancer management to step forward and become involved in both the determination of the true incidence of NMSC in New Zealand (as requested by Brougham) and monitor the outcomes of treatment that we are currently providing. Given the predicted surge of demand coming our way, this information is vital to planning our cancer services.

Competing interests: None.

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References:

1. Brougham ND, Dennett ER, Tan ST. Non-melanoma skin cancers in New Zealand—a neglected problem. *N Z Med J.* 2010;123(1325). <http://www.nzma.org.nz:8080/journal/123-1325/4421/content.pdf>
2. Mackenzie KA, Wells JE, Lynn KL, Simcock JW, Robinson BA, Roake JA, Currie MJ. First and subsequent nonmelanoma skin cancers: incidence and predictors in a population of New Zealand renal transplant recipients. *Nephrol Dial Transplant.* 2010 Jan;25(1):300–6.
3. Birch-Johansen F, Jensen A, Mortensen L, et al. Trends in the incidence of nonmelanoma skin cancer in Denmark 1978-2007: Rapid incidence increase among young Danish women. *Int J Cancer.* 2010 Nov 1;127(9):2190–8
4. Brewster DH, Bhatti LA, Inglis JH, et al. Recent trends in incidence of nonmelanoma skin cancers in the East of Scotland, 1992-2003. *Br J Dermatol.* 2007 Jun; 156(6):1295–300
5. Staples MP, Elwood M, Burton RC, et al. Non-melanoma skin cancer in Australia: the 2002 national survey and trends since 1985. *Med J Aust* 2006;184(1):6–10
6. Jung GW, Metelitsa AI, Dover DC, Salopek TG. Trends in incidence of nonmelanoma skin cancers in Alberta, Canada, 1988-2007. *Br J Dermatol.* 2010 Jul;163(1):146–54.
7. Rademaker M, Thorburn M. Pathology referrals for skin lesions—are we giving the pathologist sufficient clinical information? *N Z Med J.* 2010;123(1325). <http://www.nzma.org.nz:8080/journal/123-1325/4420/content.pdf>



BCG and bladder cancer

Peter J Gilling

The overall lifetime risk of developing bladder cancer is said to be 1 in 28 and it is three times more common in men than women. It is currently the most expensive cancer from diagnosis to death in the United States.¹ The aetiology is multifactorial being a combination of exogenous environmental factors and endogenous molecular factors. At presentation, approximately 70–75% of these tumours are confined to the mucosa (75%) or lamina propria (25%)—i.e. are non-muscle invasive—with the most common pathological subtype being transitional cell carcinoma (in 90%).

Non-muscle invasive tumours are initially treated by complete endoscopic resection with all visible lesions being removed. Intravesical chemotherapy or immunotherapy can be used perioperatively or postoperatively to prevent recurrence and progression following initial trans-urethral resection of bladder tumour (TURBT).

The most common immunomodulatory agent used, since its introduction for this purpose in the 1970s, has been Bacillus Calmette-Guerin (BCG). Typically this is commenced several weeks after tumour resection, once healing of the urothelium has occurred, as an induction course followed by maintenance therapy for up to 3 years. The mechanism of action of this agent is thought to involve a massive local immune response following direct binding of BCG to fibronectin in the bladder wall. A cell-mediated cytotoxic mechanism is thought to be responsible for the efficacy of BCG.²

The article by Akbulut and colleagues³ in this issue of the *Journal* describes an unfortunate sequence of events following the inadvertent intravenous administration of BCG (which is an attenuated mycobacterium initially developed as a vaccine). Although serious, for obvious reasons this is not one of the side-effects commonly seen in practice but is a timely reminder nonetheless.

Intravasation usually occurs only if the BCG is given in the presence of gross haematuria, active urinary tract infection, following traumatic catheterisation or if given too soon after TURBT and may result in severe systemic symptoms. More usual are mild irritative voiding symptoms, mild fever and haematuria. Occasionally two or three drug anti-mycobacterial therapy is required for periods of up to 6 months when symptoms are severe.

BCG is particularly useful for the treatment of carcinoma-in-situ (CIS) with initial tumour-free response rates of up to 80%, although long-term progression for this 'high-risk' disease may still be seen in 20% of these initial responders. Tumour recurrence rates are generally reduced by around 40% overall when compared to TURBT alone in non-muscle invasive disease. Unlike chemotherapy, progression to higher stage disease can also be reduced by 20–30% though an overall survival advantage has yet to be demonstrated.⁴

A common conundrum in clinical practice is defining and managing BCG failures. These patients have a 50% chance of disease progression and death with ongoing

intravesical treatment . A second 6-week induction course will result in ‘salvage’ of a further 15–20% of cases but beyond this, further intravesical therapy with interferon, gemcitabine and taxanes is considered experimental.

‘Early’ cystectomy (i.e. before evidence of muscle-invasive disease) can be considered following BCG therapy, for any sign of tumour recurrence unless it is a delayed recurrence with low-grade disease. In this situation up to 40% of patients will be proven to have muscle-invasive disease and/or positive lymph nodes at the time of surgery. At 5 years however, most patients who have cystectomy for early disease will be cured.

Competing interests: None.

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References:

1. Jemal A Seigel R, Ward E, et al Cancer Statistics,2009. CA Cancer J Clin 2009;59:225–49.
2. Hall MC, Chang SS, Dalbagni G, et al. Guidelines for the management of non-muscle invasive bladder cancer (Stages Ta, T1 and Tis): 2007 Update. J Urol 2007;178:2314–30.
3. Akbulut Z, Canda AE, Atmaca AF, et al. BCG sepsis following inadvertent intravenous BCG administration for the treatment of bladder cancer can be effectively cured with anti-tuberculosis medications. N Z Med J. 2010;123(1325). <http://www.nzma.org.nz/journal/123-1325/4412>
4. Persad R, Lamm D, Brausi M, et al. Current approaches of the management of non-muscle invasive bladder cancer :Comparison of current guidelines and recommendation. Eur Urol 2008;Supp 7:637–650.



The Voice of Experience: Results from Cancer Control New Zealand's first national cancer care survey

Inga O'Brien, Emma Britton, Diana Sarfati, Wayne Naylor, Barry Borman,
Lis Ellison-Loschmann, Andrew Simpson, Craig Tamblyn, Chris Atkinson

Abstract

Aims The 2009 Cancer Care Survey aimed to gather information from patients about their experiences receiving outpatient cancer care.

Methods In mid-2009, Cancer Control New Zealand sent an NRC+Picker postal survey to a stratified sample of 3251 eligible adults, who had received outpatient cancer care between October 2008 and March 2009. Eight cancer treatment facilities across New Zealand provided patient lists from which potential respondents were selected.

Results The final response rate to the survey was 68%. Most of the patients surveyed responded very positively to questions related to specialist care coordination (91% positive response; 95%CI: 90–93), the level of privacy (87% positive response; 95%CI: 85–89), and the dignity and respect provided by healthcare professionals (86% positive response; 95%CI: 85–88). However, patients tended to be much less positive about the level of information they received on the effects of cancer treatment on their day-to-day life (responses ranging between 30% and 40% positive) and the level of emotional support provided (36% positive response; 95%CI: 33–39). Responses from different cancer services tended to follow similar patterns, although for twelve questions there was at least a 20% difference in response between services.

Conclusions Overall, patients rated their outpatient cancer care experiences as positive, but important gaps exist in the provision of information, emotional support, and treating patients within the context of their living situation. Cancer patient experience surveys can achieve high response rates and generate useful information on patient perceptions of their care. This data can be used to inform quality improvement efforts at both national and cancer treatment service levels.

Quality cancer care has both technical and service components. Technical skills are critical to the effective diagnosis and treatment of disease. Service skills are required to holistically meet the needs of patients' and address their expectations. A combination of technical and service skills is necessary to address cancer patients' medical and non-medical needs and wants over the duration of a cancer journey. Patients' experiences of care can be sought and reviewed with the goal of incorporating these voices of experience into quality improvement efforts.

The importance of including the patient's perspective in evaluations of care is reflected in key New Zealand government documents such as the New Zealand Health Strategy (2000), which has an underlying fundamental principle that there should be "active involvement of consumers and communities at all levels".¹ Additionally, two of the guiding principles of the New Zealand Cancer Control Strategy (2003) are that

activities should “*reflect a person-centred approach*” and “*actively involve consumers and communities*”.²

Furthermore, the recent Ministerial review of the Health System has described the “...*Government’s vision of a public health and disability service that is more patient-than provider-centric, giving patients more control*”.³ Analysing patients’ experiences of care, to inform improvements in health services, adheres to these principles and ideas.

With the general public, healthcare professionals and the Government all looking for improvements in the quality and delivery of cancer care, surveying cancer patients’ experiences is a promising method of exploring if the services consumers receive match their needs and expectations. This type of survey puts the consumer at the centre of the evaluation of care and the results provide stakeholders with a unique perspective on New Zealand’s performance in providing quality cancer care.

In 2009, Cancer Control New Zealand (CCNZ), formerly known as the Cancer Control Council, initiated the Cancer Care Survey, which was the first national survey of cancer patient experiences.

The overall aim of this study was to collect data on how well outpatient cancer treatment services were meeting the needs and expectations of cancer patients. CCNZ’s specific 2009 Cancer Care Survey objectives were to:

- Generate and analyse data on patients’ experiences of cancer care, to inform advice provided to the Minister of Health and other key stakeholders
- Provide baseline data on cancer patient experiences that can be compared with data collected in subsequent surveys, so that the impact of system changes on patients’ experiences of care can be explored over time.

Three reports on the Cancer Care Survey have been written and are available on CCNZ’s website (www.cancercontrolnz.govt.nz). The *Voice of Experience Part One* report provides preliminary national results from the survey.⁴ The *Voice of Experience companion* report provides the results for the eight participating cancer treatment services.⁵ The *Voice of Experience Part Two* report⁶ includes a select review of the patient experience literature, in-depth analysis of the survey data, and recommendations.

This article focuses on summarising the overall national findings from the survey.

Methods

Participants and setting—The target population for the survey was patients 18 years of age and older with a confirmed diagnosis of cancer who had undergone, or were undergoing, publicly funded cancer outpatient treatment (specifically chemotherapy or radiotherapy) in New Zealand. Patients were excluded from the survey if they had no fixed address, had moved out of New Zealand, were not residents of New Zealand, had received only inpatient services, or were deceased.

The participating facilities included the six regional cancer centres providing the majority of cancer services in New Zealand (Canterbury Oncology Service, Northern Region Cancer Centre, Palmerston North Regional Cancer Treatment Service, Southern Blood and Cancer Service, Waikato Regional Cancer Centre, and Wellington Blood and Cancer Centre) and two satellite chemotherapy treatment facilities with vocationally registered oncologists on staff (Nelson Oncology Service and Tauranga & Whakatane Cancer Centres). These services provided lists of all their outpatients who met the criteria for the study during the 6-month period from 1 October 2008 to 31 March 2009.

A sample was randomly drawn from these lists with an aim to select 500 people from each cancer treatment service. If less than 500 people were submitted by a cancer treatment service then all individuals were selected.

All those who had their ethnicity recorded as Māori but had not been randomly selected, were also included in the sample. This oversampling process aimed to increase the precision of the estimates for Māori. Prior to proceeding with the survey, ethics approval was obtained from the Multi-Region Ethics Committee (MEC/09/22/EXP).

Survey instrument—The 2009 Cancer Care Survey, a tool developed and validated⁷ by NRC+Picker (USA), was mailed to the selected participants. The questionnaire included 96 questions, with multiple response options, covering a variety of patient experiences related to their cancer treatment, including their diagnosis, treatment (surgery, chemotherapy and radiotherapy), symptom management, healthcare team, care environment and overall impressions of care. The survey also contained demographic questions including the respondent's age, gender, household annual income after tax and ethnicity.

Surveys were posted to 3525 selected participants and were also available online. A covering letter, signed by Dame Catherine Tizard (CCNZ chair 2005–2009), explaining the importance of the survey was attached to each survey form. To increase the response rate, posters about the survey were placed in cancer treatment service waiting rooms and patient support service buildings. A telephone help line was set up to answer any queries from survey recipients. Reminder post cards and a second survey were sent out to those who had not responded to the initial survey after 6 weeks.

Analysis—The data analysis was carried out using Stata (StataCorp LP) software, versions 9.2 and 10. The distribution of the target, surveyed and eligible respondent populations were compared across key demographic characteristics, and the response rates were also compared across the different ethnic, age, gender, income and cancer treatment service groups.

The sample data were weighted to take into account the probability of selection. Post stratification weights were also calculated for each respondent using the age and ethnicity distribution of the target population in each cancer treatment service.

Sixty-five of the 96 survey questions had ordinal categorical response options which could be grouped into dichotomised positive and negative response categories. In this 'top box' scoring approach, only the 'ideal' response, such as "always" or "definitely", was equated with a positive response. This categorisation process allowed the questions to be rank ordered from the highest percentage of positive responses to those with the lowest percentage of positive responses, so the questions in the upper and lower quartiles could be examined both nationally and at the individual cancer treatment service levels.

Results

In total 3525 people were mailed surveys. A total of 410 people were excluded (136 during pre-mailing checks, 254 post mailing and 20 post data cleaning). Replacements were randomly selected from the corresponding patient lists for the 136 people excluded pre-mailing.

Reasons for the 274 exclusions post-mailing included: the person was deceased, the person stated they did not receive treatment for cancer, the person was too unwell or otherwise unable to complete the survey, the survey being returned to sender as undeliverable, or the returned questionnaire being illegible or damaged. The final total survey population was 3251. Of the 3251 eligible respondents, surveys were returned by 2221 people resulting in a 68% final response rate.

According to the ethnicity information obtained from the cancer treatment services, New Zealand Europeans made up 69% of the respondent population, while 13% were Māori, 3% were Asian, and 2% were Pacific Islanders (Table 1). The highest proportion of eligible respondents was in the 50–69 years age group and over half of the survey population was female.

The proportion of those within the different ethnic, age, and gender groups in the targeted, surveyed and respondent populations were similar (Table 1). For example, 50% of the targeted population, 49% of the surveyed population and 51% of the respondent population were in the 50–69 years age group.

Although the overall response rate for Māori was lower (51%) than for New Zealand Europeans (74%), the proportion of Māori in the surveyed population was higher than the proportion of Māori in the targeted population due to deliberate over-sampling.

Table 1. Characteristics of the target, surveyed and eligible respondent populations

Characteristics	Original target population* % (n)	Surveyed population** % (n)	Eligible respondents % (n)
Ethnicity†			
NZ European	68 (4885)	63 (2051)	69 (1525)
Māori	10 (736)	18 (586)	13 (296)
Pacific Island	3 (185)	2 (79)	2 (35)
Asian	4 (267)	3 (99)	3 (58)
Other	15 (1104)	13 (436)	14 (307)
Age group			
<30	2 (155)	2 (76)	1 (32)
30–49	17 (1219)	18 (590)	16 (357)
50–69	50 (3611)	49 (1590)	51 (1129)
>70	31 (2192)	31 (995)	32 (703)
Cancer treatment service location			
Auckland City	33 (2355)	18 (586)	16 (354)
Tauranga	12 (895)	16 (504)	16 (348)
Waikato	20 (1427)	15 (494)	14 (308)
Palmerston North	13 (945)	16 (517)	16 (352)
Wellington	7 (503)	15 (489)	17 (369)
Nelson	1 (100)	3 (88)	3 (70)
Canterbury	11 (805)	14 (442)	15 (337)
Dunedin	2 (147)	4 (131)	4 (83)
Gender‡			
Female	59 (4234)	59 (1920)	59 (1317)
Male	41 (2943)	41 (1331)	41 (904)
Total	7177	3251	2221

* The original target population includes everyone submitted from the cancer service patient lists and includes people who were subsequently excluded on the basis of being deceased, not having received treatment for cancer, being too unwell or unable to complete the survey, the survey being returned to sender as undeliverable, or the returned questionnaire being illegible or damaged.

** Includes Maori selected by the oversampling process.

† Cancer treatment services are required to collect ethnicity data from people accessing their services based on the Ministry of Health Ethnicity Data Protocols for the Health and Disability Sector. There were minor variations between the ethnicity data collected by the cancer treatment services and the self-reported ethnicity data collected by the survey.

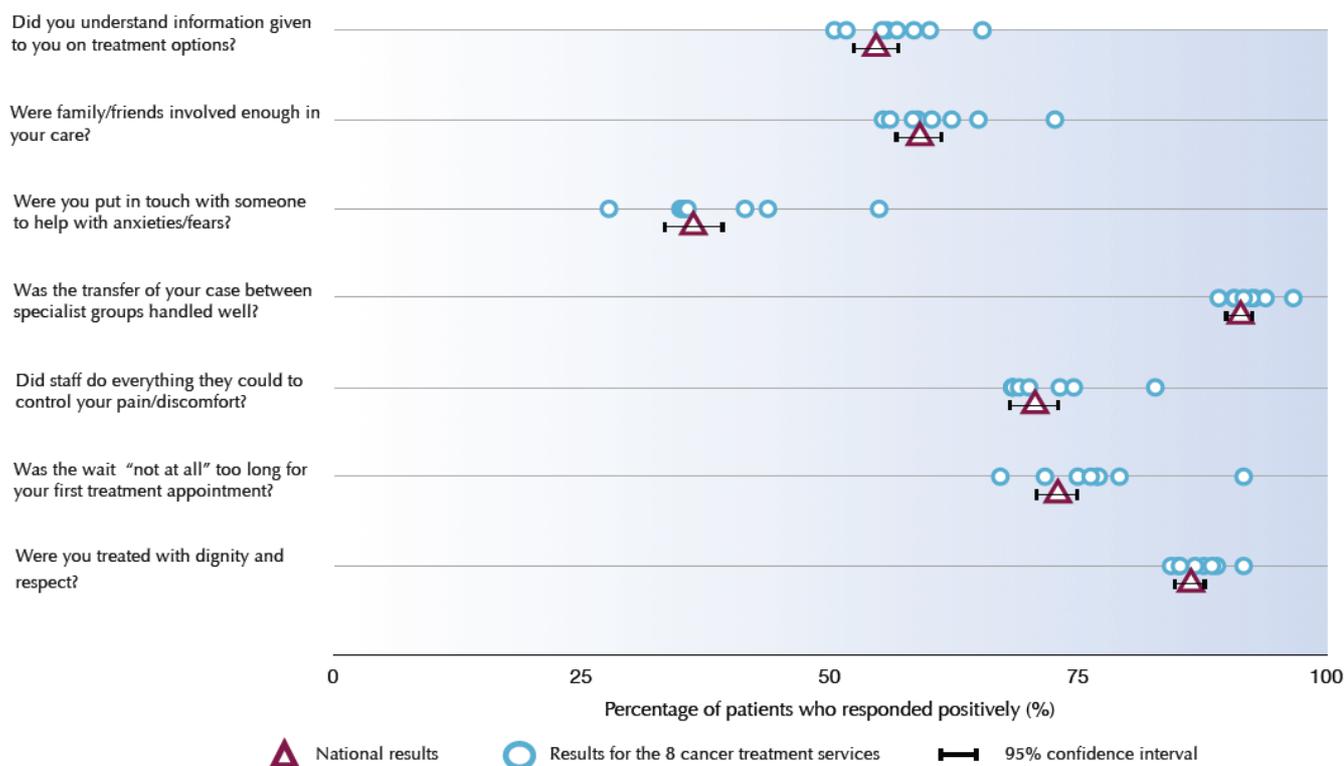
‡ For the sample and eligible respondent populations, gender was imputed from the person's title or name, as gender information was not included in the cancer treatment services' lists. Target population proportions for gender were estimated using sample proportions.

Figure 1 presents the range of national and individual treatment service percent positive score results for a subset of survey questions. The national score is shown

alongside the participating cancer treatment service scores for comparison purposes. Overall, the survey questions tended to have a high percentage of positive responses.

In Figure 1, the questions with the highest percentage of positive responses were related to specialist care coordination (91% positive response; 95% CI: 90–93) and the level of dignity and respect provided by healthcare professionals (86% positive response; 95% CI: 85–88). Whereas, the questions on information clarity (54% positive response; 95% CI: 52–57) and provision of referrals for emotional support (36% positive response; 95% CI: 33–39) received a lower percentage of positive responses.

Figure 1. National responses to a selection of key survey questions



For most survey questions, the individual treatment service results were tightly clustered around the national-level result (Figure 1). However, as shown in Table 2, 12 survey questions demonstrated at least a 20% difference between the lowest and highest treatment service scores, indicating some significant differences in patient experiences between treatment services.

The key national strengths and opportunities for improvement are shown in Tables 3 and 4. These were identified from examining the upper and lower quartiles of the ranked survey questions. The aspects of care with a high percentage of positive responses were identified as strengths (Table 3).

Table 2. Aspects of care with substantial differences in positive scores between cancer treatment services

Survey question response indicating positive outcome	Highest cancer treatment service % positive score (CI)	Lowest cancer treatment service % positive score (CI)
Never waited longer than expected for first treatment appointment	92 (82–96)	67 (62–72)
Travel concerns definitely considered in treatment planning	71 (54–83)	43 (37–50)
Waited less than 30 minutes for scheduled chemotherapy treatment appointment	88 (82–93)	62 (54–69)
Never waited longer than expected for chemotherapy treatment	85 (74–91)	51 (43–58)
Staff always did everything they could to make the wait for chemotherapy comfortable	88* (69–96)	63 (53–72)
Never waited longer than expected for radiation therapy	63 (56–68)	36 (30–42)
Staff always did everything they could to make the wait for radiation therapy comfortable	96 (75–99.6)	53* (20–84)
Someone always told the patient how to manage any side effects of radiation therapy	95 (82–99)	71 (61–80)
Always offered counseling or support relating to issues such as concerns about cancer or coping at home / work	57 (42–71)	36 (31–42)
Over the past 12 months, someone at the hospital definitely put the patient in touch with other doctors, nurses or healthcare professionals who could help with anxieties and fears, if it was needed	55* (40–70)	28 (22–34)
Always got as much help as was wanted in figuring out how to pay for any extra costs related to cancer care	79 (65–89)	56 (46–65)
Availability of parking good, very good or excellent	84 (80–88)	38 (27–51)

CI=confidence interval; * For these confidence intervals the sample sizes in the strata were very small, so strata were collapsed together to calculate the confidence intervals. The confidence intervals here are very wide indicating that the point estimate is not a reliable estimate of the 'true' value.

Table 3. National strengths of outpatient cancer care, as indicated by aspects of care receiving the highest percentage of positive responses in the survey

Aspects of care	National % positive response (CI)	Comments
Coordination of specialist care	91 (90–93)	Many of the surveyed patients reported they had visited multiple doctors, with nine out of ten people (91%) indicating that the specialist care co-ordination was good, very good or excellent.
Ease of understanding directions/signs	90 (89–92)	
Noise control at the cancer treatment services	90 (88–91)	
Level of privacy provided	87 (85–89)	
Dignity and respect provided	86 (85–88)	
Care providers doing everything they could to treat cancer	83 (81–85)	8 out of 10 people (83%) reported that they felt doctors, nurses and other healthcare professionals did everything they could to treat their cancer. A similar proportion (81%) reported they would recommend their healthcare team to family and friends.

The aspects of care with a low percentage of positive responses were identified as opportunities for improvement (Table 4).

Table 4. National opportunities for improvement in outpatient cancer care, as indicated by aspects of care receiving the lowest percentage of positive responses in the survey

Aspects of care	National % positive response (CI)	Comments
Information provided on changes in relationships (when needed)	32 (29–35)	Less than half of those surveyed reported always getting enough information on these aspects of daily living.
Information provided on changes in sexual activity (when needed)	31 (28–34)	
Explanations provided for any treatment waiting times	34 (30–37)	Seven out of 10 people reported that they did not feel it was adequately explained why they had to wait for their first cancer treatment appointment. However, only three out of ten people (27%) reported that they felt that they waited too long to get their first cancer treatment appointment.
Being put in touch with care providers to help with anxiety and fear (if this was required in the 12 months post diagnosis)	36 (33–39)	More people, about half of those who had anxieties and fears (47%), felt that they were put in touch with other healthcare professionals who could help them at the time of their initial diagnosis compared with post diagnosis, when only four out of 10 (36%) reported being provided satisfactory emotional support.
Being put in touch with care providers to help with anxiety and fear (when first told of illness)	47 (44–50)	
Information provided on changes in emotions (when needed)	39 (36–42)	Less than half of those surveyed reported always getting enough information on this aspect of daily living.
Living situation taken into account when planning for treatment	49 (46–51)	Approximately half of those sampled reported that they did not feel that healthcare providers did their best to take their family or living situation into account when planning for treatment. A similar number (48%) had travel concerns that they felt had not been adequately considered in their treatment planning.

Discussion

Most of the people who participated in the 2009 Cancer Care Survey responded very positively to questions on care coordination and the level of privacy, dignity and respect provided. The majority also felt that care providers were doing everything they could to treat their cancer. Even though overall care was rated highly, focusing on patients' experiences exposed areas where action is needed to improve the quality of care. These areas include providing more information on potential changes in aspects of day-to-day living, facilitating opportunities for emotional support, providing better explanations for waiting times, and treating patients more within the context of their own lives.

The results of this first Cancer Care Survey in New Zealand provide a baseline for future monitoring of patients' experiences of cancer care. This survey found that each cancer treatment service had different areas where it performed relatively well and where there were opportunities for improvement.

While cancer treatment service results tended to follow the same general pattern as the national-level results, responses to 12 questions demonstrated variation with at least a 20% difference in scores between the cancer treatment service with the highest positive score and the cancer treatment service with the lowest positive score.

Although this analysis found that there is no single 'best' patient experience of care model, cancer treatment services can collaborate to allow regional champions for particular aspects of care to share approaches for improving the patient experience.

Both technical and service aspects of care comprise quality service delivery just as both clinical outcome and personal experiences are important aspects of patient expectations.⁸

Results from this first Cancer Care Survey suggest that technical aspects of care tend to meet most patients' expectations while service aspects of care often do not meet patients' highest expectations. These indicate important opportunities for improvement given that service responsiveness has been posited to:

- Improve adherence by individuals to medical advice;⁹
- Lead to better compliance with cancer treatment regimens;¹⁰ and
- Improve patient health status.⁸

Because quality care is the "...cumulative result of the interactions of people, individuals, teams, organizations and systems",¹¹ multiple strategies and voices will be required to advocate for the need to optimise both the technical and service aspects of providing effective cancer care.

This was a relatively large survey, of a randomly selected population, which used a questionnaire that, although it contained minor adaptations for a New Zealand context, was based on material that had been well tested and administered internationally.^{7,12,13} A high response rate was achieved.

The study did, however, have several limitations. The strengths and opportunities for improvement sections presented in the results reflect the experiences of the majority of respondents, whose experiences may be different from those of minority groups within the sample, and those who chose not to respond to the survey. Furthermore, there is no single definition of 'best patient experience' due to differing expectations and perceptions of what is effective and appropriate cancer care delivery across individuals and population groups. Additionally, as this was a quantitative study, responses were restricted to categories provided in the survey, limiting the amount of information that could be gathered on the context of the patients' experiences.

Whilst the questionnaire has been validated in the USA by NRC+Picker, and utilised in Australia and Canada, a few of the questions may not be as applicable to the New Zealand health system. However, the authors believe that the survey utilised provides valuable insights into the perceptions of cancer management in New Zealand and that

no survey instrument can perfectly measure the complexities of patient experiences of healthcare services.

Further research could assist in addressing some of these limitations. There is a need for more in-depth exploration of Pacific and Asian cancer patient experiences as these population groups were under-represented in this survey. Further, large cancer centres, particularly Auckland, have been under-represented in this project and would benefit from follow-up research to validate the findings from this survey. In regard to the data analysis, a sensitivity analysis of the different positive response thresholds would provide useful information on the utility of the 'top box' scoring approach for reporting results.

It is recommended that the findings from this survey progress the debate around the role and meaning of patient-centred care in New Zealand. Results can also inform the development of actionable and standardised patient-reported outcomes. Further, survey findings can be linked to findings from related projects, such as the National Cancer Psychosocial Services Stocktake 2005–2006,¹⁴ to inform development of the Implementation Plan for the Supportive Care Guidance.¹⁵

Finally, a robust cancer patient needs assessment, one that is able to identify changing needs along a cancer journey, can provide a broader patient-focused perspective on quality improvement priorities.

The 2009 Cancer Care Survey data provides a consumer-perspective on the performance of cancer outpatient services. CCNZ has found that cancer patient experience surveys can achieve high response rates and generate useful consumer-reported views on cancer care delivery that could be integrated with evidence-based practice to inform quality improvement efforts.

The results of this first Cancer Care Survey in New Zealand will act as a benchmark against which the results of future cancer patient experience surveys can be compared. This will be crucial for monitoring the impact of new initiatives to improve the quality of cancer care.

Notes: This is a summary of information presented in the Voice of Experience report series available from Cancer Control New Zealand (www.cancercontrolnz.govt.nz).

Competing interests: None.

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Acknowledgements: We thank Cancer Control New Zealand and The New Zealand Population Health Charitable Trust for project funding; the 2009 Cancer Care Survey Advisory Group (Dr Diana Sarfati, Associate Professor Barry Borman, Professor Neil

Pearce, Professor Tony Blakely, Dr Andrew Simpson, Ms Astrid Koorneef and Dr Lis Ellison-Loschmann) for survey advice; and Robert Templeton (Principal Technical Specialist) and Roy Costilla (Advisor-Statistics) at the Ministry of Health for statistical advice. Lastly, we gratefully acknowledge the willingness of people affected by cancer to share their views and experiences of health services they received.

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References:

1. Minister of Health. The New Zealand Health Strategy. Wellington: Ministry of Health; 2000.
2. Minister of Health. The New Zealand Cancer Control Strategy. Wellington: Ministry of Health and the New Zealand Cancer Control Trust; 2003.
3. Ministerial Review Group. Meeting the Challenge: enhancing sustainability and the patient and consumer experience within the current legislative framework for Health and Disability Services in New Zealand. Report of the Ministerial Review Group. Wellington; 2009.
4. Cancer Control Council of New Zealand. The Voice of Experience, Part One National Report: Preliminary results from the 2009 Cancer Care Survey. Wellington: Cancer Control Council of New Zealand; 2009.
5. Cancer Control Council of New Zealand. The Voice of Experience companion report: New Zealand Cancer Care Survey results for eight treatment services. Wellington: Cancer Control Council of New Zealand; 2010.
6. Cancer Control New Zealand. The Voice of Experience, Part Two: Themes and results of New Zealand's first Cancer Care Survey. Wellington: Cancer Control New Zealand; 2010.
7. National Research Corporation. Development and validation of the Picker Ambulatory Oncology Survey Instrument. Nebraska, USA: NRC+Picker: 2005, p8.
8. Bredart A, Bottomley A, Blazeby JM, et al. An international prospective study of the EORTC cancer in-patient satisfaction with care measure (EORTC IN-PATSAT32). *Eur J Cancer*. 2005;41:2120-2131.
9. Clemes M, Ozanne L, Laurensen W. Patient's perceptions of service quality dimensions: an empirical examination of health care in New Zealand. *Health Marketing Quart*. 2001;19(1):3-22.
10. Borrás J, Sanchez-Hernandez A, Navarro M, et al. Compliance, satisfaction, and quality of life of patients with colorectal cancer receiving home chemotherapy or outpatient treatment: a randomised controlled trial. *BMJ*. 2001;322:826-831.
11. Minister of Health. Improving Quality (IQ): A systems approach for the New Zealand health and disability sector. Wellington: Ministry of Health; 2003.
12. Cancer Institute NSW. New South Wales Cancer Patient Satisfaction Survey 2008. Sydney, Australia: Cancer Institute NSW; July 2009.
13. Watson DE, Mooney D, Peterson S. Patient experiences with ambulatory cancer care in British Columbia, 2005/06. Vancouver, Canada: UBC Health Services and Policy Research; March 2007.
14. Surgenor L, Costello H, McKello M, Duke J. A report to the Ministry of Health on the national cancer psychosocial services stocktake 2005-2006 [unpublished]; 2006.
15. Ministry of Health. Guidance for improving supportive care for adults with cancer in New Zealand. Wellington: Ministry of Health; 2010.



Predictors of physical activity and quality of life in New Zealand prostate cancer survivors undergoing androgen-deprivation therapy

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Abstract

Aims The aims of this study were to: quantify the levels and predictors of physical activity in prostate cancer survivors on androgen-deprivation therapy (ADT); gain some insight into the effect of physical activity on the quality of life of prostate cancer survivors on ADT; and compare the quality of life of prostate cancer survivors on ADT with matched controls.

Methods A sample of 84 prostate cancer survivors on ADT were recruited from a register held by the Auckland District Health Board. Participants were mailed a collection of self-report surveys probing quality of life, physical activity and determinants of physical activity.

Result Less than half the prostate cancer sample were categorised as physically active, and there was no relationship between physical activity and age, PSA levels, or time on ADT. Compared to a matched control group the sample had lower scores for global quality of life, as well as on the physical and environmental quality of life domains. Results also showed that those prostate cancer survivors classified as active had higher levels of quality of life on average than those classified as insufficiently active. Attitude towards physical activity was the dominant predictor of the intention to be physically active, while perceived behavioural control was the dominant predictor of actual behaviour.

Conclusions Our findings describe a positive relationship between physical activity and quality of life in men with prostate cancer currently undergoing ADT. However, only half the sample was physically active, indicating that physical activity interventions aimed at prostate cancer survivors are of utility. Our data suggests targeting both attitudes and factors related to the ability to perform physical activity will be fruitful approaches.

Prostate cancer (PCa) is the most common cancer affecting men in New Zealand, with the incidence rates rivalling that of breast cancer for women.¹

Many PCa survivors undergo androgen-deprivation therapy (ADT) as it slows down the progression of the disease and increases survival rates by reducing testosterone production.² This reduction in testosterone causes many side-effects. It directly results in a significant loss of muscle and bone mass and a gain in fat mass,^{2,3} which appears to be a major determinant of the significant losses in muscular strength and endurance as well as functional capacity in tasks like sit to stand, stair climbing and fast walking.⁴⁻⁶

As a result of these (and possibly other) physiological and psychological changes, PCa survivors on ADT also report significant increases in fatigue and decreases in quality of life (QOL).^{2,7,8}

Several cross-sectional studies have shown a link between physical activity and QOL in PCa survivors.^{9,10} For example, PCa survivors who were more physically active had greater health-related QOL⁹ and lower levels of fatigue¹⁰ than their less-active PCa peers.

Recent reviews suggest that physical activity comprising strength, aerobic or combined strength and aerobic training can reduce fatigue as well as improve health and QOL (e.g. psychological, social and sexual), muscular strength and aerobic fitness in PCa survivors, with the magnitude of most of these changes of clinical significance.^{11,12}

It has also been reported that physical activity can reduce PSA levels, meaning that the PCa survivors could avoid or delay conventional treatments such as ADT for a period of two years.^{13,14}

The American Cancer Society recommends that cancer survivors would improve their overall health if they were to perform ≥ 150 minutes of moderate-strenuous intensity physical activity per week, of which ≥ 60 minutes should be strenuous.¹⁵

A recent review of the literature, however, suggests that many PCa survivors are insufficiently active, with some of the reviewed studies reporting physical activity rates of only 29–30%.¹⁶ In order to increase the physical activity levels to generate health benefits, the determinants of physical activity in PCa survivors on ADT need to be understood. Currently little information is available on the physical activity patterns of PCa survivors on ADT, particularly within New Zealand.

This exploratory cross-sectional study will collect this information and will also utilise the Theory of Planned Behaviour (TPB) model^{17, 18} to assess PCa survivors' attitudes about physical activity, subjective norm and self-efficacy. Finally, measures of health quality of life will be taken using the New Zealand version of the World Health Organization WHOQOL-BREF³⁵ questionnaire; with this QOL data compared to those of an age-matched sample from the general population.

Research design and methods

This cross-sectional survey-based study involved a convenience sample recruited using the PCa survivor register held by the Auckland District Health Board's (ADHB) Urology Department. Initially, a cover letter was sent out explaining the study and how they could participate. One week later, a series of questionnaires, an information sheet, and a stamped return-addressed envelope were mailed to 205 potential participants currently diagnosed with PCa and undergoing ADT.

Two weeks following the initial distribution of surveys, another letter was dispatched thanking those who had responded and encouraging those who had not returned the questionnaires to do so. Concurrently, normative data were collected from age- and gender-matched individuals to afford comparison with a healthy sample.

Participants—From the initial 205 questionnaires posted to potential participants, 84 replies were received, yielding a 41% response rate. The mean age of participants was 78.4 years (SD=8.21), and 70 (84%) identified themselves as New Zealand European. Fifty-two participants reported recent PSA levels (M=9.94 ng/mL, SD=22.76, Min=0.05, Max=130), and the mean time elapsed since undertaking ADT was 3.9 years (SD=3.6).

A second convenience sample from 26 organisations, including senior citizens clubs and retirement villages, was undertaken to provide comparative QOL data. From these organisations, 362 valid surveys were completed and returned, 82 of which provided a balanced sample of age, gender and ethnically matched but healthy individuals.

Measures—A self-report questionnaire asking about QOL, physical activity, and factors related to physical activity was utilised. QOL was assessed using the brief version of the World Health Organization's Quality of Life (WHOQOL-BREF) scale.¹⁹ This scale consists of physical (7 items), psychological (6 items), social (3 items), and environmental (8 items) domains, and two general items probing global quality of life and self-assessed health.

Physical activity was gauged using the Rapid Assessment of Physical Activity Scale (RAPA), a nine-item scale (each question requiring a Yes/No response) designed to assess levels of physical activity among adults older than 50 years.²⁰

The nine questions of the RAPA cover a range of physical activity levels, from sedentary to active, as well as strength training and flexibility. The responses to the nine items allows the RAPA to classify participants into one of five activity groups:

- Sedentary—"I rarely or never do any physical activities;"
- Under-active—"I do some light or moderate physical activities, but not every week;"
- Under-active regular—light activities—"I do some light physical activity every week;"
- Under-active regular—"I do moderate physical activities every week, but less than 30 minutes a day or five days a week" or "I do vigorous physical activities every week, but less than 20 minutes a day or three days a week,"; and
- Active—"I do 30 minutes or more of moderate physical activities, five or more days a week." The first four groups are then collapsed to produce an "insufficiently active" category, while the fifth is the "active" category.

Factors influencing physical activity were assessed using a pre-existing 47-item inventory probing intention to be physically active, perceived control of factors that prevent or encourage physical activity, attitudes towards physical activity, and pressures from significant others to be physically active or not.^{17, 18} Additionally, demographic items elicited information about the participant's age and ethnicity, their time on ADT, length of PCa diagnosis, and if known, the most recently assessed PSA levels.

Statistical analyses—Data analyses were conducted in SPSS v17 software. Because of the modest sample size (n=84), a thorough screen was undertaken to ensure that the data adhered to the assumptions stipulated by the respective tests, and nonparametric alternatives were employed when assumptions were violated. Prior to constructing composite measures, item mean and standard deviations were calculated to identify any floor or ceiling effects, corrected item-total correlations were scrutinised to ensure the unidimensionality of item sets, and then Cronbach's alpha computed to assess internal consistency.

Group differences in QOL domains were assessed using independent samples t-tests or Mann Whitney U-Tests. The Theory of Planned Behaviour (TPB) was applied to investigate the relationship between behavioural intention and a linear combination of the following composite variables: attitudes to physical activity, pressure from others to partake (or not) in physical activity (subjective norm), and perceived behavioural control (PBC) in relation to undertaking physical activity. In its standard form, the TPB is represented by the following equality:

$$\text{Behavioural Intention} = (W_1)\text{ATTITUDE} + (W_2)\text{SUBJECTIVE NORM} + (W_3)\text{PBC}$$

where W_i are empirically derived weights. The TPB was assessed using a multiple linear regression analysis, with standardised beta coefficients (β) examined to gauge the predictive utility of each component. The association between the three components of the TPB and actual physical activity as defined by the RAPA (i.e. active vs. insufficiently active) was determined using a binary logistic regression.

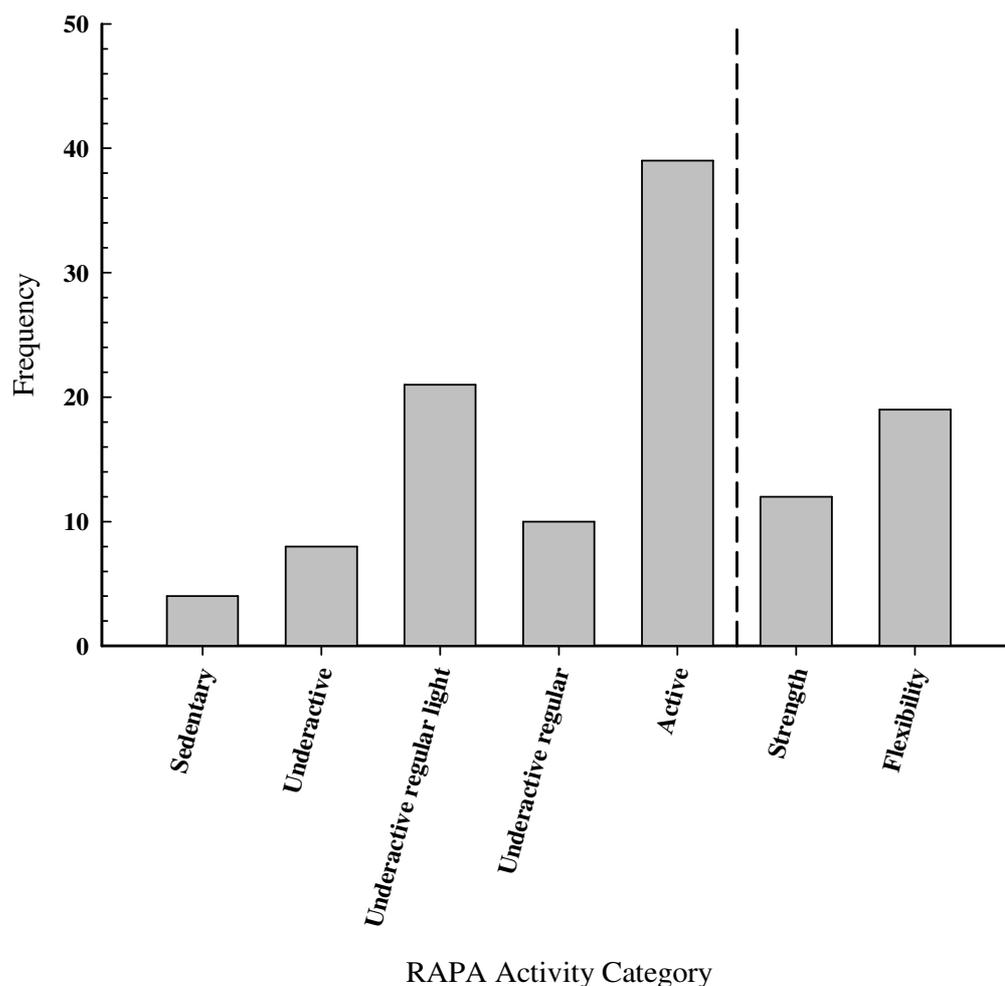
In both regression analyses covariates were not included due to sample size limitations. However, the lack of correlation between potential covariates (e.g. age, time on ADT, PSA level) and the components of the TPB meant any exclusion was likely only to reduce the overall predictive accuracy of the models rather than introduce specification errors.

Results

Levels of physical activity—Physical activity levels, shown in Figure 1, indicate that 45% of participants ($n=38$) lead an active lifestyle, while the remainder ($n=46$) were insufficiently active. Correlation coefficients (r) indicated a lack of association between physical activity level and either age ($r=0.106$, $p=0.343$), time since diagnosis ($r=0.141$, $p=0.252$), time on ADT ($r=-0.081$, $p=0.470$), or PSA level ($r=0.118$, $p=0.382$).

Twelve participants (14%) responded yes to the question “I do activities to increase muscle strength, such as lifting weights or callisthenics, once a week or more.”, and 19 participants (23%) responded yes to “I do activities to improve flexibility, such as stretching or yoga, once a week or more.”

Figure 1. The left portion of the figure shows the number of individuals falling into the five physical activity categories specified by the RAPA scale. The right portion shows those undertaking activities improving strength or flexibility



Differences in QOL—PCa survivors had significantly lower mean physical ($p<0.001$) and environmental ($p=0.025$) QOL scores than the matched sample. Furthermore, mean ratings of global QOL ($p<0.001$) and self-assessed health status ($p<0.001$) were also lower than the matched sample.

When PCa survivors were sorted into active and insufficiently active groups, the active group had higher mean physical QOL ($p=0.034$), global QOL ($p=0.023$), and self-assessed health ($p=0.037$). An additional series of Mann-Whitney U-tests were undertaken to assess group differences between those who reported engaging in strength training ($n=12$) and those who did not ($n=72$), and those engaging in flexibility activities ($n=19$) and those who were not ($n=65$).

Those undertaking some form of strength training had significantly higher global ($p=0.039$) and environmental ($p=0.023$) QOL than those not reporting such activities. Those partaking in activities designed to improve flexibility had significantly higher global ($p=0.006$), physical ($p=0.018$) and environmental ($p=0.008$) QOL. Note that, for any of the difference tests performed between groups, there were no significant differences recorded between mean QOL scores on the psychological or social domains ($p>0.05$).

Predictors of physical activity—Table 1 shows the results from a multiple linear regression, which indicates the association between the intention to exercise and the three components of the Theory of Planned Behaviour. Of remark in Table 1 is the strong link between attitudes towards physical activity and the intention to be physically active ($p<0.001$). Societal and peer pressures (i.e. subjective norm), and perceptions of efficacy to undertake activity (i.e. PBC), do not appear to be significantly associated with the intention to partake in physical activity.

Table 1. Estimates of unstandardised and standardised coefficients for a multiple linear regression of intention to partake in physical activity

Variables	<i>B</i>	Std Error	β	<i>t</i>
Constant	-1.385	2.149	–	-0.645($p=0.521$)
Attitude	0.322	0.047	0.781	6.814 ($p<0.001$)
Subjective Norm	0.075	0.063	0.115	1.179 ($p=0.242$)
PBC	-.142	0.142	-.113	-0.999 ($p=0.321$)

Note: $R=0.775$; $R^2=0.601$, $adj-R^2=0.585$; $SE_{est}=4.893$

Predictors of actual physical activity—Table 2 displays the results of a binary logistic regression, where positive values of the regression coefficient *B* indicate that the predicted odds increase as the predictor value increases (i.e. more likely to be in the physically active group). Scrutiny of the Wald chi-square statistics in Table 2 indicates that attitude and subjective norm are not significant predictors of physical activity category.

The PBC component of the TPB emerges as a significant predictor of physical activity ($p=0.015$), indicating that the odds of being in the active group is positively related to PBC. Goodness-of-fit tests (Hosmer-Lemeshow) suggested an adequate fit

to the data. The improvement of the model displayed in Table 2 over a baseline model (i.e. one containing none of the predictors) is evident in the classification table displayed in Table 3, which indicates the agreement between predicted and actual outcomes.

Table 2. Logistic regression analysis of prostate cancer survivor’s activity levels. The table displays maximum likelihood parameter estimates, both in raw form as logits (i.e. B) and as odds ratios (e^b), accompanied by 95% confidence intervals

Variables	B	Std Error B	Wald χ^2	e^b	95% CI for e^b
Constant	-4.796	1.34	0.02 ($p<.001$)	0.008	–
Attitude	0.003	0.022	0.02 ($p=0.886$)	0.997	0.955–1.041
Subjective Norm	0.038	0.031	1.445 ($p=0.228$)	1.038	0.977–1.104
PBC	0.178	0.073	5.881 ($p=0.015$)	1.196	1.035–1.391

Note: Cox & Snell $R^2=0.242$. Nagelkerke $R^2=0.323$. Hosmer and Lemeshow test: $\chi^2(8)=9.41$, $p=0.309$.

Table 3. Observed and predicted frequencies for physical activity/inactivity by logistic regression with a cut-off of 0.50. Parentheses contain the predictions from the baseline (i.e. intercept-only) model

Observed	Predicted		% Correct
	Active	Inactive	
Active	23 (0)	14 (37)	62.2 (100)
Inactive	6 (0)	39 (45)	86.7 (0)
Overall % correct:			75.6 (54.9)

Note: Sensitivity = 62.1%. Specificity = 86.6%. False positive = 20.1%. False Negative = 26.4%.

Barriers to physical activity—Responses to open-ended questions provided insight into additional preventative and supportive factors influencing participants’ physical activity. Of the preventative factors, lack of energy and health problems additional to PCa were the most common factors that prevented participants from being physically active, as well as weather and interference by paid employment. The most common factors that supported participants in being physically active were the health and mental health benefits, and the enjoyment of participating in activities that had physical components to them (e.g. gardening and household tasks).

Discussion

The majority of PCa survivors in our sample were classified as insufficiently active, with just 45% meeting the recommended guidelines set out by the American Cancer Society of at least 30 minutes of moderate intensity physical activity on five or more days per week.¹⁵

These results appears to lie within the extremes reported in a recent review of the physical activity levels of PCa survivors.¹⁶ Additionally, we found no relationship

between physical activity level and time since diagnosis, PSA levels, or time on ADT. This finding has consequences in relation to timing of interventions based on physical activity, and suggests no critical time window exists immediately after diagnosis or ADT onset.

Beyond the direct physical changes, ADT has also been shown to cause very substantial reductions in QOL for PCa survivors.⁸ In our sample we found PCa survivors had significantly lower physical and environmental QOL when compared to age-matched healthy individuals. Whether this constitutes evidence that the cancer or the cancer treatment may be affecting the physical domain is unclear, as 64% of respondents also reported other conditions. However, it can be argued that either a single disease or a combination of diseases would have the potential to degrade physical QOL.

Additionally, PCa survivors also had significantly lower global QOL and self-assessed health than the matched sample, echoing previous research.^{7,8} Physical activity has positive benefits on QOL,^{9,10} and a comparison between PCa survivors classified as active and those classified as insufficiently active supports this relationship.

On average those PCa survivors classified as active had significantly higher global and physical QOL, and higher self-rated health than insufficiently active survivors, suggesting that efforts to maintain or increase physical activity levels in this group are a worthwhile objective. Furthermore, 12 (14%) participants reported regularly partaking in strength training. This finding concurs with a recent review that suggests prevalence rates of 10–15% for strength training in older adults.²¹

While there are likely many factors contributing to the low rates of strength training in older adults (including PCa survivors), Winnett et al.²¹ argue that the primary factors may be public health policy not emphasising strength training, misinformation, and the lack of theoretically driven approaches to maintain adherence in the long-term. Such views appear somewhat consistent with the predictors of physical activity found in this study along with studies on the factors associated with the use of complementary therapies by cancer survivors.^{22–24}

These studies indicate that the misinformation regarding exercise often comes from potentially unreliable sources such as family, friends and the media,²² even though exercise counselling²³ and the support of clinicians may play an important role in cancer survivors adopting and maintaining healthy behaviours such as exercise.²⁴

In light of the way that cancer survivors obtain information about the benefits of physical activity and other complementary therapies and our findings indicating that most PCa survivors are insufficiently active, considerably more effort needs to be focused on ensuring that a greater proportion of PCa survivors especially those on ADT are physically active. Such results would suggest that clinicians working with PCa survivor are in a unique place to offer such advice.

Intention to engage in physical activity is driven by a number of factors, including attitude, subjective norms and PBC. Two studies of PCa survivors using the TPB^{25,26} report that these three factors explain a high percentage of the variance in physical activity intention.

Consistent with these previous studies,^{25,26} the best predictor of physical activity in the present dataset was the PBC. The PBC component combines the notions of perceived control and self-efficacy in relation to a behaviour, where perceived control is an assessment of external constraints and self-efficacy the belief that one has the ability to perform certain behaviours.

Our participants identified a range of factors that both prevented and supported them in being physically active, with health problems and disability being the highest preventative factor. Additionally, lack of energy was the most highly reported factor that prevented participants from engaging in physical activity. This was expected, as fatigue is a debilitating side-effect of both cancer and cancer treatment, including ADT.^{2,10}

Such a result is a sort of vicious cycle, whereby if a PCa survivor is tired, they won't exercise, and if they don't exercise, they will be more tired. However, as increasing physical activity can actually reduce fatigue in PCa survivors,²⁷ this further demonstrates the importance of exercise counselling for these individuals.

Limitations and future research—Small convenience samples increase the probability of type I errors by preventing the use of more sophisticated multivariate techniques, and also invite type II errors by providing less than satisfactory power. However, while the findings we report here may be considered somewhat speculative and need to be confirmed with a larger New Zealand sample, they are congruent with findings reported overseas.^{7,16,26}

A second limitation is the use of self-report inventories, with participants potentially overstating their levels of physical activity and under-reporting sedentary behaviours due to social desirability bias.²⁸ Furthermore, while the PCa group differed to the matched group on the basis of the cancer diagnosis and use of ADT, no attempt was made to match the groups on the basis of self-reported health or comorbidities.

It is therefore not entirely clear how the cancer, use of ADT, health status or comorbidities contributed to the findings of this study. Future research may address this question by comparing various sub-groups of PCa survivors to determine the effect of treatment type, health status or comorbidities on physical activity, QOL and their inter-relationships. Randomised controlled trials should also be conducted to examine the effect that exercise counselling has on the adoption and maintenance of physical activity levels in PCa survivors, and how these potential changes in physical activity may be associated with improved QOL and reduced fatigue.

Competing interests: None.

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Acknowledgements: We thank the Cancer Society of New Zealand and the Faculty of Health and Environmental Sciences, AUT University for funding this project; Professor Robert Newton for providing expertise and assistance in the initial design of this study; and all of the prostate cancer survivors who gave up their time to participate in this research.

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References:

1. Ministry of Health. 2010. Cancer: New registrations and deaths 2006. <http://www.moh.govt.nz/moh.nsf/indexmh/cancer-reg-deaths-2006>
2. Saylor PJ, Keating NL, Smith MR. Prostate cancer survivorship: Prevention and treatment of the adverse effects of androgen deprivation therapy. *J Gen Intern Med.* 2009;24:389–94.
3. Galvao DA, Spry NA, Taaffe DR, et al. Changes in muscle, fat and bone mass after 36 weeks of maximal androgen blockade for prostate cancer. *BJU Int.* 2008;102:44–7.
4. Galvão DA, Nosaka K, Taaffe DR, et al. Resistance training and reduction of treatment side effects in prostate cancer patients. *Med Sci Sport Exercise.* 2006;38:2045–52.
5. Clay CA, Perera S, Wagner JM, et al. Physical function in men with prostate cancer on androgen deprivation therapy. *Phys Ther.* 2007;87:1325–33.
6. Galvão D, Taaffe D, Spry N, et al. Reduced muscle strength and functional performance in men with prostate cancer undergoing androgen suppression: A comprehensive cross-sectional investigation. *Prostate Cancer Prostatic Dis.* 2009;12:198–203.
7. Spry NA, Kristjanson L, Hooton B, et al. Adverse effects to quality of life arising from treatment can recover with intermittent androgen suppression in men with prostate cancer. *Eur J Cancer.* 2006;42:1083–92.
8. Katz A. Quality of life for men with prostate cancer. *Cancer Nurs.* 2007;30:302–8.
9. Daubenmier JJ, Weidner G, Marlin R, et al. Lifestyle and health-related quality of life of men with prostate cancer managed with active surveillance. *Urology.* 2006;67:125–30.
10. Schwartz AL. Patterns of exercise and fatigue in physically active cancer survivors. *Oncol Nurs Forum.* 1998;25:485–91.
11. Antonelli J, Freedland SJ, Jones LW. Exercise therapy across the prostate cancer continuum. *Prostate Cancer Prostatic Dis.* 2009;12:110–5.
12. Newton R, Galvão D. Exercise in prevention and management of cancer. *Curr Treat Options Oncol.* 2008;9:135–46.
13. Frattaroli J, Weidner G, Kemp C, et al. Clinical events in prostate cancer lifestyle trial: Results from two years of follow-up. *Urology.* 2008;72:1319–23.
14. Ornish D, Weidner G, Fair WR, et al. Intensive lifestyle changes may affect the progression of prostate cancer. *J Urol.* 2005;174:1065–70.
15. Doyle C, Kushi LH, Byers T, et al. Nutrition and physical activity during and after cancer treatment: An American Cancer Society guide for informed choices. *CA Cancer J Clin.* 2006;56:323–53.
16. Thorsen L, Courneya K, Stevinson C, et al. A systematic review of physical activity in prostate cancer survivors: Outcomes, prevalence, and determinants. *Support Care Cancer.* 2008;16:987–97.
17. Ajzen I. The theory of planned behavior. *Organ Behav Hum Decis Process.* 1991;50:179–211.
18. Ajzen I. 2006. Constructing a TPB questionnaire: Conceptual and methodological considerations. <http://www.people.umass.edu/aizen/pdf/tpb.measurement.pdf>
19. World Health Organization Quality of Life Group. 1998. Development of the World Health Organization WHOQOL-Bref quality of life assessment. http://depts.washington.edu/yqol/docs/WHOQOL_Bibliography.pdf

20. Topolski TD, LoGerfo J, Patrick DL, et al. The rapid assessment of physical activity (RAPA) among older adults. *Prev Chronic Dis.* 2006;3:A118.
21. Winett RA, Williams DM, Davy BM. Initiating and maintaining resistance training in older adults: A social cognitive theory-based approach. *Br J Sports Med.* 2009;43:114–9.
22. Pud D, Kaner E, Morag A, et al. Use of complementary and alternative medicine among cancer patients in Israel. *Eur J Oncol Nurs.* 2005;9:124–30.
23. Dorsay JP, Cheifetz O. Cancer and exercise: A survey of patients' knowledge and preferences. *Arch Phys Med Rehabil.* 2008;89:e27.
24. Roberts CS, Baker F, Hann D, et al. Patient-physician communication regarding use of complementary therapies during cancer treatment. *J Psychosoc Oncol.* 2005;23:35–60.
25. Blanchard CM, Courneya KS, Rodgers WM, et al. Determinants of exercise intention and behavior in survivors of breast and prostate cancer: An application of the theory of planned behavior. *Cancer Nurs.* 2002;25:88–95.
26. Hunt-Shanks TT, Blanchard CM, Baker F, et al. Exercise use as complementary therapy among breast and prostate cancer survivors receiving active treatment: Examination of exercise intention. *Integr Cancer Ther.* 2006;5:109–16.
27. Monga U, Garber SL, Thornby J, et al. Exercise prevents fatigue and improves quality of life in prostate cancer patients undergoing radiotherapy. *Arch Phys Med Rehabil.* 2007;88:1416–22.
28. Stewart AL, Mills KM, King AC, et al. CHAMPS physical activity questionnaire for older adults: Outcomes for interventions. *Med Sci Sports Exerc.* 2001;33:1126–41.



Robot-assisted laparoscopic prostatectomy: a 2010 update

James B Duthie, Joanna E Pickford, Peter J Gilling

Abstract

Aim To build on the previous article and further explore the safety and efficacy of robotic-assisted laparoscopic prostatectomy (RALP) in the first 100 cases from a single institution in New Zealand.

Method A prospective database was created to monitor perioperative and postoperative outcomes of men undergoing RALP for clinically localised carcinoma of the prostate.

Results The first 100 cases were followed prospectively with a mean follow-up of 13.9 months. There were no conversions to open surgery, or re-operations. Average blood loss was 281 ml, and there was only one blood transfusion. Mean hospital stay was 1.1 nights. Mean console time improved from 251.4 minutes over the first 10 cases to 104.6 minutes over the last 10. The overall positive margin rate was 18%. The positive margin rate from pT2 tumours was 8%. The majority of patients had well-differentiated, organ-confined disease. Postoperatively, five have a detectable PSA level. 68% use no incontinence pads at 12 months. At one year, 12% of the men who were previously fully potent have achieved full potency again without assistance

Conclusion The results further support RALP as a safe, effective, and well tolerated procedure for the management of carcinoma of the prostate. The early local experience compares favourably with other published early series.

Robotic-Assisted Laparoscopic Prostatectomy (RALP) is increasingly popular as the mode of surgical management of early prostate cancer worldwide. Shorter hospital stay and earlier return to activities make RALP attractive as a treatment option. This paper reports on the first 100 RALP cases performed at Tauranga, New Zealand, adding more cases and longer follow-up to previously published data on the first 30 cases.¹

RALP is now the most common modality of radical treatment for organ-confined prostate cancer in the US, with 80% of cases this year expected to be treated with RALP. The first RALP was performed on 3 September 2007 at Grace Hospital in Tauranga. The da Vinci surgical system (Intuitive Surgical, Sunnyvale, CA, USA) is a master-slave tele-manipulative device controlled by a surgeon sitting at a console, controlling a laparoscopic camera and three operative arms through the use of two hand controls (Figure 1). RALP is at present an uncommon but increasing treatment in New Zealand.

The previously published data reported a single surgeon experience with his first 30 cases.¹ The data compared favourably with published international data on learning curves for RALP in terms of intra-operative complications, cancer clearance, analgesia requirements, and time to discharge. The present study provides additional data up to the first 100 cases, with longer follow up.

Methods

Prospective data were obtained on the first 100 consecutive patients undergoing RALP by a single surgeon (PJG) at our institution. Preoperative data—including presenting prostate-specific antigen (PSA), clinical stage, and biopsy histology—were obtained, as well as erectile function questionnaires (Sexual Health Inventory Male score) with Normal being a score >21, Quality of Life (QOL score), and urinary function including daily pad usage. Perioperative data including total theatre and actual robot (console) operating time, blood loss, surgical technique, postoperative analgesia requirements, length of hospital stay, and surgical complications were collected. Serial PSAs, erectile function, continence, and quality of life continue to be measured at 3, 6, 12, and 24 months. The surgical technique was subtly modified and developed over the series, but remains largely unchanged from the description in the previous paper. The technique is based on that pioneered by Menon and modified by Patel.^{2,3}

Results

The cases were performed between September 2007 and December 2009. Patient characteristics are described in Table 1. Follow-up data ranges from 3 to 24 months, with the mean being 13.9 months. All but one patient had clinically organ-confined disease (T1 or T2). A single patient clinically had T3 disease and was treated as part of a multi-modal protocol. Transrectal biopsy showed moderately well differentiated cancer in nearly all patients (from Gleason Grade 3+3 to 4+3); however, five patients had a secondary Gleason grade of 5.

Operative time—Operative times are described in Table 1. The mean total theatre time for the first 10 cases was 341.5 minutes, with a mean console time of 251.4 minutes. The last 10 cases had a total theatre time of 177.3 minutes, with a mean console time of 104.6 minutes. Average blood loss was 281 ml (range <50 ml–2000 ml).

Postoperative—The mean hospital stay was 1.1 nights, 29 patients only stayed overnight. Twenty-one patients required only paracetamol for analgesia; most required only a modest dose of tramadol (average 163 mg) or non-steroidal anti-inflammatory in addition to this. Seven patients required IV morphine, with an average of 7 mg used.

Perioperative morbidity—Three patients had more than 1 L estimated blood loss, of these one lost approximately 2 L. One patient (1%) required a blood transfusion. Five patients had hospital stays of 3 nights, and four exceeded 3 nights; one due to an acute anxiety episode, and one with significant bladder spasm and penile tip pain. One patient developed a urinoma due to vesico-urethral anastomotic leak, which was managed conservatively with an extended period of catheterisation. One patient experienced a postoperative ileus.

Table 1. Average operating times

Quartile	Total Theatre Time	Console Time
First 25 patients	273	206
Second 25 patients	238	166
Third 25 patients	192	120
Fourth 25 patients	190	121

Figure 1. The robotic theatre



Pathology—There was generally good agreement between TRUS biopsy grading and final pathology. The overall positive margin rate was 18%, but only eight patients with positive surgical margins in the absence of extracapsular spread (pT2) on final pathology. Only one of these was in the last 25 patients, and this patient had less than 1 mm involved at the surgical margin.

Functional outcomes—The mean preoperative SHIM score was measured in 87 patients, and was found to have a mean of 19.9. Only 47 were fully potent (SHIM score >21) at baseline. Figure 2 shows SHIM scores increasing with time postoperatively, but at present only 66 patients have been followed up to 12 months. The mean score of these patients is 8.7. Of the fully potent men, 38 have been followed up at 1 year and 12% are fully potent without medical assistance. Of the 20 patients followed up to 24 months, the overall mean is 9.3. Mean AUA score at 12 months is 4.6.

The mean preoperative QOL score was 1.6, of the 69 patients followed up to 12 months the mean is again 1.6. Figure 3 shows that mean pad usage was 0.02 preoperatively, 1.8 at 3 months, and 0.5 for the 75 patients with 12 months follow up. 52 patients (68%) use no pads at 12 months.

Figure 2. SHIM score

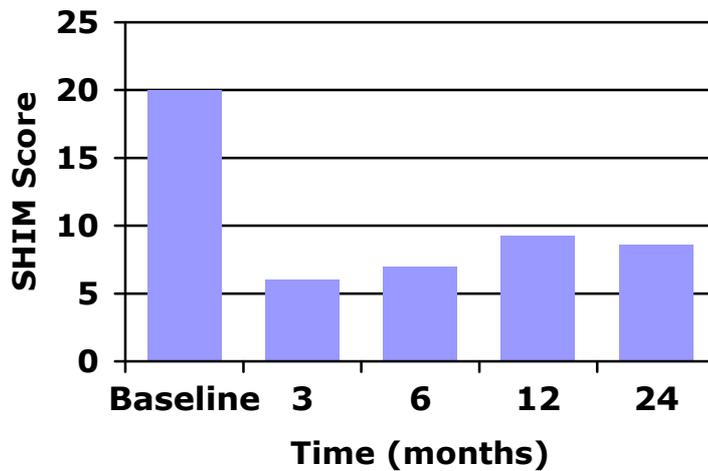
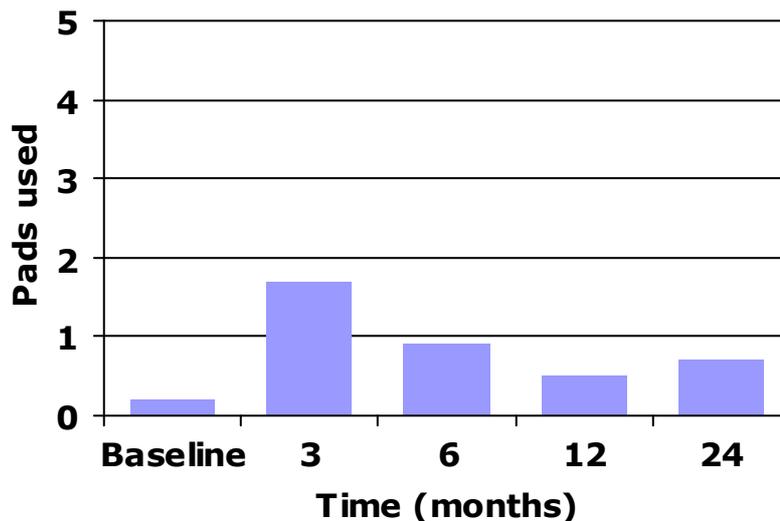


Figure 3. Number of pads used per day



PSA—Follow up PSA levels at three months have been measured in 96 of the patients, with 94.8% recording undetectable levels. Of the five patients with a detectable PSA, three had pT3 disease, and one had a PSA level that continued to drop to a level below 0.2 ng/ml at 24 months. Only 44 have a 12 month postoperative level, of these three are greater than 0.2 ng/ml.

Discussion

This series compares favourably with published international data in terms of safety and efficacy.^{4,5} A relatively short learning curve is suggested by the rapid improvement in operative time and low positive margins rates observed over the

series. That the console time reached a plateau at the third quartile further supports this theory. The hypotheses that RALP provides effective oncological outcomes with the benefits of reduced blood loss and perioperative pain are supported.

Perioperative morbidity is low, and again comparable to published series. Operative and total theatre times rapidly improved, and are by the end of the series comparable to conventional radical retropubic prostatectomy in many centres, and superior to laparoscopic prostatectomy⁵. Both erectile function recovery and post-operative continence in this series are good, particularly given the short follow up, as continence may improve up to a year, and erectile function up to two years post-operatively.

Further analysis of preoperatively potent patients (SHIM scores >21) will be performed at the two year mark to further assess this, but early data is satisfactory. Little long term PSA data has been recorded so far, but is so far encouraging and is in line with what is expected following Open Prostatectomy in a similar group. RALP has been demonstrated to have advantages over other techniques for organ confined prostate cancer, and has a growing role in the management of prostate cancer in New Zealand.

Competing interests: None.

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References:

1. Wilson L, Pickford J, Gilling P. Robot-assisted laparoscopic radical prostatectomy (RALP)—a new surgical treatment for cancer of the prostate. *N Z Med J.* 2008;121(1287):32–38. <http://www.nzma.org.nz/journal/121-1287/3422/content.pdf>
2. Menon M, Tewari A, Peabody JO, et al. Vattikuti Institute prostatectomy, a technique of robotic radical prostatectomy for management of localized carcinoma of the prostate: experience of over 1100 cases. *Urol Clin North Am.* 2004;31(4):701–17.
3. Patel VR, Palmer KJ, Coughlin G, Samavedi S. Robot-assisted laparoscopic radical prostatectomy: perioperative outcomes of 1500 cases. *J Endourol.* 2008;22(10):2299–305.
4. Menon M, Tewari A, Baize B, et al. Prospective comparison of radical retropubic prostatectomy and robot-assisted anatomic prostatectomy: the Vattikuti Urology Institute experience. *Urology.* 2002;60(5):864–8.
5. Touijer K, Eastham JA, Secin FP, et al. Comprehensive prospective comparative analysis of outcomes between open and laparoscopic radical prostatectomy conducted in 2003 to 2005. *J Urol.* 2008;179 (5):1811–7.



Immunohistochemical testing for colon cancer—what do New Zealand surgeons know?

Simon J Harper, Alison R McEwen, Elizabeth R Dennett

Abstract

Aim 8–12% of colorectal cancers are associated with genetic syndromes. The most common of these is Lynch syndrome (also known as Hereditary Non-Polyposis Colorectal Cancer). Clinical criteria (Bethesda criteria) exist that can be used to identify colorectal cancer patients who may benefit from immunohistochemical screening of their tumour for Lynch syndrome. Treating surgeons need to know these criteria in order to request appropriate testing. The aim of this study was to assess the knowledge of New Zealand surgeons about the Bethesda criteria.

Methods We conducted a postal survey of all New Zealand General Surgical Fellows of the Royal Australasian College of Surgeons.

Results Of the surgeons returning surveys 88% knew screening using immunohistochemistry was available; 7% would not refer an abnormal result to a genetic service. Results of the practice based questions showed only 45% of respondents knew that a colorectal cancer diagnosed before the age of 50 was one of the Bethesda criteria. The correct response rates for the rest of the survey ranged from 32–96%. Questions about Lynch syndrome associated cancers returned fewest correct answers. In general, surgeons are poorly informed about cancers associated with Lynch syndrome.

Conclusion The study demonstrates limited awareness of the Bethesda criteria amongst New Zealand General Surgeons. Those treating colorectal cancer should be aware of the classic features of Lynch syndrome and test appropriately.

Cancers of the colon and rectum are the second most common cancers in New Zealand (NZ). The most recent figures released by the NZ Ministry of Health show 2716 new cases were registered in 2005¹ (66 cases / 100,000 population²). The majority of cases are sporadic but 8–12% are associated with genetic syndrome.

The most common of the known colorectal cancer (CRC) predisposing syndromes is Lynch syndrome,³ also known as hereditary non-polyposis colorectal cancer (HNPCC). This is an autosomal dominant syndrome, which confers a predisposition to colonic and other cancers. Lynch syndrome is responsible for 2–3% of the colorectal cancer burden.

Typically, families with Lynch syndrome are diagnosed with colonic cancers at an earlier age than sporadic colonic cancers and multiple tumours (either synchronous or metachronous) are more frequent than in sporadic colon cancer patients. Other tumours associated with the syndrome include endometrial carcinoma with a lifetime risk of 40–60%⁴ as well as a modestly increased risk of ovarian, stomach, small bowel, hepatobiliary, renal pelvic, and ureteric tumours.⁵ A personal or family history

of any of these malignancies in a patient with colon cancer should raise the suspicion of Lynch syndrome.

Lynch syndrome is caused by germline mutations in one of several DNA mismatch repair genes which results in faulty DNA repair. Laboratory techniques including microsatellite instability (MSI) testing and immunohistochemistry (IHC—which tests for the expression of DNA repair proteins suggesting the possibility of MSI) can help identify patients with suspected Lynch syndrome. Thus genetic counseling, genetic testing and appropriate surveillance can be offered to families. The revised Bethesda criteria⁶ (Table 1) are the currently accepted guidelines used when deciding which patients to screen for potential Lynch syndrome.

Table 1. Revised Bethesda criteria

Lynch syndrome should be suspected and MSI testing of the tumour carried out if any of the following criteria apply:	
1	Colorectal cancer diagnosed in a patient under 50 years of age
2	Presence of synchronous, metachronous colorectal cancer or other HNPCC associated tumours*, regardless of age
3	Colorectal cancer with MSI-H like histology diagnosed in a patient less than 60 years of age
4	Colorectal cancer diagnosed in a patient with one or more first degree relatives with an HNPCC associated tumour*, with one of the tumours being diagnosed under 50 years of age
5	Colorectal cancer diagnosed in a patient with two or more, first or second degree relatives with HNPCC associated tumours*, regardless of age

* HNPCC associated tumours are; endometrial; ovarian; stomach; small bowel; hepatobiliary; renal pelvis; and ureteric

Surgeons who treat patients with colorectal cancer need to be alert to the possibility of Lynch syndrome and arrange appropriate testing in response to clinical triggers. Previously this required identification of those that fit the Bethesda criteria and subsequent referral onto a genetic service. The genetic service would then take a detailed family history and determine if genetic testing was required.

IHC staining is a relatively rapid method of screening for those that may benefit from genetic testing. IHC looks for the presence or absence of certain proteins with the results influencing the decision making process about the need to refer for a genetics opinion.

We questioned the current knowledge and practice of general and sub-specialty colorectal surgeons in NZ with regard to IHC testing and indications for referral to genetic services.

Methods

Using the revised Bethesda criteria a questionnaire (Fig 1) was designed. In designing this tool we consulted geneticists in our institution. There was concern regarding the misrepresentation of IHC as a test for MSI, which it is not. IHC shows loss of expression of the protein product of one or more mismatch repair genes. Positive IHC staining merely indicates the possibility of MSI in the DNA and hence the chance of finding Lynch syndrome mutations.

In the interest of trying to keep the questionnaire at a length likely to be responded to we agreed not to have lengthy explanations before any questions and use the word ‘possible’ in question 2. We felt that

this was an acceptable compromise as the question therefore did not state categorically IHC was a test for micro satellite instability but an abnormal result meant that MSI was a possibility.

Our finalised questionnaire was then sent to all NZ surgeons identified by the Royal Australasian College of Surgeons as general surgeons, with or without a sub-specialty interest in colorectal surgery (CRS). The questionnaire was sent out by the college to preserve confidentiality. The only identifier was a small 'c' on the questionnaire of those surgeons who had declared sub-specialty interest in colorectal surgery to the college. One month after the original mailing a follow-up letter was distributed to improve the response rate.

Figure 1. Questionnaire

Please answer the following questions by placing a tick in the appropriate box(es). Once you have completed the questionnaire please return it to us in the enclosed envelope.				
All responses will be treated anonymously				
<hr/>				
Q1. Do you resect Colorectal cancers?				
	Yes	<input type="checkbox"/>	No	<input type="checkbox"/>
Q2. Are you aware that immunohistochemistry testing of colorectal cancer for possible Microsatellite Instability (MSI) is available in New Zealand?				
	Yes	<input type="checkbox"/>	No	<input type="checkbox"/>
Q3. Which of the following patient criteria would prompt you to test for MSI in a patient with confirmed colorectal cancer? (Please tick all that apply)				
Cancer diagnosed aged	<40	<input type="checkbox"/>	<45	<input type="checkbox"/>
	<50	<input type="checkbox"/>	<55	<input type="checkbox"/>
Right-sided colon cancer		<input type="checkbox"/>		
Metachronous colon cancer		<input type="checkbox"/>		
One first degree relative with colon cancer		<input type="checkbox"/>		
Family history of;	bladder cancer	<input type="checkbox"/>		
	ovarian cancer	<input type="checkbox"/>		
	prostate cancer	<input type="checkbox"/>		
	renal tract cancer	<input type="checkbox"/>		
	endometrial cancer	<input type="checkbox"/>		
History of Inflammatory bowel disease		<input type="checkbox"/>		
Q4. What would be your response to receiving an abnormal immunohistochemistry result?				
Talk to relatives about screening for colon cancer	Yes	<input type="checkbox"/>	No	<input type="checkbox"/>
Refer to tertiary surgical unit	Yes	<input type="checkbox"/>	No	<input type="checkbox"/>
Refer to regional genetics service	Yes	<input type="checkbox"/>	No	<input type="checkbox"/>

The returned questionnaires were analysed against the revised Bethesda criteria and the results tabulated. Statistical significance was set at $p < 0.05$. Chi-squared tests were used to test for this except when a cell had < 5 observed responses Fisher's exact test was used.

Results

183 questionnaires were distributed and after reminder letters 94 (51%) were returned. Of the 94 responses, 77 surgeons were involved in treating patients with CRC, 23 of these were identified as having a sub-specialty interest in colorectal surgery. The remaining 17 were no longer practicing or not involved with CRC. As this was a

study of current practice the responses of the 17 surgeons no longer practicing or not treating CRC were excluded from the final analysis.

Overall, 68/77 (88%) surgeons knew about IHC testing. Predictably, those surgeons with a specialist interest in colorectal surgery had a greater awareness of the availability of IHC testing than those with no declared interest (96% vs 85%, p=0.3)

The results for the practice based questions are shown in Table 2. Forty-five percent of the respondents correctly identified that a colorectal cancer diagnosed before the age of 50 was one of the Bethesda criteria. A similar proportion (40%) thought a patient should be younger than 45 years of age.

Table 2. Correct responses (raw numbers and (%)) to the practice based questionnaire

Criteria to suspect Lynch syndrome	All surgeons n=77 (%)	General surgeons n=54 (%)	Colorectal interest n=23 (%)
CRC diagnosed <50 years (Yes)	35 (45)	23 (43)	12 (52)
Right sided CRC (no)	49 (64)	33 (61)	16 (70)
Metachronous CRC (yes)	54 (70)	40 (74)	14 (61)
One 1 st degree relative CRC (no)	36 (47)	25 (46)	11 (48)
FHx bladder cancer (no)	67 (87)	46 (85)	21 (91)
FHx ovarian cancer (yes)	37 (48)	29 (54)	8 (35)
FHx prostate cancer (no)	74 (96)	51 (94)	23 (100)
FHx renal tract cancer (yes)	25 (32)	18 (33)	7 (30)
FHx endometrial cancer (yes)	43 (56)	31 (57)	12 (52)
Hx IBD (no)	72 (94)	49 (91)	23 (100)

For the remaining questions the correct response rate ranged from 32–96%. The questions with the lowest correct response rate are those about Lynch syndrome associated cancers. Only 32% knew renal tract cancers were associated with Lynch syndrome, 48% knew about ovarian cancer and 56% about endometrial cancer. There is no statistically significant difference between the response rates for those with and without a declared interest in colorectal surgery.

After receiving a positive IHC result 63/77 (83%) surgeons would discuss screening/surveillance with the patient and their family, 15/77(19%) would refer the case onto a tertiary surgical unit and 70/77(93%) would refer to the regional genetic service on the basis of such a result.

Discussion

IHC staining of tumour tissue for the presence or absence of proteins indicating the possibility of a mismatch repair gene mutation is quick, inexpensive and efficient, being 100% specific and 94% sensitive⁷ compared to genetic testing. Therefore it is reasonable to regard IHC as an initial screening test for Lynch syndrome.

IHC testing and results should be regarded as genetic information and thus appropriate consent should be obtained from patients prior to testing. Ideally this should be done by the operating surgeon. However, to obtain valid consent the surgeon needs to be aware of whom to offer the test to. Our results show that 40% of

surgeons would not offer testing to patients older than 45 years and, depending on the other types of cancer in the family, 50–75% or potentially eligible patients would not be identified and tested.

The Royal College of Pathologists of Australasia released a position statement in 2006,⁸ which recommends no additional consent is required to perform IHC. However, with the exception of age, pathologists are not routinely given the necessary clinical information to make a decision about IHC.⁹ The surgical team cannot hope to provide this information on specimen forms if suspicious details in the patients history are overlooked or not enquired about.

The survey results show that New Zealand surgeons know IHC is available but knowledge about who to test is lacking. It could be argued that the results are simply a reflection of the low response rate to the questionnaire. Surveys of New Zealand Surgeons have in the past achieved response rates of 70%.¹⁰ However, these previous surveys are of all fellows of the college and not just general surgeons.

When surveying General Surgeons in NZ this may be the best we can hope to achieve. Even if we had had a 100% response rate New Zealand surgeons show gaps in their knowledge of the Bethesda criteria. The 89 non-responding surgeons were included and a sensitivity analysis (not shown here) was undertaken. The results showed that there would still be approximately 30% of patients with suspicious criteria who would go unidentified.

It is possible that some more knowledgeable surgeons found the stems of question 3 harder to answer (with a potential skewing effect on the results) as the main question could be taken to imply that IHC tests directly for MSI, which as we have noted, is incorrect. As the focus of this paper is knowledge and education we have made efforts to clarify this point in our introduction and methodology.

Conclusion

This study has shown a lack of awareness about aspects of the Bethesda criteria amongst New Zealand general surgeons. Any delay in the diagnosis of Lynch syndrome may have a negative impact on patients and their families. For those treating colorectal cancer it is prudent, if not essential, to be aware of the classic features of Lynch syndrome and test appropriately.

Competing interests: None known.

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References:

1. Ministry of Health, New Zealand Health Information Service. Cancer: New Registrations and Deaths 2005 available at <http://www.nzhis.govt.nz/moh.nsf/pagesns/32#01>
2. Statistics New Zealand, New Zealand population figures available at <http://www.stats.govt.nz/products-and-services/new-zealand-in-profile-2006/Population.htm>
3. Lynch HT, Smyrk TC, Watson P, et al. Genetics, natural history, tumor spectrum, and pathology of hereditary nonpolyposis colorectal cancer: an updated review. *Gastroenterology* 1993;104:1535–1549
4. Aarnio, M, Mecklin, J, Aaltonen, LA, et al. Lifetime risk of different cancers in hereditary non-polyposis colorectal cancer (HNPCC) syndrome. *Int J Cancer* 1995;64:430.
5. Mecklin, JP, Jarvinen, HJ. Tumor spectrum in cancer family syndrome (hereditary nonpolyposis colorectal cancer). *Cancer* 1991;68:1109.
6. Umar A, Boland CR, Terdiman JP, et al. Revised Bethesda Guidelines for Hereditary Nonpolyposis Colorectal Cancer (Lynch Syndrome) and Microsatellite Instability. *J National Cancer Inst.* 2004;96:261-8.
7. Hampel H, Frankel WL, Martin E, et al. Screening for the Lynch Syndrome (Hereditary Nonpolyposis Colorectal Cancer). *N Eng J Med* 2005;352:1851-1860
8. Royal Australasian College of Pathologists. Position Statement on IHC at <http://www.rcpa.edu.au//static/File/Asset%20library/public%20documents/Policy%20Manual/Position%20Statements/Consent%20for%20performing%20MSI%20and%20IHC%20pre-screening%20for%20HNPCC.pdf>
9. Personal communication with pathologists at Capital and Coast District Health Board; 2006 - 2010
10. Personal Communication with the New Zealand Manager of the New Zealand Office of the Royal Australasian College of Surgeons; 2009



A comparative analysis of cardiovascular disease risk profiles of five Pacific ethnic groups assessed in New Zealand primary care practice: PREDICT CVD-13

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Abstract

Background Data on the cardiovascular disease risk profiles of Pacific peoples in New Zealand is usually aggregated and treated as a single entity. Little is known about the comparability or otherwise of cardiovascular disease (CVD) risk between different Pacific groups.

Aim To compare CVD risk profiles for the main Pacific ethnic groups assessed in New Zealand primary care practice to determine if it is reasonable to aggregate these data, or if significant differences exist.

Methods A web-based clinical decision support system for CVD risk assessment and management (PREDICT) has been implemented in primary care practices in nine PHOs throughout Auckland and Northland since 2002, covering approximately 65% of the population of these regions. Between 2002 and January 2009, baseline CVD risk assessments were carried out on 11,642 patients aged 35–74 years identifying with one or more Pacific ethnic groups (4933 Samoans, 1724 Tongans, 1366 Cook Island Māori, 880 Niueans, 1341 Fijians and 1398 people identified as Other Pacific or Pacific Not Further Defined). Fijians were subsequently excluded from the analyses because of a probable misclassification error that appears to combine Fijian Indians with ethnic Fijians. Prevalences of smoking, diabetes and prior history of CVD, as well as mean total cholesterol/HDL ratio, systolic and diastolic blood pressures, and Framingham 5-year CVD risk were calculated for each Pacific group. Age-adjusted risk ratios and mean differences stratified by gender were calculated using Samoans as the reference group.

Results Cook Island women were almost 60% more likely to smoke than Samoan women. While Tongan men had the highest proportion of smoking (29%) among Pacific men, Tongan women had the lowest smoking proportion (10%) among Pacific women. Tongan women and Niuean men and women had a higher burden of diabetes than other Pacific ethnic groups, which were 20–30% higher than their Samoan counterparts. Niuean men and women had lower blood pressure levels than all other Pacific groups while Tongan men and women had the highest total cholesterol to HDL ratios. Tongan men and women had higher absolute 5-year CVD risk scores, as estimated by the Framingham equation, than their Samoan counterparts (Age-adjusted mean differences 0.71% [95% CI 0.36% to 1.06%] for Tongan men and 0.52% [95% CI 0.17% to 0.86%] for Tongan women) although these risk differences were only about 10% higher in relative terms.

Conclusion The validity of the analyses depend on the assumption that the selection of participants for CVD risk assessment in primary care is similar between Pacific groups. The ethnic-specific CVD risk profiles presented do not represent estimates of population prevalence. Almost all previous Pacific data has been aggregated with Pacific peoples treated as a single entity because of small sample sizes. We have analysed data from the largest study to date measuring CVD risk factors in Pacific peoples living in New Zealand. Our findings suggest that aggregating Pacific population data appears to be reasonable in terms of assessing absolute CVD risk, however there are differences for specific CVD risk factors between Pacific ethnic groups that may be important for targeting community level interventions.

Studies since the 1990s have consistently shown that Pacific peoples in New Zealand have higher incidence, mortality and case-fatality rates from cardiovascular diseases (CVD) than New Zealand Europeans.¹⁻⁴ Research also suggests that these outcomes are the result of a more adverse CVD risk factor profile among Pacific peoples compared to Europeans.⁵⁻¹⁰

However, Pacific peoples in New Zealand are not a homogeneous population. The term 'Pacific peoples' is an umbrella term describing about 7% of the New Zealand population who identify with at least one of the ethnic groups originating from the Pacific Islands of Polynesia, Melanesia and Micronesia.¹¹ There are over 12 nations represented in New Zealand's Pacific community. However most Pacific peoples identify with one or more of the four main ethnic groups (Samoan, Tongan, Cook Island Māori and Niuean).¹²

Most health-related surveys present aggregated data from these different Pacific ethnic groups, in part because of the small samples of each contributing ethnic group. Research into CVD has been no exception. Despite recommendations to investigate each Pacific ethnic group separately,¹³⁻¹⁵ only two previous studies have attempted ethnic-specific analyses of the CVD risk profiles of Pacific peoples in New Zealand, and both studies only had the statistical power to find substantial differences between Pacific groups.^{13,16} Also of note, the New Zealand Census Mortality Study (NZCMS) reported much higher CVD mortality among Cook Island Māori (RR 1.66 compared with Samoans), which was reproduced in both 1991-99 and 2001-04 cohorts.¹⁵ This mortality differential is unexplained.

PREDICT is a clinical decision support programme aimed at assisting primary care practitioners with CVD risk assessment and management.¹⁷ Since 2002, it has been implemented in nine PHOs throughout Auckland and Northland, representing about 65% of the population in these two regions. Over 10,000 Pacific participants have now been recruited into the PREDICT programme, which has generated the largest Pacific cohort ever assembled in New Zealand. This cohort does not currently include a representative sample of people living in New Zealand as only about 20% of the eligible population have been risk assessed to date. However it seems unlikely that there would be any systematic differences in the selection of patients from different Pacific groups for CVD risk assessment. Therefore, we present a comparative analysis of CVD risk factor profiles in the major Pacific populations living in New Zealand.

While the ethnic-specific risk factor profiles reported here do not represent prevalence estimates for the Pacific populations in New Zealand, the PREDICT study provides

an opportunity to add to our very limited knowledge on CVD risk factor differences between Pacific populations. Comparing these risk profiles could help answer the question “Is it appropriate to aggregate data on CVD risk profiles from different Pacific ethnic groups living in New Zealand?”

Methods

PREDICT is a web-based clinical decision support programme for CVD risk assessment and management in routine primary care practice. Study methods and data definitions are described in full elsewhere.¹⁷

The software programme has been integrated with several of the most commonly used primary care patient management systems. This integration allows uniform, systematically coded CVD risk data to be automatically (and anonymously) extracted from a patient’s electronic medical record. Gaps in the data required to undertake a formal CVD risk assessment are then completed by either the GP or practice nurse on the PREDICT templates, which are then automatically written back to the patient medical record. Health data captured by PREDICT, including history of CVD, family history of CVD, and total cholesterol/HDL ratio, have previously been shown to be highly consistent with data held in electronic patient records.¹⁸

Risk profiles are sent via a secure broadband internet connection to a central server at the time of the assessment. Within seconds the clinician receives the patient’s calculated 5-year CVD risk as well risk management recommendations based on New Zealand CVD risk management guidelines.¹⁹ The central server stores the CVD risk factor profiles of each patient, and with permission from participating PHOs, these are extracted anonymously and linked, via an encrypted National Health Index (NHI) number, to national hospitalisation and mortality datasets. PREDICT data can also be linked to the New Zealand Health Information Service (NZHIS) NHI dataset that holds details of date of birth, gender, ethnicity and socioeconomic status according to the NZDep01 Deprivation index.

Ethnicity data can therefore be collected from the PREDICT template (originally from the patient’s electronic medical record, or entered manually by the practitioner) or via linkage to the NHI dataset. Both datasets have provision for up to three different ethnicities to be entered, so that each patient in the PREDICT cohort could potentially have up to six ethnicities.

Any person in the PREDICT cohort aged 35–74 years with a Pacific ethnicity on any one of the six potential ethnicity fields was included in these analyses. Pacific ethnicities were defined according to the Ministry of Health’s *Ethnicity Data Protocols for the Health and Disability Sector* as Level 2 codes 30 to 37.²⁰ However, preliminary analyses revealed that the Fijian group (code 36) was an anomaly, making up over 11% of the total Pacific cohort but only 4% of Pacific peoples in official New Zealand statistics.¹² Furthermore, as the CVD risk profile of the Fijian group was much closer to that of the Indian cohort than to that of the other Pacific groups,²¹ we suspected that some of those classified as Fijians were not ethnic Fijians, but rather Fijian Indians. Therefore this group (n=1341) was excluded from these analyses.

The Pacific groups included in this study were thus the level 2 ethnicity codes 31 (Samoan), 32 (Cook Island Māori), 33 (Tongan), 34 (Niuean), and a combined group of both codes 37 (Other Pacific peoples, including Tokelauans) and code 30 (Pacific peoples Not Further Defined, NFD).

Less than 1% of patients in the Pacific cohort identified with more than one Pacific ethnicity. Classification of ethnicity prioritised the smaller Pacific groups over the larger ones, as was done in the 2001 New Zealand Census²² and the Diabetes, Heart and Health Study 2002/03.¹⁶ This method gave first priority to Niuean, followed by Cook Island Māori, Tongan and lastly Samoan ethnicity. Only 70 people (0.7% of the Pacific cohort) identified themselves as Tokelauan (code 35), therefore they were included in the ‘Other Pacific peoples’ ethnic group (code 37).

The data extracts for these analyses included all PREDICT first risk assessments from August 2002 until January 2009. Data were analysed using Stata v10.0 statistical software. Men and women were analysed separately. Deprivation was assessed according to the NZDep01 index. NZDep01 is a census based small area index of deprivation, which assigns a relative deprivation score to each meshblock in New Zealand.²³ Each individual was assigned the value according to their meshblock of residence. Risk ratios (RRs) with 95% confidence intervals (CIs) were calculated for each ethnic group with Samoans as the reference group, as they comprised the largest group, making up almost 50% of the

Pacific cohort. A binomial regression model was used to calculate risk ratios (RR) adjusted for age. A post-regression test was used to test for any overall differences in proportions between groups. A linear regression model adjusting for age was used to calculate mean differences and 95% CIs for continuous CVD risk factor data, again with Samoans as the reference group.

Mean differences in Framingham risk scores were also calculated, using the original Framingham risk prediction equation²⁴ rather than the New Zealand Guidelines Group-adjusted Framingham equation which adds a 5% 5 year risk increment to high risk ethnic groups including Pacific peoples.¹⁹ We decided not to adjust for deprivation, as there were only small differences in NZDep between Pacific groups and it is a relatively crude and indirect measure of deprivation which is most useful when there are major differences between groups.

Ethical approval—The PREDICT research project was approved by the Northern Region Ethics Committee Y in 2003 (AKY/03/12/314) and the national Multi Region Ethics Committee in 2007 (MEC/07/19/EXP).

Results

Between 2002 and June 2009, baseline PREDICT CVD risk assessments were conducted on 10,301 people aged 35–74 identifying with at least one Pacific ethnic group, after excluding those classified as Fijian (as discussed in the Methods). Of these, 48% identified as Samoan, 17% as Tongan, 13% as Cook Island Māori, 8% as Niuean and 14% as Other Pacific or Pacific NFD.

Table 1 shows the baseline demographic characteristics of Pacific peoples in the PREDICT cohort by Level 2 ethnic group. In most Pacific groups, there were similar proportions of men and women receiving CVD risk assessments. The only exception to this was for the Tongan cohort (54% men).

On average, Pacific women were approximately 3 years older than their male counterparts. The distribution of age groups at first CVD risk assessment was similar across Pacific groups, with approximately 25% aged 35–44, 35% aged 45–54 and 25% aged 55–64. All Pacific groups were over-represented in areas of high deprivation, with approximately 75% residing in the two most deprived NZDep01 quintiles (deciles 7–10).

Table 2 presents the gender-specific age-adjusted risk ratios for three CVD risk factors (smoking, diabetes and a prior history of CVD) for Pacific groups, using Samoans as the reference group. There were no overall differences in the proportion of smokers among Pacific men ($p=0.16$). Cook Island women had the highest proportion of smokers among women (21%) and were almost 60% more likely than Samoan women to smoke. Niuean men had the highest burden of diabetes (almost 40%) among Pacific males.

Pacific women in all groups had a higher prevalence of diabetes than their male counterparts, with over one-third of all Pacific women having a diagnosis of diabetes. Tongan women were 26% more likely, and Niuean women 17% more likely, than Samoan women to have diabetes. Overall differences between Pacific groups in the proportion with a prior history of CVD were small.

Table 1. Baseline demographic characteristics of people in Pacific ethnic groups in the PREDICT cohort

Variables		Samoaan	Tongan	Cook Island Māori	Niuean	Other Pacific/Pacific NFD
Total, n (%)	10301	4933 (48%)	1724 (17%)	1366 (13%)	880 (8%)	1398 (14%)
Gender, n (%)	Male	2586 (52%)	937 (54%)	679 (50%)	423 (48%)	699 (50%)
	Female	2347 (48%)	787 (46%)	687 (50%)	457 (52%)	699 (50%)
Mean age (SD)	Male	52.0 (9.8)	51.0 (10.0)	52.1 (10.1)	52.5 (10.0)	50.7 (9.7)
	Female	54.9 (9.2)	54.6 (9.4)	55.1 (9.5)	55.4 (9.8)	54.7 (9.0)
Age group, n (%)	35-44 y	1209 (24%)	482 (28%)	316 (23%)	203 (23%)	354 (25%)
	45-54 y	1724 (35%)	602 (35%)	522 (38%)	315 (36%)	510 (37%)
	55-64 y	1367 (28%)	418 (24%)	301 (22%)	208 (24%)	379 (27%)
	65-74 y	633 (13%)	222 (13%)	227 (17%)	154 (17%)	155 (11%)
NZDep01 Deprivation quintiles, n (%)	1-2	111 (2%)	31 (2%)	41 (3%)	18 (2%)	64 (5%)
	3-4	307 (6%)	99 (6%)	136 (10%)	61 (7%)	101 (7%)
	5-6	625 (13%)	181 (10%)	185 (14%)	153 (17%)	192 (14%)
	7-8	1469 (30%)	416 (24%)	290 (21%)	247 (28%)	480 (34%)
	9-10	2416 (49%)	995 (58%)	712 (52%)	400 (46%)	557 (40%)

Table 2. Age-adjusted estimates and risk ratios, with 95% confidence intervals, for smoking, diabetes and prior history of CVD for Pacific groups in PREDICT cohort by gender (reference group is Samoaan)

	Pacific subgroup	Male, n (%) with risk factor	RR (95% CI) adjusted for age	Female, n (%) with risk factor	RR (95% CI) adjusted for age
Smoking	Samoaan	684 (26%)	Reference	312 (13%)	Reference
	Tongan	285 (29%)	1.13 (1.01-1.27)	84 (10%)	0.79 (0.63-0.99)
	Cook Island Māori	183 (26%)	1.02 (0.90-1.18)	144 (21%)	1.59 (1.34-1.89)
	Niuean	99 (23%)	0.90 (0.74-1.07)	67 (15%)	1.12 (0.88-1.43)
	Pacific Other/NFD	176 (24%)	0.93 (0.81-1.07)	92 (13%)	0.97 (0.78-1.21)
Diabetes	Samoaan	734 (28%)	Reference	807 (32%)	Reference
	Tongan	271 (29%)	1.04 (0.93-1.16)	346 (41%)	1.26 (1.15-1.39)
	Cook Island Māori	173 (25%)	0.89 (0.78-1.03)	211 (29%)	0.89 (0.79-1.01)
	Niuean	161 (37%)	1.31 (1.15-1.48)	187 (38%)	1.17 (1.04-1.32)
	Pacific Other/NFD	183 (26%)	0.93 (0.81-1.07)	235 (32%)	0.98 (0.88-1.10)
History of CVD	Samoaan	264 (9%)	Reference	192 (6%)	Reference
	Tongan	80 (8%)	0.89 (0.70-1.11)	44 (4%)	0.70 (0.51-0.97)
	Cook Island Māori	81 (10%)	1.13 (0.91-1.42)	58 (6%)	1.01 (0.77-1.33)
	Niuean	55 (11%)	1.23 (0.95-1.59)	35 (6%)	0.90 (0.63-1.25)
	Pacific Other/NFD	84 (11%)	1.29 (1.03-1.60)	56 (6%)	1.01 (0.76-1.34)

Table 3 shows the age-adjusted mean systolic and diastolic blood pressures (BPs), total cholesterol/HDL ratio and Framingham 5-year CVD risk scores of Pacific groups, by gender. Niuean men and women had the lowest mean systolic and diastolic BPs of all groups (mean systolic and diastolic BPs 2 to 3 mmHg lower than Samoans). Tongan men had the highest mean total cholesterol/HDL ratio (0.26 higher than Samoan men), and Niuean men the lowest (0.16 lower than Samoan men). Tongan women had the highest mean total cholesterol/HDL ratio (0.14 higher than Samoan women after adjusting for age and deprivation).

Tongan men and women had the highest 5-year Framingham CVD risk scores and while these were statistically significantly higher than the Samoan reference categories, they only represented about a 10% relative difference in risk.

Table 3. Age-adjusted mean values and mean differences for systolic and diastolic blood pressures, total cholesterol/HDL ratio and Framingham 5-year CVD risk score for Pacific groups in PREDICT cohort by gender (Reference group is Samoan)

	Pacific subgroup	Male Age-adjusted mean (95% CI)	Age-adjusted mean difference (95% CI)	Female Age-adjusted mean (95% CI)	Age-adjusted mean difference (95% CI)
Systolic BP (mmHg)	Samoan	131.2 (130.5 to 131.8)	Reference	132.2 (131.4 to 132.9)	Reference
	Tongan	131.7 (130.7 to 132.8)	0.6 (-0.7 to 1.8)	131.9 (130.6 to 133.2)	-0.3 (-1.8 to 1.2)
	Cook Island	132.8 (131.5 to 134.0)	1.6 (0.2 to 3.0)	133.1 (131.7 to 134.5)	0.9 (-0.6 to 2.5)
	Niuean	128.7 (127.1 to 130.3)	-2.5 (-4.2 to -0.8)	129.9 (128.2 to 131.6)	-2.3 (-4.1 to -0.5)
	Pacific Other/NFD	132.7 (131.5 to 133.9)	1.6 (0.1 to 2.90)	133.9 (132.5 to 135.2)	1.7 (0.1 to 3.2)
Diastolic BP (mmHg)	Samoan	82.6 (82.2 to 83.1)	Reference	82.0 (81.6 to 82.5)	Reference
	Tongan	81.9 (81.1 to 82.6)	-0.8 (-1.6 to 0.1)	80.7 (79.9 to 81.5)	-1.3 (-2.2 to -0.5)
	Cook Island	83.5 (82.7 to 84.4)	0.9 (-0.1 to 1.8)	83.0 (82.2 to 83.8)	1.0 (0.1 to 1.9)
	Niuean	80.1 (79.0 to 81.2)	-2.5 (-3.7 to -1.4)	79.1 (78.1 to 80.1)	-2.9 (-4.0 to -1.8)
	Pacific Other/NFD	82.8 (82.0 to 83.7)	0.2 (-0.7 to 1.1)	81.8 (81.0 to 82.6)	-0.2 (-1.2 to 0.7)
Tot chol: HDL ratio	Samoan	4.24 (4.19 to 4.29)	Reference	3.78 (3.74 to 3.83)	Reference
	Tongan	4.50 (4.42 to 4.59)	0.26 (0.16 to 0.36)	3.93 (3.85 to 4.00)	0.14 (0.05 to 0.23)
	Cook Island	4.32 (4.22 to 4.42)	0.08 (-0.04 to 0.18)	3.89 (3.81 to 3.97)	0.11 (0.01 to 0.20)
	Niuean	4.09 (3.96 to 4.21)	-0.16 (-0.29 to -0.02)	3.75 (3.65 to 3.85)	-0.03 (-0.14 to 0.08)
	Pacific Other/NFD	4.33 (4.23 to 4.43)	0.09 (-0.02 to 0.20)	3.83 (3.74 to 3.91)	0.05 (-0.05 to 0.14)
Framingham 5yr CVD risk %	Samoan	8.17 (7.99 to 8.34)	Reference	5.20 (5.03 to 5.38)	Reference
	Tongan	8.88 (8.57 to 9.18)	0.71 (0.36 to 1.06)	5.72 (5.42 to 6.02)	0.52 (0.17 to 0.86)
	Cook Island	8.51 (8.16 to 8.87)	0.35 (-0.05 to 0.75)	5.45 (5.13 to 5.77)	0.25 (-0.12 to 0.62)
	Niuean	7.86 (7.41 to 8.30)	-0.30 (-0.79 to 0.17)	5.23 (4.83 to 5.62)	0.02 (-0.41 to 0.46)
	Pacific Other/NFD	8.37 (8.02 to 8.72)	0.20 (-0.19 to 0.59)	5.54 (5.22 to 5.86)	0.33 (-0.03 to 0.70)

Body mass index (BMI) is not used in the Framingham risk equation to calculate CVD risk and it is therefore not part of the risk assessment template. BMI is only mandatory if clinicians wish to receive CVD management recommendations based on New Zealand guidelines. Therefore, BMI was only available for the 30% of this cohort whose general practitioner completed both a risk assessment and risk management template. Mean BMI for this subset did not differ significantly between different ethnic groups, with BMI ranging from 32.6 to 33.9 in men and 34.3 to 36.7 in women.

Discussion

This study presents a comparative analysis of CVD risk profiles by the major Pacific ethnic groups living in New Zealand. The data were generated from a web-based CVD risk assessment and management decision supported system used in the majority of PHOs in the Northland and Auckland regions and represents the largest cohort of Pacific people ever assembled in New Zealand. As only about 20% of the eligible population has been risk assessed to date, these findings cannot be used as population prevalence estimates. However we assumed that there were unlikely to be important systematic differences in CVD risk factor screening between the different Pacific populations, so that it was reasonable to undertake comparative analyses. Interestingly, the age, gender and NZDep01 profiles of the individual Pacific groups were remarkably similar.

We found relatively small differences in overall CVD risk factor profiles between the five Pacific groups assessed, suggesting that it is reasonable to combine these data when describing the overall CVD risk profiles of Pacific peoples living in New Zealand. However there were substantial differences in the prevalence of some individual CVD risk factors. The largest relative difference for a single risk factor was the almost 60% higher proportion of smokers among Cook Island women compared to Samoan women, which is consistent with results from previous studies.^{16,25,26}

Of note, smoking rates among Pacific groups in the PREDICT cohort were lower than those reported by other studies, including the 2006 New Zealand Census²⁷ and the Pacific Drug and Alcohol Survey (PDAS).²⁶ However, the reported Census data included all Pacific peoples aged 15 years and over, while our study included those aged between 35 and 74 years, and the prevalence of smoking decreases significantly with age.²⁸ The PDAS reported smoking rates for Pacific peoples aged 13 to 65 years, and had only about one tenth the sample size of our study (n=338 Samoans, 228 Cook Island Māori, 232 Tongans and 207 Niueans aged 13–65 years).²⁶ The PDAS did not have the statistical power and precision to provide meaningful comparisons between Pacific groups.

In our study, Tongan women and Niuean men and women had the highest burden of diabetes (approximately 40%), making them 20-30% more likely to have diabetes than their Samoan counterparts. Tongan women have previously been reported to have the highest prevalence of diabetes among Pacific groups in the Diabetes Heart and Health Study (DHAHS) 2002/03,¹⁶ and Niuean men the highest prevalence of diabetes among Pacific men in the Workforce Diabetes Survey (WDS) 1988-1990.¹³ However, our finding of a high burden of diabetes among Niuean women has not

been described previously—possibly because of the small numbers of Niuean women included in earlier studies (only 13 in the WDS and 60 in the DHAHS).^{13, 16}

Despite their high burden of diabetes, Niuean men and women in our study had the lowest mean total cholesterol/HDL ratios and systolic and diastolic blood pressures of the five Pacific groups (approximately 2-3 mmHg lower than Samoan men and women). Our finding that Niueans had lower blood pressure levels than other Pacific groups has not been reported previously. However, Niuean men and women were reported to have significantly lower mean total cholesterol:HDL ratios compared to other Pacific peoples in both the WDS and the DHAHS, suggesting that the differences in this particular CVD risk factor between Niueans and other Pacific groups are real.

The lower mean levels of systolic and diastolic BP and total cholesterol:HDL ratio in Niueans compared to other Pacific groups in our study may possibly be due to targeted risk management in people with diabetes, given they also had the highest burden of diabetes in these analyses. We intend to explore this hypothesis further once we have linked these data to national pharmaceutical dispensing records. Comparing our data with all other New Zealand surveys (except the Census) that have attempted to make comparisons of CVD risk factors between the different Pacific populations living in New Zealand is problematic because the largest of these other surveys was about one-tenth the size of the PREDICT cohort. Therefore random error is likely to be a major problem when considering the validity of comparative differences.

Unfortunately we were unable to provide any insights into the observation by Blakely et al.¹⁵ of an increased risk of CVD death among Cook Island Māori compared to other Pacific peoples. While we identified some differences between groups for individual risk factors, our summary measure of overall risk, using the Framingham score, suggested that Tongan men and women had a small increased risk compared to other Pacific groups, which had similar Framingham estimated risks. However the Framingham risk score was developed to help improve the targeting of risk factor management in individual patients and at best only explains about one third the variability in CVD event rates in a population.

While it is possible there were systematic differences in the selection criteria for risk assessing different Pacific groups which might explain our inability to detect a higher overall risk among Cook Island Māori, it is more likely that the tool was simply not sufficiently sensitive. We plan to investigate the predicted risk versus the actual observed event rate in this cohort, with a view to developing more accurate risk prediction equations.

This study highlights the ongoing problems with the collection of ethnicity data in the health sector. Prior to 2004, when the Ministry of Health introduced *Ethnicity Protocols for the Health and Disability Sector*,²⁰ there were no standards for the coding and recording of ethnicity data in primary care. Consequently, inconsistencies in ethnicity recording have been noted in hospital²⁹ and primary care records.³⁰ A study by Riddell et al. involving a sample (n=665) of people who had previously been risk assessed using PREDICT also found that self-identified ethnicity was the same as that recorded in the primary care record for only two-thirds of the sample.³¹

Notably, this study found that only 37.5% of those recorded as Fijian on the primary care record agreed with this when asked to self-identify their ethnicity. This finding further supports our exclusion of PREDICT participants classified as Fijian from the analyses. Furthermore, Riddell's study found zero agreement among those recorded as Pacific Not Further Defined (NFD) on the primary care record with their self-identified ethnicity—presumably because the majority of these people identify with a specific Pacific ethnic group (for example, Samoan, Tongan etc).³¹ Our finding that 959 people (9.3% of our cohort) were only classified as Pacific NFD, rather than as a particular Pacific ethnic group, suggests that there are still significant gaps in the accuracy of recording of ethnicity in primary care.

In conclusion, this study has highlighted several differences in CVD risk factors between five Pacific ethnic groups assessed in routine primary care, including higher proportions of smoking in Cook Island women, a higher burden of diabetes among Tongan women and Niuean men and women, and slightly higher estimates of 5-year absolute CVD risk in Tongan men and women. These differences suggest that Pacific peoples should not necessarily be treated as a single entity when designing community-based health promotion and disease prevention programmes, and that a targeted ethnic-specific approach to the reduction of some CVD risk factors may be appropriate.

Research is currently limited in this area, however, and studies will need to be conducted to evaluate whether ethnic-specific Pacific interventions are more effective than general Pacific interventions. In contrast, overall (absolute) CVD risk, as estimated by the Framingham equation, is remarkably similar between the Pacific ethnic groups, suggesting that the current practice of aggregating CVD risk data is reasonable for describing the overall risk factor burden for Pacific peoples living in New Zealand.

Competing interests: None.

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Acknowledgements: The authors would like to thank the following PHOs, as well as their affiliated general practitioners, practice nurses and patients, for providing the data for analysis: ProCare Network North, Auckland and Manukau, HealthWest, Te Tai Tokerau, Manaia, Kaipara Care, Tihewa Mauriora and Whangaroa PHOs.

PREDICT was developed by a collaboration of clinical epidemiologists at the University of Auckland, IT specialists at Enigma Publishing Ltd (a private provider of online health knowledge systems), primary health care organisations, non-governmental organisations (New Zealand Guidelines Group, National Heart Foundation, Diabetes New Zealand, Diabetes Auckland), several district health boards and the Ministry of Health.

PREDICT software platform is owned by Enigma Publishing Ltd (PREDICT is a trademark of Enigma Publishing Ltd).

Funding: The PREDICT research project has support by HRC grants 03/183 and 08/121 from the Health Research Council. CG received a training endowment from the New Zealand College of Public Health Medicine. SW has been, and TR is currently, a recipient of a National Heart Foundation Research Fellowship.

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References:

1. Tukuitonga C, Stewart A, Beaglehole R. Coronary heart disease in Pacific Islands people in New Zealand. *N Z Med J.* 1990;103:448–9.
2. Bell C, Swinburn B, Stewart A, et al. Ethnic differences and recent trends in coronary heart disease in New Zealand. *N Z Med J.* 1996;109:66–8.
3. Bullen C, Beaglehole R. Ethnic differences in coronary heart disease case fatality rates in Auckland. *Aust N Z J Public Health.* 1997;21:688–93.
4. Blakely T, Tobias M, Atkinson J, et al. Tracking Disparity: Trends in ethnic and socioeconomic inequalities in mortality, 1981–2004. Wellington: Ministry of Health; 2007.
5. Scragg R, Baker J, Metcalf P, Dryson E. Hypertension and its treatment in a multicultural workforce. *N Z Med J.* 1993;106:96–9.
6. Scragg R, Baker J, Metcalf P, Dryson E. Serum lipid levels in a New Zealand multicultural workforce. *N Z Med J.* 1993;106:96–9.
7. Bullen C, Tipene-Leach D, Vander Hoorn S, et al. Ethnic differences in blood pressure: findings from the Fletcher Challenge-Auckland University Heart and Health Study. *N Z Med J.* 1996;109:395–7.
8. Simmons D, Harry T, Gatland B. Prevalence of known diabetes in different ethnic groups in inner urban South Auckland. *N Z Med J.* 1999;112:316–9.
9. Gentles D, Metcalf P, Dyal L, et al. Blood pressure prevalences and levels for a multicultural population in Auckland, New Zealand: results from the Diabetes, Heart and Health Survey 2002/2003. *N Z Med J.* 2006;119(1245). <http://www.nzmj.com/journal/119-1245/2318/content.pdf>
10. Gentles D, Metcalf P, Dyal L, et al. Serum lipid levels for a multicultural population in Auckland, New Zealand: results from the Diabetes Heart and Health Survey (DHAH) 2002–2003. *N Z Med J.* 2007;120(1265). <http://www.nzmj.com/journal/120-1265/2800/content.pdf>

11. Ministry of Health. Improving Quality of Care for Pacific Peoples. Wellington: Ministry of Health; 2008.
12. Statistics New Zealand. Pacific profiles 2006. Wellington: Statistics New Zealand; 2007. <http://www.stats.govt.nz/census/2006-census-data/2006-census-reports/pacific-profiles-2006.aspx>
13. Schaaf D, Scragg R, Metcalf P. Cardiovascular risk factor levels of Pacific people in a New Zealand multicultural workforce. *N Z Med J.* 2000;113:3-5.
14. Sundborn G, Metcalf P, Schaaf D, et al. Differences in health-related socioeconomic characteristics among Pacific populations living in Auckland, New Zealand. *N Z Med J.* 2006;119(1228). <http://www.nzmj.com/journal/121-1281/3238/content.pdf>
15. Blakely T, Richardson K, Young J, et al. Does mortality vary between Pacific groups in New Zealand? Estimating Samoan, Cook Island Maori, Tongan and Niuean mortality rates using hierarchical Bayesian modelling. *N Z Med J.* 2009;122(1307). <http://www.nzmj.com/journal/122-1307/3910/content.pdf>
16. Sundborn G, Metcalf PA, Gentles D, et al. Ethnic differences in cardiovascular disease risk factors and diabetes status for Pacific ethnic groups and Europeans in the Diabetes Heart and Health Survey (DHAH) 2002-2003, Auckland New Zealand. *N Z Med J.* 2008;121(1281). <http://www.nzmj.com/journal/121-1281/3238/content.pdf>
17. Bannink L, Wells S, Broad J, et al. Web-based assessment of cardiovascular disease risk in routine primary care practice in New Zealand: the first 18,000 patients (PREDICT CVD-1). *N Z Med J.* 2006;119(1245). <http://www.nzmj.com/journal/119-1245/2313/content.pdf>
18. Riddell T, Kenealy T, Wells S, et al. Audit of health data captured routinely in primary healthcare for the clinical decision support system PREDICT (PREDICT CVD-4). *Health Care and Informatics Review Online*, March 2008.
19. New Zealand Guidelines Group. The assessment and management of cardiovascular risk. Wellington: New Zealand Guidelines Group; 2003.
20. Ministry of Health. Ethnicity data protocols for the health and disability sector. Wellington: Ministry of Health; 2004.
21. Perumal L. Cardiovascular disease risk burden of Indians in a New Zealand primary care setting. Auckland: University of Auckland; 2009.
22. Allan J. Review of measurement of Ethnicity: Classification and Issues. Wellington: Statistics New Zealand; 2001.
23. Salmond C, Crampton P. NZDep2001 index of deprivation – User’s manual. Wellington: Department of Public Health, Wellington School of Medicine and Health Sciences; 2002.
24. Anderson KM, Odell PM, Wilson PWF, Kannel W. Cardiovascular disease risk profiles. *American Heart Journal.* 1991;121(1 Pt 2):293-8.
25. Erick-Peleti S, Paterson J, Williams M. Pacific Islands Families Study: maternal factors associated with cigarette smoking amongst a cohort of Pacific mothers with infants. *N Z Med J.* 2007;120(1256). <http://www.nzmj.com/journal/120-1256/2588/content.pdf>
26. Pacific Research and Development Services & SHORE/Whariki. Pacific drugs & alcohol consumption survey 2003. Final report: volume 1. Wellington: Massey University; 2004.
27. Statistics New Zealand. Pacific profiles: 2006. <http://www.stats.govt.nz/analytical-reports/pacific-profiles-2006/default.htm>
28. Grey C, Wells S, Riddell T, et al. A comparative analysis of the cardiovascular risk factor profiles of Pacific peoples and Europeans living in New Zealand assessed in routine primary care: PREDICT CVD-11. *N Z Med J.* 2010; 123(1309). <http://www.nzmj.com/journal/123-1309/3987/content.pdf>
29. Swan J, Lillis S, Simmons D. Investigating the accuracy of ethnicity data in New Zealand hospital records: still room for improvement. *N Z Med J.* 2006;119(1239). <http://www.nzmj.com/journal/119-1239/2103/content.pdf>

30. Health Utilisation Research Alliance. Ethnicity data and primary care in New Zealand: lessons from the Health Utilisation Research Alliance (HURA) study. N Z Med J. 2006;119(1231). <http://www.nzmj.com/journal/119-1231/1917/content.pdf>
31. Riddell T, Lindsay G, Kenealy T, et al. The accuracy of ethnicity data in primary care and its impact on cardiovascular risk assessment and management - PREDICT CVD-8. N Z Med J. 2008;121(1281). <http://www.nzmj.com/journal/121-1281/3239/content.pdf>



Pathology referrals for skin lesions—**are we giving the pathologist sufficient clinical information?**

Marius Rademaker, Murray Thorburn

Abstract

Aim To assess the quality of data included in the histology request form.

Method We prospectively reviewed the histology request forms of 375 consecutive skin lesions. In addition, the appropriateness of the surgical specimen was determined.

Results There were 196 women and 179 men with a mean age of 58.4 years. The majority of specimens (84.5%) derived from primary care. 233 lesions (62%) were removed by excision, 57 (15%) by shave, three by curettage, with 82 lesions (22%) by punch/incisional biopsy. The clinical diagnosis was either not specified in 56 cases (15%), or simply labelled as 'lesion' in 84 (22%) patients. In 140/375 cases (37%), no useful clinical information was available.

The clinical diagnosis matched the histopathological diagnosis in 145 cases (39%). Sixty percent (78/131) of histologically confirmed malignant lesions had not been identified clinically as being malignant: only 2 of 12 (17%) melanomas, 33/74 (45%) BCCs and 18/45 SCCs (57%) were diagnosed clinically. The specimen type was considered inadequate to make a histopathological diagnosis in 25 cases (6.7%).

Conclusion In over a third of histology requests, diagnostic clinical information was absent. In addition, punch biopsy was used in 40% of lesions where a melanoma was being considered clinically.

The pathology request form is a crucial communication document between treating physician and histopathologist.¹ The data set should include demographics (age and sex of patient), site and type of specimen, clinical history and preferably the diagnostic query the treating physicians wants answered by the pathologist.

Anecdotal evidence suggests that the information routinely captured on the pathology request form is often minimal, thereby affecting the ability of the histopathologist to correctly report the specimen.²⁻⁶

In addition, the recent Australasian / New Zealand Melanoma guidelines recommend that the optimal biopsy approach for a pigmented skin lesion suspicious of melanoma is complete excision with a 2 mm margin, as partial biopsies may not be fully representative of the lesion.⁶ They do however, go on to comment, "*Incisional, punch or shave biopsies may be appropriate in carefully selected clinical circumstances, for example, for large facial or acral lesions, or where the suspicion of melanoma is low.*"

This study aimed to assess the quality of information available to the pathologist from the histology request forms, the accuracy of the preliminary clinical diagnosis, and the appropriateness of the biopsy sample provided.

Methods

The histology request forms of skin lesions, read by a single pathologist over a 4-week period, were prospectively assessed for the quality of information. Particulars assessed included demographic data (age and sex of patient), body site of specimen, nature of specimen and all clinical information included on the form.

The histopathologist graded each request form as to whether it contained enough information to determine the purpose of the procedure (was it diagnostic or therapeutic), the type of sample provided (whether curettage, shave, punch or excision), the anatomical site, and whether the specimen was thought to potentially be a melanoma, a pigmented lesion, a non-melanoma skin cancer, or some other skin lesion. The histopathological diagnosis was then compared to the preliminary clinical diagnosis. The suitability of the specimen was also determined according to the clinical and histological diagnosis.

Results

Over a 4-week period during October/November 2009, 375 skin specimens were received. These were from 196 women and 179 men, with a mean age of 58.4 years (SD 16.4 yrs, range 6 - 105 yrs). The majority of the skin specimens had been taken in the primary care setting (317 lesions, 84.5%), with the remainder by plastic surgeons (43 lesions, 11.5%), dermatologists (eight lesions, 2%) and 'other' specialist surgeons (seven lesions, 2%).

The face was the most common site (92 lesions) followed by the back (66), trunk (62), legs (59), arms (58) and neck (28). In 10 instances, body site of the specimen was not specified. The majority of specimens were removed by excision (233 lesions, 62%), with 57 (15%) lesions removed by shave and three by curettage (Table 1). Seventy-six (20%) lesions were punch biopsies and six were incisional diagnostic biopsies. Eighty-one specimens (22%) were marked as being diagnostic, 74 (20%) therapeutic, with the majority not specified (220, 59%).

Table 1. Type of biopsy with number of specimens inadequate to make a histological diagnosis

Type of biopsy	Number (%)	Specimen insufficient for diagnosis (%)
Excision	233 (62.1%)	4 (1.7%)
Punch	76 (20.3%)	10 (13.2%)
Shave	57 (15.2%)	9 (15.8%)
Incisional	6 (1.6%)	0
Curettage	3 (0.8%)	2 (67%)
Total	375 (100%)	25 (6.7)

From the request form, the histopathologist concluded that the referring doctor thought 123 (33%) lesions might be a non-melanoma skin cancer, 77 (20.5%) a pigmented lesion, 27 (7%) a melanoma with 42 (11%) other types of skin lesions. In 106 (28%) lesions, it was not possible to determine what the referring practitioners thought the lesion might be.

The most common clinical diagnoses offered were basal cell carcinoma (54 cases), naevus (48), squamous cell carcinoma or keratoacanthoma (47), seborrhoeic keratosis (15), and melanoma (12) (Table 2). The clinical diagnosis was either not specified in

56 cases (15%), or simply labelled as 'lesion' in 84 (22%) patients. Therefore in 140/375 cases (37%), no useful clinical information was available to the pathologist.

Table 2. Clinical diagnosis with number (%) confirmed by histology

Variables	Clinical diagnosis	Confirmed by histology
Lesion – (not otherwise specified)	84	–
No diagnosis	56	–
Basal cell carcinoma	54	33 (61%)
Naevus	48	25 (52%)
Squamous cell carcinoma/keratoacanthoma	47	20 (42.5%)
Seborrhoeic keratosis	15	13 (87%)
Melanoma	12	2 (17%)
Solar keratosis	10	4 (40%)
Papilloma	10	2 (20%)
Dermatofibroma	5	3 (60%)
Haemangioma	5	1 (20%)
Keratosis	5	1 (20%)

The clinical diagnosis matched the histopathological diagnosis in 145 cases (39%) (Table 3). Of the 230 (61%) cases where the clinical diagnosis (or lack of one) did not match the histological diagnosis, in 136 instances (36%) this was not considered clinically significant. Examples include a histological diagnosed seborrhoeic keratosis having been diagnosed clinically as a benign naevus, or a squamous cell carcinoma having been diagnosed as a basal cell carcinoma. Sixty percent (78/131) of histologically confirmed malignant lesions had not been identified on the request form as being malignant: only two of 12 (17%) melanomas, 33 of 74 (45%) BCCs and 18 of 45 SCCs (57%) were diagnosed clinically.

Table 3. Histological diagnosis with number (%) of patients diagnosed clinically

Variables	Histology diagnosis	Diagnosed clinically
Naevus	74	40 (54%)
Basal cell carcinoma	74	33 (44.6%)
Seborrhoeic keratosis	54	13 (24%)
Squamous cell carcinoma/keratoacanthoma	45	18 (40%)
Solar keratosis	35	4 (11%)
Keratosis (type not specified)	26	2 (8%)
Papilloma	12	2 (17%)
Melanoma	12	2 (17%)
Dermatofibroma	7	3 (43%)
Haemangioma	5	1 (20%)

The specimen and/or request form were considered inadequate to make a histopathological diagnosis in 25 cases (6.7%) (Table 1).

There were 82 punch and incisional biopsies; of these there were no clinical details in 23% and in a further 22%, it was simply labelled 'lesion'. Despite the 2008 Australian/NZ Melanoma Guidelines, punch biopsy was used in 40% of lesions where

a melanoma was being considered clinically. Punch biopsy was used for 32.5% of lesions identified as pigmented and in a third of suspect non-melanoma skin cancer.

Discussion

Dermatosurgery is increasingly being performed by general practitioners. Whilst many GPs have up-skilled themselves technically by attending surgical skills workshops, little attention seems to have been devoted to the communication between doctor and pathologist.

Communication between the medical disciplines is an essential component of quality medical care. Few of us would regard an inter-specialist referral with no clinical information as being either appropriate or adequate. Despite this, practicing clinicians tend not to regard referral to specialist diagnostic services in the same light. This is particularly unfortunate as our colleagues in specialist diagnostic services often have no direct contact with the patient, and are therefore unable to obtain any clinical information independently.

This study has shown that, in a third of cases, the histopathologist is not provided with any clinical information to help in the diagnostic process. There may be a number of reasons for this including both cognitive and procedural. The referring practitioner may be so confident of their clinical diagnosis they do not feel it is necessary to share their opinion. Alternatively they may not know what the lesion is and are embarrassed to show this. The request form may be completed by a surgical assistant, ignorant of the potential diagnoses. It may be pressure of time; most request forms were generated electronically and it requires an extra few minutes to fill the diagnostic/clinical sections. Most likely however, is a simple lack of appreciation of the value the pathologist places on good clinical information.

Skin lesions are removed/biopsied for a variety of concerns including that they are of health, cosmetic, or diagnostic concern (both current or potential), or because they are a nuisance to the patient. The biopsy technique used generally reflects the technical ability of the doctor, but it may also reflect the patient/doctor's degree of concern about the skin lesion. Unfortunately, in this study, a significant number of lesions were not sampled/removed in the most appropriate manner.

Punch biopsy is generally used for diagnostic purposes. It is simple and heals well, often without sutures. However, it only provides the pathologist with small, 2 to 5 mm tissue samples to work with and therefore, clinical information is essential.⁷⁻⁸ Of the 82 punch and incisional biopsies taken, there were unfortunately no clinical details in 23%, and a further 22% were simply labelled 'lesion'.

In addition, punch/shave biopsies may not be appropriate in certain circumstances.⁸ In a study of 2470 patients with melanoma, punch and shave biopsy significantly increased the odds of misdiagnosis by 16.6- and 2.6-fold respectively, compared to excisional biopsy. Moreover, punch biopsy increased the risk of a misdiagnosis with adverse outcome by 20-fold ($p < 0.001$). The 2008 Australian/New Zealand guidelines recommend complete excision of suspect pigmented lesions with a 2 mm margin.⁶ This audit showed that 40% of lesions where a melanoma was considered, and 32.5% of lesions identified as pigmented lesions, and therefore potentially a melanoma, were punch biopsied. Some of this may reflect the regional public plastic

surgery service's request for histological confirmation of non-melanoma skin cancer, prior to allocating priority for first specialist assessment.

As surgical techniques go, the shave biopsy appears very simple, although it actually requires a certain degree of understanding of cutaneous pathology to perform correctly. Whilst it may be appropriate for the removal of benign lesions such as papillomatous melanocytic naevi, skin tags and seborrhoeic keratoses, it is not the most appropriate technique for pigmented lesions where there is diagnostic concern. This study shows that 16% of shave specimens were inadequate to make a histopathological diagnosis compared to only 1.7% of lesions that had been sampled by excision.

Conclusion

This audit shows that in over a third of histology requests, diagnostic clinical information was absent. Sixty percent of histologically confirmed malignant lesions had not been identified on the request form as being malignant, including 87% of melanomas, 55% of BCCs and 43% of SCCs. The specimen was inadequate to make a histopathological diagnosis in 6.7% of cases. Finally, 40% of lesions suspected of being a melanoma were sampled by punch biopsy. Clearly there is room for improvement.

Quality medicine requires good communication. Pathologists need good clinical information to interpret subtle histological features. Whilst the size of the tissue sample may never satisfy the pathologist, small incisional punch biopsies of pigmented lesions, particularly if melanoma is being considered, may not be appropriate. Shave excisions, whilst simple, are often suboptimal.

Competing interests: None.

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References:

1. Fleming MG. Pigmented lesion pathology: what you should expect from your pathologist, and what your pathologist should expect from you. *Clin Plast Surg.* 2010;37:1-20.
2. Nutt L, Zemlin AE, Erasmus RT. Incomplete laboratory request forms: the extent and impact on critical results at a tertiary hospital in South Africa. *Ann Clin Biochem.* 2008;45:463-6.
3. Burnett L, Chesher D, Mudaliar Y. Improving the quality of information on pathology request forms. *Ann Clin Biochem.* 2004;41:53-6.
4. Plebani M. Exploring the iceberg of errors in laboratory medicine. *Clin Chim Acta.* 2009; 404: 16-23. Epub 2009 Mar 18.
5. Zemlin AE, Nutt L, Burgess LJ, et al. Potential for medical error: incorrectly completed request forms for thyroid function tests limit pathologists' advice to clinicians. *S Afr Med J.* 2009;99:668-71.
6. Clinical Practice Guidelines for the Management of Melanoma in Australia and New Zealand. The Cancer Council Australia and Australian Cancer Network, Sydney and New Zealand Guidelines Group, Wellington, 2008.

7. Leong AS, Braye S, Bhagwandeem B. Diagnostic 'errors' in anatomical pathology: relevance to Australian laboratories. *Pathology*. 2006;38:490-7.
8. Ng JC, Swain S, Dowling JP, et al. The impact of partial biopsy on histopathologic diagnosis of cutaneous melanoma: experience of an Australian tertiary referral service. *Arch Dermatol*. 2010;146:234-9.
9. Heal CF, Raasch BA, Buettner PG, Weedon D. Accuracy of clinical diagnosis of skin lesions. *Br J Dermatol*. 2008;159:661-8. Epub 2008 Jul 4.
10. Heal CF, Weedon D, Raasch BA, et al. Agreement between histological diagnosis of skin lesions by histopathologists and a dermato-histopathologist. *Int J Dermatol*. 2009;48:1366-9.



Non-melanoma skin cancers in New Zealand—a neglected problem

Nicholas D L Brougham, Elizabeth R Dennett, Swee T Tan

Abstract

Basal cell carcinoma (BCC) and squamous cell carcinoma (SCC) are the commonest types of non-melanoma skin cancer (NMSC). The incidence of NMSC has been increasing globally with Australia recording a 1.5-fold increase over the last 17 years. Given that Australia and New Zealand share similar latitude, sun exposure levels, population skin types, and other risk factors, it is conceivable that this increase has also occurred in New Zealand. However, the incidence of NMSC in New Zealand is unknown.

The cost of treating NMSC in New Zealand is estimated to be more than NZ\$50 million annually, based on extrapolated Australian data. In Australia, NMSC is the most costly burden to its healthcare system, and therefore the Australian Government has allocated resources to improve epidemiological research, and preventative efforts. Currently within New Zealand there is a lack of focus on the NMSC problem.

The absence of New Zealand data on the incidence of NMSC has hampered the development of consistent healthcare policies (including preventative measures), that achieve an integrated and sustainable service delivery. A critical analysis of this problem based on longitudinal data is now vitally important to address this neglected problem.

Non melanoma skin cancers (NMSC) are the most commonly diagnosed group of cancers globally. An estimated 2% of the Australian population are treated for NMSC each year¹ an incidence five times greater than the incidence of all other cancers combined.¹ Data on the number of patients treated annually in New Zealand for NMSC is currently unknown.

Since the 1970s the incidence of NMSC in predominantly Caucasian populations such as Canada,² the United States,³ Switzerland,⁴ and Australia⁵ has increased at an annual rate of 2–8%. Basal cell carcinoma (BCC) and squamous cell carcinoma (SCC) constitute the majority with BCC the most common malignancy in the world. BCC and SCC account for 65–75% and 15–25% of all cutaneous malignancies respectively.^{6–9} Rarer forms of NMSC include adenocarcinoma, sarcoma and Merkel cell carcinoma.^{10,11}

The fact that the New Zealand population consists predominantly of fair skinned Europeans with a high incidence of NMSC as well as Māori with a lower incidence, but potentially higher mortality, makes NMSC a prominent and relevant health issue affecting all sectors of our society.^{1,7,8,12–16}

The lack of data

A 1982 study in the upper central North Island of New Zealand by Freeman et al.¹⁷ shows that New Zealand has a high incidence of NMSC amongst Caucasians with reported incidence of 231 and 124 per 100,000 population for BCC and SCC respectively.¹⁷ This translates to approximately 12,000 new cases every year amongst the non-Māori, non-Pacific islander population of 3,100,000.¹⁸ The incidence of NMSC among Māori was much lower at 6/100,000.

The 2006 Australian study by Staples et al.¹ shows a significant increase in the incidence of NMSC within Australia over the last 17 years. The incidence of BCC increased from 657/100,000 in 1985, to 884/100,000 in 2002, an increase of 35%. The incidence of SCC has risen more dramatically from 166/100,000 in 1985, to 387/100,000 in 2002, an increase of 133%.

Given that Australia and New Zealand share a similar latitude, sun exposure levels, population skin types, and other risk factors, it is conceivable that this cumulative 1.5-fold increase in the incidence of SCC and BCC in Australia over 17 years may have also occurred within New Zealand.^{1,19-23} A study of the population in the Bay of Plenty in 1998 supports this assumption reporting an incidence of 1,120/100,000 for BCC and 598/100,000 for SCC.²⁴ This represents a total incidence of 1,718/100,000 for NMSC, one of the highest reported in the world and comparable to that reported in Australia by Staples et al.¹ and Buettner et al.²⁵

Assuming both the 1982¹⁷ and 1998²⁴ New Zealand studies were a representative sample of the entire population, this is an increase in incidence of nearly 385% for BCC and SCC over a 16-year period. This is a substantially greater increase than that reported by Staple et al.¹ in the Australian population over a similar period. It is unlikely that these two New Zealand studies accurately reflect the changing incidence of NMSC for the whole country. An increase in the incidence of NMSC comparable to recent Australian data is more likely. This illustrates the lack of longitudinal data on NMSC in New Zealand.

There is a lack of focus within New Zealand on generating up-to-date epidemiological data on NMSC. A 2005 report by Reeder²⁶ to the Skin Cancer Steering Committee responsible for developing New Zealand's skin cancer control programme, highlights this emphasising the need for developing "social, behavioural, environmental, psychological and health service research to determine, and evaluate better methods of preventing cancer." Within New Zealand it is estimated that epidemiological research is allocated only 6% of cancer research funding annually.^{26,27} Without accurate epidemiological data on the extent of the NMSC problem within New Zealand it will be very difficult to evaluate the effectiveness of any preventive measure.

In contrast, two recent reports from the Australian Cancer Council identifies NMSC as the most costly burden to the health system, and recognises the importance of having current epidemiological data by committing to conduct "regular surveys and other measures of national non-melanoma skin cancer," acknowledging this "will require support through ongoing funding,"^{28,29}

The unquantified cost of treatment

A health economy report in 2000 estimates that NMSC costs the New Zealand health care system NZ\$22 million per year, making it one of the most expensive cancers to treat.²⁴ The report which uses a variety of approaches, notes that this estimate is likely to be conservative with considerable difficulty encountered due to a lack of “available information on the prevalence of skin cancer.”²⁴

In Australia, from 2000 to 2001, NMSC is estimated to have cost the Australian health system A\$264 million (9% of total costs for cancer treatment).³⁰ Assuming New Zealand and Australia have the same incidence of NMSC, the estimated number of cases treated in New Zealand annually would amount to 80,000. If the cost of treatment was comparable between the two countries, NMSC would cost over NZ\$50 million per year.

Prevention

Adequate protection against ultraviolet radiation at any stage of a person’s life will reduce the risk of NMSC development.³¹⁻³⁴ A recent World Health Organization report concludes “that the encouragement of sun-protective behaviour is the most effective and cost effective public health measure to reduce the incidence of skin cancer.”³⁵

Australia is recognised to have the most extensive, comprehensive and sustainable skin cancer prevention programme in the world.³⁶ Currently within New Zealand there are limited resources allocated by District Health Boards (DHBs) to support any skin cancer prevention efforts.²⁶ This is acknowledged by Reeder who cites “successful collaboration with local Division of the New Zealand Cancer Society” as frequently being the principle instigator of campaigns that promote positive sun protection behaviour.²⁶ New Zealand needs to follow Australia’s example and commit to a “substantial increase in current expenditure on skin cancer.” Allocation of these funds to community-wide interventions, is seen as the most cost effective method of encouraging the use of sunscreen and other sun protective measures.²⁶

The challenge

In 1958 the New Zealand Cancer Registry abandoned mandatory reporting of BCC and SCC, because of incomplete reporting and a lack of resources to manage the large number of these cancers.^{17,37} Mandatory reporting is only required for malignant melanoma, and rarer forms of NMSC, such as Merkel cell carcinoma, atypical fibroxanthoma, and dermatofibrosarcoma protruberans. The visible location of BCC and SCC and their relatively low associated mortality has led to the assumption that most lesions can be simply treated.

The indolent nature of the majority of NMSC means many are treated non-surgically and generate no histology record. This is supported by a 2003 study in the United Kingdom which reports 13% of NMSC cases from general practitioners (GPs) have no matching histological records.³⁸ This practice is evidenced in New Zealand where a WaiMedCa survey of GPs in 1994 shows New Zealand GPs treated an estimated 0.48 new skin cancers per 100 patients.³⁹ This rate was used in O’Dea’s report that estimates 70,000 new cases of skin cancer each year, an order of magnitude greater

than previous estimates by other New Zealand studies that reported incidence based on histological reports from laboratories.^{17,24} Consequently any incidence generated by a retrospective descriptive epidemiological study based on pathology records will significantly underestimate the true incidence of NMSC.

Management of NMSC is also characterised by the large number of treatment providers including primary care and various specialties, DHB and private providers. This is quite different from other cancers and makes accurate data collection difficult. That is why the Australian study¹ relies on patient recall of NMSC treatment to obtain more reliable information.

The lack of accurate data on the incidence of BCC and SCC in New Zealand has prevented effective service planning and delivery. This is reflected by the implementation of a variety of unproven and inconsistent primary care models⁴⁰ for skin lesion removal by different DHBs within the country.

Tertiary services involved in the treatment of the most advanced forms of these lesions such as plastic surgery are being inundated by the increasing numbers of NMSC lesions requiring treatment that inhibits their ability to provide adequate service in other areas.⁴¹ Issues over the sustainability of the skin cancer service have been raised⁴⁰, and there is now a call for a multidisciplinary approach, with appropriate credentialing, and auditing encompassing a variety of treating specialties.

Whilst an epidemiological study to assess the size of the NMSC problem within New Zealand is now vitally important, there is no easy way of performing this accurately. A prospective study may be potentially expensive and time consuming, generating a significant workload.²⁴ Despite these difficulties, it is now important to carry out a properly designed survey, possibly similar to either the WaiMedCa survey for 1991–1992, now nearly 18 years old, or the recent Australian study by Staples et al.¹

Conclusion

Recent epidemiological data from Australia has shown a dramatic increase in the incidence of NMSC, particularly SCC over the last 17 years. Historically New Zealand has one of the highest incidences of NMSC in the world. Given that Australia and New Zealand share similar latitude, sun exposure levels, population skin types, and other risk factors, it is conceivable that this increase has also occurred in New Zealand. Australia is now addressing its growing NMSC problem through the allocation of appropriate resources to continuing epidemiological research and community-wide preventive measures. However, in New Zealand the current incidence of NMSC is unknown.

An epidemiological study within New Zealand is now needed to assess the size of the NMSC problem. If the incidence of NMSC is rising as rapidly in New Zealand as seen in Australia and other countries there is a need for an increase in appropriate resources to community-based preventative measures, the development and implementation of a consistent and sound national healthcare delivery model, and a commitment to following Australia's lead by committing to continued monitoring of the incidence of NMSC.

Competing interests: None.

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Acknowledgements: We thank Mr Des O’Dea and Ms Jyoti Rauniyar for their valuable input in the preparation of this manuscript. We are also grateful to the Reconstructive Plastic Surgery Research Foundation, the Wellington Regional Plastic Surgery Unit Research & Education Trust, the Henry Cotton Charitable Trust, and the New Zealand Cancer Society for funding aspects of this research.

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References:

1. Staples MP, Elwood M, Burton RC, et al. Non-melanoma skin cancer in Australia: the 2002 national survey and trends since 1985. *Med J Aust* 2006;184(1):6–10.
2. Gallagher RP, Ma B, McLean DI, et al. Trends in basal cell carcinoma, squamous cell carcinoma, and melanoma of the skin from 1973 through 1987. *J Am Acad Dermatol* 1990;23(3 Pt 1):413–21.
3. Hodgson NC. Merkel cell carcinoma: changing incidence trends. *J Surg Oncol* 2005;89(1):1–4.
4. Levi F, La Vecchia C, Te VC, Mezzanotte G. Descriptive epidemiology of skin cancer in the Swiss Canton of Vaud. *Int J Cancer* 1988;42(6):811–6.
5. Kaldor J, Shugg D, Young B, et al. Non-melanoma skin cancer: ten years of cancer-registry-based surveillance. *Int J Cancer* 1993;53(6):886–91.
6. Wermuth BM, Fajardo LF. Metastatic basal cell carcinoma. A review. *Arch Pathol* 1970;90(5):458–62.
7. Halder RM, Bridgeman-Shah S. Skin cancer in African Americans. *Cancer* 1995;75(2 Suppl):667–73.
8. Gloster HM, Jr., Neal K. Skin cancer in skin of color. *J Am Acad Dermatol* 2006;55(5):741–60; quiz 761–4.
9. Ashby MA, Smith J, Ainslie J, McEwan L. Treatment of nonmelanoma skin cancer at a large Australian center. *Cancer* 1989;63(9):1863–71.
10. Cook TF, Fosko SW. Unusual cutaneous malignancies. *Semin Cutan Med Surg* 1998;17(2):114–32.
11. Moody BR. Less Common Cutaneous Malignancies. In: American Academy of Dermatology 63rd Annual Meeting; 2005; New Orleans: Medscape; 2005.
12. Yakubu A, Mabogunje OA. Skin cancer in African albinos. *Acta Oncol* 1993;32(6):621–2.
13. Bykov VJ, Marcusson JA, Hemminki K. Effect of constitutional pigmentation on ultraviolet B-induced DNA damage in fair-skinned people. *J Invest Dermatol* 2000;114(1):40–3.
14. Rowe DE, Carroll RJ, Day CL, Jr. Prognostic factors for local recurrence, metastasis, and survival rates in squamous cell carcinoma of the skin, ear, and lip. Implications for treatment modality selection. *J Am Acad Dermatol* 1992;26(6):976–90.

15. Dhir A, Orengo I, Bruce S, et al. Basal cell carcinoma on the scalp of an Indian patient. *Dermatol Surg* 1995;21(3):247–50.
16. Pink B. 2001 Census Snapshot 1: Cultural Diversity. Wellington: Statistics New Zealand; 2002.
17. Freeman NR, Fairbrother GE, Rose RJ. Survey of skin cancer incidence in the Hamilton area. *N Z Med J* 1982;95(713):529–33.
18. 2001 Census of Population and Dwellings – Cultural Diversity Tables: Statistics New Zealand; 2009.
19. Holme SA, Malinovsky K, Roberts DL. Changing trends in non-melanoma skin cancer in South Wales, 1988–98. *Br J Dermatol* 2000;143(6):1224–9.
20. Hannuksela-Svahn A, Pukkala E, Karvonen J. Basal cell skin carcinoma and other nonmelanoma skin cancers in Finland from 1956 through 1995. *Arch Dermatol* 1999;135(7):781–6.
21. Kricger A, Armstrong BK, English DR. Sun exposure and non-melanocytic skin cancer. *Cancer Causes Control* 1994;5(4):367–92.
22. Armstrong BK, Kricger A. Epidemiology of sun exposure and skin cancer. *Cancer Surv* 1996;26:133–53.
23. Giles GG, Marks R, Foley P. Incidence of non-melanocytic skin cancer treated in Australia. *Br Med J (Clin Res Ed)* 1988;296(6614):13–7.
24. O'Dea D. A Report to the Cancer Society. The Costs of Skin Cancer to New Zealand: Wellington School of Medicine, University of Otago; 2000.
25. Buettner PG, Raasch BA. Incidence rates of skin cancer in Townsville, Australia. *Int J Cancer* 1998;78(5):587–93.
26. Reeder AI. Report to the Skin Cancer Steering Committee to inform development of the Skin Cancer Control Programme Plan 2005. Dunedin: Social and Behavioural Research in Cancer Group, University of Otago; 2004.
27. Primary Cancer Prevention: Report of the Primary Prevention Expert Working Group to the Cancer Control Steering Group. Wellington: Ministry of Health and The New Zealand Cancer Control Trust; 2003.
28. National Cancer Prevention Policy 2007–09. NSW: The Cancer Council Australia; 2007.
29. National Cancer Prevention Policy 2004–06. NSW: The Cancer Council Australia; 2004.
30. Health system expenditures on cancer and other neoplasms in Australia, 2000–01. Canberra: Australian Institute of Health and Welfare; 2005. Report No.: 22.
31. Stern RS, Weinstein MC, Baker SG. Risk reduction for nonmelanoma skin cancer with childhood sunscreen use. *Arch Dermatol* 1986;122(5):537–45.
32. Non-melanoma skin cancer: Guidelines for treatment and management in Australia: NHMRC; 2003. Report No.: 119.
33. World Health Organization sun protection and schools: How to make a difference. Geneva: World Health Organization; 2003.
34. Green A, Williams G, Neale R, et al. Daily sunscreen application and betacarotene supplementation in prevention of basal-cell and squamous-cell carcinomas of the skin: a randomised controlled trial. *Lancet* 1999;354(9180):723–9.
35. Stewart BS, Kleihues P, editors. World Health Organization, World Cancer Report. Lyon: IARC Press; 2003.
36. Montague M, Borland R, Sinclair C. Slip! Slop! Slap! and SunSmart, 1980–2000: Skin cancer control and 20 years of population-based campaigning. *Health Educ Behav* 2001;28(3):290–305.
37. Cancer: New Zealand Registrations and deaths: New Zealand Health Information Service; 2006.
38. Stefoski Mikeljevic J, Johnston C, Adamson PJ, et al. How complete has skin cancer registration been in the UK? A study from Yorkshire. *Eur J Cancer Prev* 2003;12(2):125–33.

39. McAvoy B, Davis P, Raymont A, Gribben B. The Waikato Medical Care (WaiMedCa) Survey 1992-1992. N Z Med J 1994;107(986).
40. Field B, Lespearance A, Bridges L. Skin Lesion Service Review Hutt Valley District Health Board: Central Region's Technical Advisory Service Limited; 2007.
41. Plastic Surgeons Hutt Hospital. Sustainability of NMSC service. In. Lower Hutt; 2008.



Changes to the eligibility to bill on Medicare in Australia: a threat to New Zealand's medical workforce?

Katie Elkin

Abstract

Previously, New Zealand citizens and those who studied medicine in New Zealand were subject to restrictions in terms of their eligibility to bill on Medicare in Australia. As of April 2010, those restrictions have been removed, making Australia an even more attractive destination for New Zealand doctors, particularly those who have completed their specialist training. At a time when the New Zealand health system can ill afford to lose more doctors overseas, this situation is of significant concern.

For the most part, New Zealanders and Australians have an unfettered ability to live and work in each other's country. Since 1994, this arrangement has been facilitated by the automatic granting of a Special Category Visa (SCV) to New Zealanders upon arrival in Australia.¹

While New Zealanders must still apply for (and be granted) permanent residence in Australia to enable them to access some social security benefits, apply for Australian citizenship, or sponsor others to migrate to Australia,² for many New Zealanders residing across the Tasman, such action never becomes necessary. However, until 2010, a further (and little known) restriction was imposed on New Zealand doctors wishing to practice in Australia.

In Australia, services provided in the public health system are funded by the government under the Medicare scheme. Treatment provided in a public hospital is fully subsidised by Medicare and so is free to any eligible patient. General practitioner and other specialist services provided in the community are also subsidised by Medicare, although a doctor is able to charge patients a fee in addition to the amount covered by the Medicare subsidy. However, in order to be able to "bill on Medicare" outside of the public hospital system the doctor providing the services must have a Medicare provider number.³

The section 19AB restrictions

In the mid-1990s, amidst concerns about an apparent oversupply, and unequal distribution, of medical practitioners, the Australian Government amended the Health Insurance Act 1973 to restrict the granting of Medicare provider numbers.⁴

Of particular importance to New Zealanders was the enactment of section 19AB, which came into force on 1 January 1997. Under that section, any doctor defined as a "former overseas medical student" or "overseas-trained doctor" was unable to bill on Medicare for 10 years from the latter of the date on which they became a permanent resident or citizen of Australia, or the date of first medical registration in that country.

The only way around this so-called “10 year moratorium” was for the doctor to agree to work in a designated District of Workforce Shortage (DWS), as defined by the Australian Department of Health and Ageing, or to be granted a personal exemption under the Act (for example, where the doctor was taking up an academic appointment).

Indeed, allowing exemptions for those working in a DWS was designed to influence the “*distribution of the medical workforce in rural and remote areas of Australia*”⁵ and is generally considered to have been successful in this aim, with the contribution of overseas-trained doctors “*fundamental to the delivery of health care in rural and remote areas*”.⁶

The issue for New Zealanders and for New Zealand medical schools was the way in which the critical terms were defined. Despite New Zealand medical schools being accredited by the Australian Medical Council (in the same way as Australian medical schools), those gaining a primary medical qualification from Auckland or Otago in New Zealand were designated as “overseas-trained doctors”, irrespective of residency and citizenship.⁷

The situation for New Zealanders studying at Australian medical schools was even more counter-intuitive, as illustrated by the case of Dr Mike Belich which was picked up by the mainstream media last year.⁸ Dr Belich is a New Zealand citizen who migrated to Australia at the age of 14. Having graduated from the University of New South Wales in 2002 and nearing the end of his vocational training as a general practitioner, he was refused a Medicare provider number on the basis of section 19AB. Having not been an Australian permanent resident or citizen at the time he enrolled at medical school (despite having become one since), Dr Belich was considered to be a “former overseas medical student”.

Due to family reasons Dr Belich was unable to relocate to a DWS so, instead, he filed a “human rights” challenge to the section 19AB restrictions in the Federal Court of New South Wales.⁹

Changes to section 19AB

Due to a recent change in the law, Dr Belich’s case now does not need to be heard. On 1 April 2010, the Health Insurance Amendment (New Zealand Overseas Trained Doctors) Act 2009 came into effect, amending section 19AB and associated sections of the principal Act.

The Act no longer refers to former overseas medical students. Instead, the term “foreign graduate of an accredited medical school” is used. An accredited medical school is one accredited by the AMC, either in Australia or New Zealand, and includes both Otago and Auckland Medical Schools.

A foreign graduate of such a school is a person who was not an Australian or New Zealand permanent resident or citizen at the time of first enrollment.¹⁰ As a result, no New Zealand or Australian permanent resident or citizen enrolling in a medical school in Australia or New Zealand will be subject to the 10-year moratorium upon graduation any longer, including:

- Australians who study in New Zealand but later return to Australia;
- New Zealanders who study in Australia and stay in, or later return to, Australia; and
- New Zealanders who study in New Zealand but later migrate to Australia.

However, a person who was not an Australian or New Zealand permanent resident or citizen at the time of enrolling in medical school or who graduates from a non-accredited medical school (including all medical schools outside of the two countries) will still be subject to the restrictions in section 19AB.

The Explanatory Memorandum to the Bill expressly recognises the pattern of overseas-trained doctors entering New Zealand and obtaining New Zealand citizenship as a pathway to residence and medical practice in Australia.⁵

Such doctors will continue to be covered by the moratorium for 10 years after the date of their first medical registration in Australia, provided Australian permanent residence or citizenship has also been obtained by that date.¹¹

While the restrictions in section 19AB have been relaxed, the conditions on Medicare billing imposed by section 19AA remain. In short, to bill on Medicare, all doctors registering to practice in Australia on or after 1 November 1996 must have completed specialist training, including as a general practitioner.¹²

Consequently, the changes to section 19AB will not impact directly on new graduates, but on those who have already completed specialist training or who are making decisions with that future in mind.

Likely impact of the changes

Clearly the changes to the legislation will have significant personal impact for doctors such as Dr Belich who now will not be subject to the moratorium. There are also those for whom the moratorium will end earlier than they had anticipated, either because it will cease to apply or due to a change in the date from which the 10-year period is calculated.¹¹ What is perhaps more controversial is the impact the changes are likely to have on the medical workforce on each side of the Tasman, particularly in New Zealand.

Australia—The Bill Digest for the Act predicts that the removal of New Zealanders from the ambit of the section 19AB restrictions is “*unlikely to have any significant effect*” on the number of doctors required to work in DWS.⁶ This conclusion is based on the “*negligible number of New Zealand medical students studying in Australian medical schools*”⁶ and so the relatively small number of New Zealanders previously able to be directed to DWS under section 19AB. However, it is not only New Zealanders who have studied medicine in Australia who were subject to the moratorium, but any New Zealander wishing to bill on Medicare who had been

registered to practice in Australia for less than 10 years or who had been a permanent resident of Australia for less than that period.

As about 25% of the Australian medical workforce completed their primary medical training outside of Australia, and around 8% of that number trained in New Zealand,¹³ the impact could be rather greater than is anticipated. Indeed, over the next few years, as the pressure on internship places grows, it is likely that fewer overseas students will be able to continue their medical education and career in Australia; leading to fewer doctors who can be directed to DWS.¹⁴

However, while the number of doctors who can be required to work in DWS is likely to decrease, the total number available for work may well increase due, in no small part, to the additional contribution of New Zealand-trained doctors.

New Zealand—With higher salaries and arguably more opportunities on offer elsewhere, New Zealand loses significant numbers of doctors every year.¹⁵ Due to its proximity and the ability to work, Australia is the destination of choice for many. Two years after graduation, retention rates for New Zealand-trained doctors are about 83%, dropping to between 76% and 78% over the next few years and levelling out at between 63% and 68% in years 8 to 12 after graduation.¹⁶

For three groups, the disincentive to relocate to (or remain in) Australia has been removed: New Zealanders trained at New Zealand medical schools, Australians trained at New Zealand medical schools, and New Zealanders trained at Australian medical schools. Such doctors will now be able to bill on Medicare immediately upon satisfying the training criteria in section 19AA of the Act. As expressed by Dr Tim Malloy of the New Zealand Rural General Practice Network, “[i]t’s one less barrier—or one more hurdle for us in retaining our own medical practitioners”.¹⁷

At present, many doctors move to Australia immediately after graduation in order to begin practice in the Australian health system as soon as possible. While many are motivated by the impact of higher salaries on the size of their student loans,¹⁵ others have sought to begin their 10-year moratorium so that they will be free from location restrictions soon after completing specialist training. It is quite possible that the removal of the moratorium will lead some new graduates to delay moving across the Tasman as the urgency to be registered and resident in Australia (so as to begin the 10-year period) decreases.

The bigger impact of the change in the legislation is likely to be those doctors in the years of specialist training or who have already qualified as specialists or general practitioners—the very doctors in whom New Zealand has made the most investment to date.

But what can be done to stem this tide? Clearly, it is not just a problem for the medical workforce, with around 550,000 New Zealanders citizens currently residing in Australia, many of them other skilled professionals.² But for medicine, the shortages are already being felt. For some time, overseas-trained doctors have been recruited to fill the gaps and now constitute around 38% of the New Zealand medical workforce. However, retention of overseas-trained doctors in New Zealand is very poor, with fewer than 50% remaining 1 year after initial registration, dropping to around 30% over the next couple of years; representing a less than ideal return on investment for this country.¹⁶

Again, this situation has an Australian dimension as research suggests that New Zealand is commonly used as a gateway for those wishing to ultimately end up practicing in Australia.¹⁸

Putting all of these pieces together creates a concerning picture for the New Zealand medical workforce and the public that rely on it for services. Already New Zealand has the highest proportion of overseas-trained doctors in the OECD¹⁸ so the changes to section 19AB will likely increase this percentage.

As more New Zealand-trained doctors are attracted to Australian shores, New Zealand will need to compensate somehow, probably by importing more and more overseas-trained doctors. While there is no evidence that overseas-trained doctors are of lesser skill or quality, there is inevitably a period of cultural, social, and systems adjustment needed for every such doctor, the degree of which will depend on the doctor's country of origin. Then there are the ethical issues inherent in recruiting doctors from countries such as Zimbabwe where the shortages in the health workforce are of an entirely different magnitude to our own.¹⁹

One area in which the migratory flow towards New Zealand may actually increase is with respect to medical students. If they know that it will not restrict their ability to later practice in Australia, more Australians may choose to study medicine at Otago or Auckland University. While this may seem like a positive outcome for New Zealand, it is really only a short-term gain as any students motivated to study here as a result of the section 19AB changes are likely to be doing so with the plan to return to Australia afterwards, amounting to still more New Zealand-trained doctors migrating across the Tasman.

Conclusion

While the changes to section 19AB are predicted to be insignificant from Australia's perspective, they have the potential to be significant for New Zealand. It remains to be seen exactly what the impact will be but the author predicts a net-loss to New Zealand of locally trained doctors. It is anticipated that the most influenced section of the medical workforce will be those training to work as, or who currently work as, specialists or general practitioners.

Curtailing the outflow of much needed medical talent from New Zealand is one of the biggest challenges facing our country today. The Australian changes are simply one more factor to be taken into account in formulating an appropriate policy response.

Competing interests: None.

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References:

1. Migration Act 1958 (Au), as amended in September 1994.

2. Australian Government Department of Immigration and Citizenship. Fact Sheet 17 – New Zealanders in Australia. Canberra, 2010. <http://www.immi.gov.au/media/fact-sheets/17nz.htm>
3. <http://www.medicareaustralia.gov.au/provider/index.jsp>
4. Hawthorne, L. Doctor shortages and their impact on the quality of medical care in Australia. *People and Place*. 2002;10:55–67.
5. Explanatory Memorandum to the Health Insurance Amendment (New Zealand Overseas Trained Doctors) Bill 2009.
6. Parliament of Australia Department of Parliamentary Services. Bills Digest for the Health Insurance Amendment (New Zealand Overseas Trained Doctors) Bill 2009.
7. Health Insurance Act 1973, s 19AB(7), pre-2010 amendment.
8. Cresswell, A. GP goes to Court fighting country duty. *The Australian*. 15 August 2009.
9. Needham, K. Kiwis fight job discrimination on home soil. *The Sydney Morning Herald*. 20 October 2009.
10. Health Insurance Act 1973, s 19AB(7).
11. Health Insurance Act 1973, s 19AB(1)(f).
12. Health Insurance Act 1973, s 19AA.
13. Australian Institute of Health and Welfare. *Medical labour force 2006*. Canberra, 2008.
14. Elkin KJ, Studdert, DM. Restricted career paths for overseas students graduating from Australian medical schools. *Med J Aust*. 2010;192:517–519.
15. Moore J, Gale J, Drew K, Simmers D. Student debt among junior doctors in New Zealand; Part 2: effects on intentions and workforce. *N Z Med J*. 2006;119(1229). <http://www.nzmj.com/journal/119-1229/1854/content.pdf>
16. Medical Council of New Zealand. *The New Zealand Medical Workforce in 2008*. Wellington, 2009.
17. Australian move may worsen doctor shortage. *Otago Daily Times*. 6 January 2010.
18. Zurn P, Dumont J-C. Health workforce and international migration: Can New Zealand compete? Health working paper No. 33. OECD, 2008.
19. Pond B, McPake B. The health migration crisis: The role of four organizations for economic cooperation and development countries. *Lancet*. 2006;367:1448–1455.



BCG sepsis following inadvertent intravenous BCG administration for the treatment of bladder cancer can be effectively cured with anti-tuberculosis medications

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Abstract

Aim To the best of our knowledge we are presenting the very first case of inadvertent intravascular administration of BCG and its successful treatment with anti-tuberculosis medications on a patient with superficial bladder cancer.

Methods A search of the English literature (PubMed/Medline) was performed concerning inadvertent BCG administration for bladder cancer by using the key words.

Results The patient was admitted to our hospital with high fever and chills a few hours after intravascular BCG administration. Chest CT showed bilateral infiltration of the lungs. Patient was placed on anti-tuberculosis treatment including isoniazid, rifampycin, ethambutol and methylprednisolone initially; and this treatment was adjusted according to his clinical course and liver function tests. By the end of the 4th week of hospitalisation patient was responded well with normalisation of his clinical status, liver function tests and a normal chest X-ray. Thereafter, he was discharged home on isoniazid, ethambutol for 6 months, streptomycin, cycloserine-C and ofloxacin for 2 months, methylprednisolone which was stopped eventually after dose reduction. On follow-up at 6th month after discharge from the hospital, he was fully recovered with normal chest X-ray and blood tests.

Conclusions Development of severe sepsis is inevitable following inadvertent intravascular BCG administration. Therefore, urologists should warn and inform not only their patients and families but also healthcare workers such as nurses regarding the route of administration of the BCG treatment for bladder cancer. Our experience also proved that such a serious complication can be successfully treated if promptly acted.

Intravesical BCG administration applied to prevent recurrence and progression of superficial bladder cancer is associated with many side effects and disseminated BCG infection is a rare but severe complication.¹ Herein, we presented and discussed the history, clinical features, treatment and outcome of our patient who developed *Mycobacterium bovis* (*M. bovis*) sepsis following inadvertent bacillus Calmette-Guérin (BCG) administration through intravenous route for the treatment of bladder cancer.

Case report

A 51-year-old male patient had a transurethral resection of papillary multiple bladder tumours (TUR-BT) and 40-mg of single dose intravesical mitomycin-C. Intravesical

BCG instillations were recommended for his pT1G3 transitional cell carcinoma (TCC). Unfortunately however, 81 mg of BCG (ImmuCyst[®]) was given intravenously at another hospital.

A flulike illness with nausea, fatigue, cough, shortness of breath and fever occurred hours after inadvertent intravenous BCG administration. The following day, his symptoms worsened and the patient was re-admitted to our hospital.

Respiratory sounds were weak on auscultation with palpable hepatomegaly, disseminated skin eruptions (abdomen and chest). Blood pressure was 90/60 mmHg, pulse rate was between 85–120/minute and body temperature was 38.1°C.

Complete blood count (CBC) and serum biochemistry values of the patient were presented in Table 1. Urine microscopy revealed white blood cells. Urine and blood cultures including *M. bovis* were all negative.

Table 1. Laboratory values during the course of treatment

Variables	Normal values	On Admission	Hospitalisation			6 th month
			2 nd day	2 nd week	4 th week	
BUN (mmol/L)	2.5–9.2	<i>21.8</i>	8.6	<i>16.1</i>	5.4	6.4
Creatinine (µmol/L)	53–114.9	<i>282.9</i>	97.2	70.7	88.4	97.2
Sodium (mmol/L)	136–145	137	137	135	138	137
Potassium (mmol/L)	3.5–5.1	3.8	3.6	3.5	4.0	4.1
Chloride (mmol/L)	98–107	111	108	<i>113</i>	104	105
Calcium (mmol/L)	2.2–2.5	<i>1.5</i>	<i>1.6</i>	<i>1.6</i>	2.3	2.2
AST (U/mL)	8–34	<i>445</i>	<i>202</i>	<i>158</i>	31	<i>43</i>
ALT (U/mL)	10–49	<i>324</i>	<i>147</i>	<i>130</i>	30	27
Albumin (g/L)	3.4–4.8	<i>3.0</i>	<i>2.9</i>	<i>2.2</i>	4.3	4.2
Bilirubin (direct) (µmol/L)	0.0–3.4	–	<i>111</i>	<i>35.9</i>	<i>10.3</i>	<i>5.1</i>
Bilirubin (total) (µmol/L)	≤17.1	–	<i>133</i>	<i>104</i>	<i>18.8</i>	13.7
LDH (U/L)	0–190	<i>721</i>	<i>485</i>	<i>930</i>	<i>221</i>	–
Hemoglobin (g/L)	13.5–18	<i>13.2</i>	<i>11.4</i>	<i>11.6</i>	<i>12.3</i>	14.4
Hematocrit (Proportion of 1.0)	0.42–0.50	<i>0.38</i>	<i>0.33</i>	<i>0.33</i>	<i>0.35</i>	0.44
Platelet (×10 ⁹ /L)	170–450	<i>47.7</i>	<i>10.5</i>	<i>106</i>	223	209

Note: Bold italic values correspond to elevated serum levels. BUN: blood urea nitrogen; AST: aspartate transaminase; ALT: alanine aminotransferase; LDH: lactate dehydrogenase.

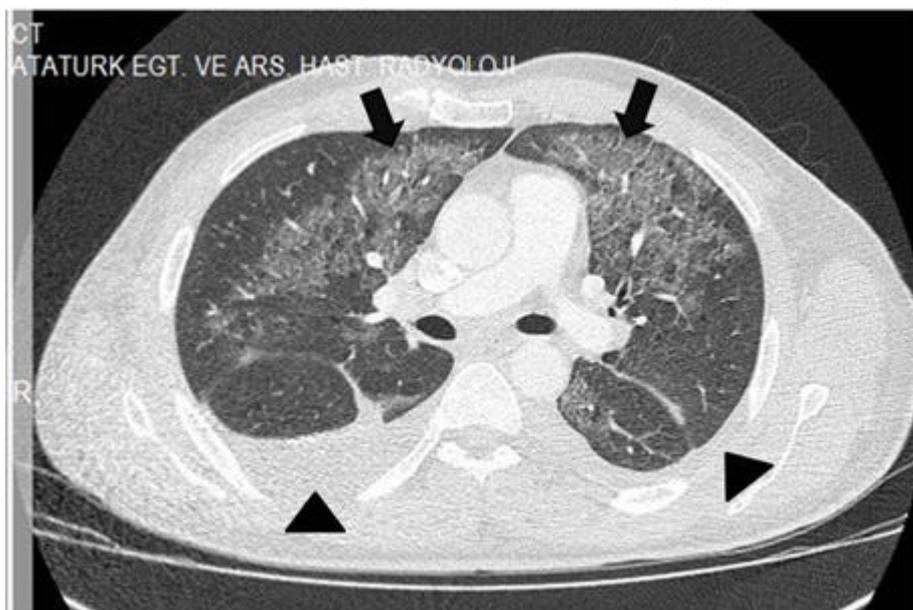
Abdominopelvic ultrasound revealed hepatomegaly. Chest computerised tomography (CT) demonstrated bilateral pleural effusions and infiltrations (Figure 1).

Patient was placed on isoniazid (1×300 mg/day, PO), rifampin (2×300 mg/day, PO), ethambutol (500 mg/day, PO, 3 times a week) and methylprednisolone 40 mg/day. On the 4th-day of hospitalisation lamivudine (100 mg, 1×1, PO) was initiated for his elevated hepatitis-B-DNA and steroid therapy. On the 3rd-day of hospitalisation, due to the elevated liver function tests, thrombocytopenia and leucopenia, rifampin dose was tapered down to 450 mg/day.

After the normalisation of renal function tests (6th day of hospitalisation), streptomycin (1 gr/day, IM) was initiated. At the end of the second week, due to the presence of dyspnoea, vomiting, fever and worsening of the chest X-ray, rifampin was

stopped due to the highly elevated serum liver enzymes and cycloserine-C (250 mg, 1×300/day, PO) and ofloxacin (2×400 mg/day, PO) were initiated.

Figure 1. Thorax computed tomography (CT) scan of the patient on the first week of hospitalisation showing bilateral septal infiltrations with groundglass appearance



Arrow: infiltrations; arrowheads: pleural effusions.

Supportive treatment including human albumin for hypoalbuminemia, thrombocyte suspensions for thrombocytopenia and fresh frozen plasma to prevent coagulation disorders were also given.

Clinical condition improved by the end of 4th-week and discharged home on isoniazid (300 mg, 1×1, PO), ethambutol (500 mg, 1×2, PO) for 6-months, streptomycin (1 gr, 1×1, IM, for 1 month), cycloserine-C (250 mg, 1×3, PO) and ofloxacin (200 mg, 2×2, PO) for 2 months, methylprednisolone (32 mg/day) and dose reduced by 4 mg every 4 days and eventually stopped. On follow-up at 6th month after discharge from the hospital, he was fully recovered with normal chest X-ray and blood tests (Table 1).

Discussion

Intravesical BCG instillations for the treatment of superficial bladder cancer are frequently associated with side effects. More serious complications include pneumonitis, hepatitis, sepsis and even death.^{1,2}

In the English literature (Pubmed/Medline), only 2 case reports exist related with inadvertent “intramuscular” injection of BCG.^{3,4} However, in our patient BCG was inadvertently administered intravenously.

In cases with intramuscular (IM) injections, severe and prolonged local reactions developed.^{3,4} Former was inadvertent intramuscular injection of BCG-vaccine into an already tuberculin-sensitive individual.³ However, latter was a 60-year-old male with bladder TCC. Following intramuscular administration, systemic symptoms including fever and headache developed with normal chest X-ray who was treated with anti-tuberculosis drugs successfully.⁴

Rare cases of disseminated *M. bovis* infection in patients with bladder cancer following intravesical BCG instillation have been reported.⁵⁻¹⁰ Although some authors proposed “hypersensitivity” as a cause of symptoms due to the presence of *M. bovis* antigens in the presence of histologic noncaseating granulomas with negative cultures in addition to rapidity and completeness of the response to short term (6 weeks) of therapy^{9,11-14} others debated that these granulomatous lesions might result due to hematogenous spread of BCG bacteria.⁵

Gonzalez et al classified the clinical course of BCG-related disease occurring after intravesical instillation as early and late presentation diseases in their literature review.⁹ They suggested rapid and complete response to therapy resulting from host immunity that becomes effective when the treatment is initiated for relatively low-grade virulence *M. bovis*.⁹ They summarised the clinical courses of 20 patients who developed early-presentation disseminated BCG infection or hypersensitivity.

In our patient, due to the presence of chest CT and X-ray findings (Figure 1), elevated serum liver enzymes, elevated BUN/creatinine, hepatomegaly and thrombocytopenia we assumed our patient had pneumonitis, hepatitis, bone marrow involvement and renal failure. Gonzalez et al reported that miliary pattern or interstitial infiltrates was present on chest X-rays in almost half of the patients whereas in some patients chest X-rays were within normal limits.⁹ Gonzalez et al reported the interval between the first instillation and the onset of symptoms as between <1 hour and 22 weeks.⁹

In our patient, symptoms occurred few hours after intravenous BCG administration. Although Gonzalez et al obtained tissue biopsies (liver, lung, bone marrow), no granuloma was detected in some biopsies in their series. Similarly, *M. bovis* was only detected in 25% of the urine and blood samples in these patients. A variety of stains could be used however none might detect any microorganisms.⁵ We did not take any tissue biopsies for microscopic investigation from our patient. Urine and blood cultures for *M. bovis* were negative. Factors affecting the culture results depend on the number of microorganisms present, handling of the biopsied tissues, culture technique and the need for highly sensitive and specific molecular techniques in order to isolate the mycobacteria.⁹

Late presentation disease has been also suggested after intravesical BCG instillation with a mean interval between instillation of BCG and onset of symptoms reported to be 15.7 months.⁹ These patients seem to have a tendency of developing disease particularly localised to the genitourinary tract without systemic symptoms or affect sites such as the spine or bones.^{6,9} Generally, laboratory or radiographic findings are normal.⁹ Therefore, long term close follow-up has been recommended to our patient.

The recommended treatment for disseminated BCG disease includes a combination of antituberculous medications with the exception of pyrazinamide, to which BCG is resistant.^{5,9} The following agents are used in different combinations for the treatment

of symptoms in these patients: isoniazid, rifampin, ethambutol, streptomycin, erythromycin, levofloxacin, ofloxacin, cycloserine, steroids and plasmapheresis.⁵⁻¹⁰ The length of the treatment may be as short as two weeks in cases of mild symptoms or as long as 6 to 9 months for severe disseminated disease.⁵

Addition of corticosteroids to the antituberculous therapy is also suggested in severe cases in preventing hypersensitivity reaction.⁵ Inclusion of antibiotics such as ofloxacin have also been suggested for the treatment of disseminated BCG infection in order to decrease the incidence of moderate to severe adverse events with BCG immunotherapy.¹⁵ Gonzalez et al reported that out of 20 patients with disseminated BCG infection, 15 patients (75%) recovered whereas in 5 patients (25%) died despite therapy.⁹ Therefore, the risk of death is very high in patients with BCG sepsis.

Recent research and studies have suggested some new agents and drugs which might be used in the treatment of disseminated tuberculosis. Tumour necrosis factor (TNF) has been suggested to play an important role in the regulation of chronic inflammatory diseases particularly in the host immune system against tuberculosis. Infliximab and etanercept are antagonists of TNF and have been suggested to inhibit TNF thereby used in the treatment of disseminated tuberculosis which warrants further research.¹⁶

Recently, it has been shown that Rifacinna[®] provides excellent *in vitro* activity against *Mycobacterium tuberculosis* and *Mycobacterium avium* complex (MAC) strains. Single daily dose of 10 mg/kg was demonstrated to provide complete eradication of mycobacteria in experimental generalised tuberculosis with good tolerability and safety profile.¹⁷ The efficacy of the presently used BCG vaccine against active tuberculosis in adults has been challenged and recently new live and attenuated strains of *M. tuberculosis*, improved recombinant BCG strains and subunit vaccines have been tested in preclinical animal models with promising results.¹⁸

In conclusion, due to the intravenously administration of BCG, we regard our case as BCG sepsis with multiorgan involvement. With prompt and proper treatment recovery is possible. Healthcare personnel and patients should be clearly informed about how to administer BCG when they decide to receive such treatment. They should re-admit in case systematic symptoms develop even after proper instillations.

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References:

1. Lamm DL, van der Meijden APM, Morales A, et al. Incidence and treatment of complications of Bacillus Calmette Guerin intravesical therapy in superficial bladder cancer. *J Urol* 1992;147:596–600.
2. Leebeek FWG, Ouwendijk RJ, Kolk AHJ, et al. Granulomatous hepatitis caused by Bacillus Calmette-Guerin (BCG) infection after BCG bladder instillation. *Gut* 1996;38:616–618.
3. Pasteur MC, Hall DR. The effects of inadvertent intramuscular injection of BCG vaccine. *Scand J Infect Dis*. 2001;33(6):473–4.
4. Yarmohammadi A, Ahmadiania H, Abolbashari M, et al. Results of inadvertent administration of bacillus Calmette-Guerin for treatment of transitional cell carcinoma of bladder. *Urol J*. 2007;4(2):121–2.
5. Nadasy KA, Patel RS, Emmett M, et al. Four cases of disseminated Mycobacterium bovis infection following intravesical BCG instillation for treatment of bladder carcinoma. *Southern Medical Journal* 2008;101:91–95.
6. Nemeth J, Stoiser B, Winkler HM, et al. Bone marrow infection with bacillus Calmette-Guérin (BCG) after intravesical immunotherapy. *Wien Klin Wochenschr* 2008;120(3–4):121–123.
7. Ozbakkaloglu B, Tunger O, Surucuoglu S, et al. Granulomatous hepatitis following intravesical Bacillus Calmette-Guerin therapy. *Int Urol Nephrol* 1999;31(1):49–53.
8. Viillard JF, Denis D, Texier-Maugein J, et al. Disseminated infection after Bacille Calmette-Guerin instillation for treatment of bladder carcinoma. *Clin Infect Dis* 1999;29:451.
9. Gonzalez OY, Musher DM, Brar I, et al. Spectrum of Bacille Calmette-Guerin (BCG) infection after intravesical BCG immunotherapy. *Clin Infect Dis* 2003;36:140–148.
10. Garcia JE, Thiel DD, Broderick GA. BCG pyelonephritis following intravesical therapy for transitional cell carcinoma. *Can J Urol* 2007;14(2):3523–5.
11. Molina JM, Rabian C, D'Agay MF, et al. Hypersensitivity systemic reaction following intravesical bacillus Calmette-Guerin: successful treatment with steroids. *J Urol* 1992;147:695–7.
12. Smith RL, Alexander RF, Aranda CP. Pulmonary granulomata: a complication of intravesical administration of bacillus Calmette-Guerin for superficial bladder carcinoma. *Cancer* 1993;71:1846–7.
13. DeHertogh D, Fierer E, Orell JA. Hypersensitivity reaction to bacillus Calmette-Guerin treated with plasmapheresis. *Am J Med* 1989;86:343–4.
14. DeHaven JI, Traynellis C, Riggs DR, et al. Antibiotic and steroid therapy of massive systemic bacillus Calmette-Guerin toxicity. *J Urol* 1992;147:738–42.
15. Colombel M, Saint F, Chopin D, et al. The effect of ofloxacin on bacillus calmette-guerin induced toxicity in patients with superficial bladder cancer: results of a randomized, prospective, double-blind, placebo controlled, multicenter study. *J Urol* 2006;176:935–939.
16. Kim JY, Park JN, Lee JG, et al. The use of etanercept in a patient with disseminated tuberculosis. *Rheumatol Int*. 2009;29(11):1377–80.
17. Velichka D, Ivana A, Haruaki T, et al. Experimental and clinical studies on Rifacinna--the new effective antituberculous drug (review). *Recent Pat Antiinfect Drug Discov*. 2010;5(1):76–90.
18. Delogu G, Fadda G. The quest for a new vaccine against tuberculosis. *J Infect Dev Ctries*. 2009;3(1):5–15.



Isolated melanoma metastasis to stomach with possible regressed primary lesion: the importance of pursuing solitary melanoma metastases

James D McKay, Alf Deacon

Abstract

This is a case of a 60-year-old man who presented with anaemia and was subsequently diagnosed with a solitary 5cm malignant melanoma metastasis of the gastric fundus. No primary lesion was identified. After surgical resection he is alive at 5 years follow-up, adding weight to the notion that solitary melanoma metastases should be aggressively pursued, as long-term survival is possible.

Case report

Mr C is a 60-year-old man who presented with anaemia. Gastroscopy revealed a smooth vascular tumour arising from the anterior aspect of the body of the stomach. It was noted to be 'unusual' in appearance and was thought to be a stromal tumour rather than an adenocarcinoma. A subsequent CT scan revealed thickening of the posterior aspect of the gastric fundus, normal serosal surface and two prominent locoregional lymph nodes, and histology from biopsies at gastroscopy was inconclusive, with no confirmed answer even after tertiary centre review.

The possibilities were a high grade stromal tumour with the differential being melanoma. Mr C subsequently underwent a laparotomy which revealed a 5cm tumour in the gastric fundus, two nodes adjacent to the gastrophrenic ligament and a total gastrectomy with roux-en-Y reconstruction was performed.

Histology on the resected tumour confirmed malignant melanoma with no lymph node involvement in 22 nodes. Further clinical review by a dermatologist, otolaryngologist and optometrist failed to find a primary melanoma. He was followed up 6-monthly with liver ultrasound and annual CT scans, and is alive and well at 5 years after diagnosis.

Discussion

Melanoma accounts for 1–3% of malignant tumours¹ and is one of the most common malignancies to metastasise to the gastrointestinal (GI) tract;² third only to adenocarcinoma of the kidneys and squamous cell carcinoma of the cervix.³ The issue of whether a melanoma in the GI tract (in this case the stomach) is primary or secondary has been raised when there is lack of a skin lesion, as with Mr C. The vast majority of GI melanomas are metastatic from a cutaneous primary; although it seems primary melanomas can also arise from the mucosal epithelial lining of the GI tract.⁴

Jelinic et al⁵ presented a case of a 54-year-old man who was diagnosed with a gastric melanoma which subsequently widely metastasised causing his death. No primary

cutaneous lesion was found. This was described as a primary gastric melanoma, a possible rare site of tumour.

Lagoudianakis et al¹ presented a case of a man with an ulcerated submucosal mass in the gastric antrum, histologically proven to be melanoma, with no clinical primary lesion found elsewhere. At the time their publication their case was only the fourth primary gastric melanoma ever published.

In contrast, High et al⁶ describes the concept of a completely regressed primary cutaneous malignant melanoma with visceral metastases, reporting five such cases. They described this as a consideration in cases like Mr C when no skin lesion is found, rather than describing them as primary GI tumours.

Whether Mr C is a case of a primary gastric melanoma or a GI metastasis of a regressed cutaneous primary is unknown, but both are extremely uncommon.

Mr C is the first known published New Zealand case of a gastric melanoma (metastatic or primary) still alive after 5 years post surgical resection. This adds weight to the notion that solitary metastases from melanoma should be aggressively pursued, as long-term survival is possible.

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References:

1. Lagoudianakis EE, Genetzakis M, Tsekouras DK, et al. Primary gastric melanoma: a case report. *World J Gastroenterol.* 2006;12:4425–7.
2. Liang KV, Sanderson SO, Nowakowski GS, Arora AS. Metastatic malignant melanoma of the gastrointestinal tract. *Mayo Clin Proc.* 2006;81:511–6.
3. Byrd BF, Morton CE 3rd. Malignant melanoma metastatic to the gastrointestinal tract from an occult primary tumour. *South Med J.* 1978;71:1036–8.
4. Schuchter LM, Green R, Fraker D. Primary and metastatic diseases in malignant melanoma of the gastrointestinal tract. *Cur Op Onc.* 2000;12:181–5.
5. Jelinic Z, Javic-Razumovic J, Petrovic I, et al. Primary malignant melanoma of the stomach. *Tumori.* 2005;9:201–3.
6. High WA, Stewart D, Wilbers CR, et al. Completely regressed primary cutaneous malignant melanoma with nodal and/or visceral metastases: a report of 5 cases and assessment of the literature and diagnostic criteria. *J Am Aca Derm.* 2005;53:89–100.



A case of a testosterone-secreting oncocytic adrenocortical carcinoma

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Abstract

Oncocytic neoplasms are most rarely found in the adrenal gland. They are usually benign and non-functioning. We present a case of a testosterone-secreting oncocytic adrenocortical carcinoma in a 19-year-old female who presented with acne, hirsutism and irregular menses. Clinical investigations revealed an elevated testosterone and DHEA-S and a 4×5 cm left adrenal mass. The tumour was successfully excised. The histology showed the tumour to be comprised of oncocytic cells with granular, eosinophilic cytoplasm, features consistent with an oncocytic carcinoma. This is the first case presented of a testosterone-secreting oncocytic adrenocortical carcinoma.

Oncocytomas are neoplasms that are characterised histologically by the appearance of epithelial cells with an abundant eosinophilic, granular cytoplasm. Most oncocytic neoplasms are non-functioning, however there have been seven reported cases of functioning oncocytic adrenocortical neoplasms.^{2,5-7,9,10} We report a case of a functioning oncocytic adrenocortical carcinoma in a 19-year-old female.

Case report

A 19-year-old female presented with recent onset hirsutism, acne affecting the face and shoulders, and irregular menses after stopping the oral contraceptive pill.

Biochemistry revealed an elevated testosterone of 12 nmol/L (0.5–2.7) and DHEA-S of 17.5 µmol/L (0.5–12). A dexamethasone suppression test revealed a 0800 plasma cortisol of 108 nmol/L (<50 nmol/L) indicating incomplete suppression. She had normal post synacthen 17-hydroxyprogesterone, electrolytes, creatinine, liver function tests, urinary catecholamines and preoperative 24-hour urinary cortisol level.

Abdominal ultrasound followed by CT scan, revealed a five-by-four centimetre left adrenal mass with central necrosis but no evidence of invasion into surrounding structures or lymphadenopathy.

The patient underwent a laparoscopic converted to open left adrenalectomy. There was no evidence of invasion into surrounding structures. Postoperative recovery was uneventful.

Six weeks postoperatively, the serum testosterone and DHEA-S levels had both returned to normal and she reported improvements in her acne, reduced hair growth and the return of regular menses. Follow-up chest/abdomen/pelvis CT scans performed at 3 and 9 months postoperatively showed no evidence of tumour recurrence. A repeat dexamethasone suppression test was also normal 9 months post discharge.

The mass weighed 67 g. Histological findings included oncocytic cells with granular, eosinophilic cytoplasm, highly pleomorphic nuclei and infrequent tumour mitoses. There was necrosis but no evidence of vascular or capsular invasion.

Based on the histological findings, the specimen fulfilled four of the Weiss criteria and therefore was a malignant adrenocortical neoplasm. The specimen was sent for a second opinion in Sydney, Australia and was confirmed to be an adrenal cortical carcinoma by means of advanced immunohistological staining using IGF2.

Discussion

Oncocytic neoplasms are most commonly found in the kidney, thyroid and salivary glands but are rare in other sites.

We have identified 42 cases of adrenal oncocytic neoplasms in the English literature comprising 18 cases of adrenocortical oncocytoma, 17 cases of oncocytic adrenocortical carcinoma and 7 cases of adrenocortical oncocytoma of unknown malignant potential (UMP).¹⁻¹⁰

Of the 17 cases of malignant oncocytic neoplasm arising in the adrenal gland, the average tumour size was 11.9 cm with a mean weight of 628.4 g. The average age at diagnosis was 52 years and there were 8 male cases and 9 female cases.

Of the 14 cases that included information regarding follow-up,^[2,3,4,10] 7 had no evidence of recurrence, 6 had documented recurrence at an average of 26 months following surgery, and 1 succumbed to their disease at 58 months.

There have been three previously reported cases of tumour functionality in oncocytic adrenocortical carcinomas. They included two cases of cortisol secretion alone^{2,9} and a case of co-secretion of cortisol and aldosterone.¹⁰ Our case is the first that we are aware of involving elevated serum testosterone and DHEA-S in an oncocytic adrenocortical carcinoma.

The main histological criteria adopted for the differentiation of adrenal oncocytic neoplasms is the Weiss criteria. Its accuracy has been debated recently and Lin et al have proposed a modified system for use in oncocytic neoplasms.¹

In summary, we have presented a unique case of a functioning oncocytic adrenocortical carcinoma associated with elevated testosterone and DHEA-S levels. To our knowledge this is the only such case reported to date in the English literature. Following surgery, the patient's clinical signs and symptoms resolved and the serum testosterone and DHEA-S levels returned to normal.

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Acknowledgements: We thank Professor David Stewart (Dunedin Hospital), Dr Steven Soule (Christchurch Hospital), and Dr Alistair Murray (Christchurch Hospital) for their assistance.

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References:

1. Lin BT-Y, Bonsib SM, Mierau GW, et al. Oncocytic adrenocortical neoplasms: A report of seven cases and review of the literature. *Am J Surg Pathol*. 1998;22:603–614.
2. Bisceglia M, Ludovico O, Mattia AD, et al. Adrenocortical Oncocytic Tumors: Report of 10 Cases and Review of the Literature. *Int J Surg Pathol*. 2004;12:231-243.
3. Song SY, Park S, Suh Y-L. Oncocytic adrenocortical carcinomas: A pathological and immunohistochemical study of four cases in comparison with conventional carcinomas. *Pathol Int*. 2004;54:603–610.
4. Hoang MP, Ayala AG, Albores-Saavedra J. Oncocytic adrenocortical carcinoma: a morphologic, immunohistochemical and ultrastructural study of four cases. *Mod Pathol*. 2002;15(9):973-978.
5. Xiao G-Q, Pertsemlidis DS, Unger PD. Functioning adrenocortical oncocytoma. A case report and review of the literature. *Ann Diagn Pathol*. 2005;9(5):295–297.
6. Logasundaram R, Parkinson C, Donaldson P, Coode PE, Co-secretion of testosterone and cortisol by a functional adrenocortical oncocytoma. *Histopathol*. 2007;51:418–420.
7. Gumy-Pause F, Bongiovanni M, Wildhaber B, et al. Adrenocortical oncocytoma in a child. *Pediatr Blood Cancer*. 2008;50:718–721.
8. Kurek R, Knobloch RV, Feek U, et al, Local recurrence of an oncocytic adrenocortical carcinoma with ovary metastasis. *J Urol*. 2001;166:985.
9. Golkowski F, Buziak-Bereza M, Huszno B, et al. The unique case of an adrenocortical malignant and functioning oncocytic tumour. *Exp Clin Endocrinol Diabetes*. 2007;115(6):401–404.
10. Ali A, Raphael SJ. Functional oncocytic adrenocortical carcinoma. *Endocr Pathol*. 2007;18(3):187–189.



Varicella-zoster virus pneumonia

Hsi-Che Shen, Tsu-Tuan Wu, Sheng-Hsiang Lin

Clinical

A 36-year-old man presented to the emergency department with fever and progressive skin rashes for 3 days, followed by cough and dyspnoea for 1 day. During this period he had contact with his 18-month-old son who had developed chickenpox. His medical history was unremarkable and he had no past history of varicella-zoster virus (VZV) infection or vaccination, and no risk factors for HIV. He was febrile (39°C) and skin examination revealed numerous characteristic varicella skin eruptions: polymorphic rashes with vesicles and pustules over face, trunk and extremities.

Biochemical studies showed elevated liver transaminase levels (aspartate aminotransferase [AST] 61 IU/L; alanine aminotransferase [ALT] 62 IU/L) and peripheral blood haemogram showed lymphocytosis (lymphocyte count 7296 cells/mm³). The initial chest radiograph (Figure 1) revealed diffuse nodular infiltrates in combination with a fine reticular pattern, compatible with interstitial pneumonitis. There was no evidence of encephalitis, nephritis or myocarditis.

The patient received intravenous acyclovir administration for the clinical diagnosis of VZV infection complicated with pneumonia. After hospitalisation, fever and respiratory symptoms gradually improved, and the skin eruptions became crusty. A follow-up chest radiograph 1 week later (Figure 2) revealed nearly total resolution of pulmonary infiltrates.

Figure 1. Initial chest radiograph



Figure 2. Follow-up chest radiograph 1 week later



Discussion

Pneumonia is a serious complication of VZV infection and occurs primarily in adults. In VZV infection, pregnancy, chronic lung disease, a history of smoking, an immunocompromised status, a close contact with chickenpox, a greater number of skin lesions and acute respiratory symptoms are associated with an increased risk of developing pneumonia.¹

Varicella pneumonia is usually a clinical diagnosis based on the presence of a typical rash associated with bilateral pulmonary infiltrates and microbiological confirmation is not usually necessary in typical cases.^{2,3} The most common radiological pattern observed is bilateral reticulonodular pattern followed by patchy airspace consolidations.^{2,3} After the introduction of acyclovir, the average fatality rate of VZV complicated by pneumonia decreased from 19% during the 1960s and 1970s to 6% during the 1980s and 1990s.¹

Performing chest radiographs in all adults with VZV infection and recognising the characteristic radiological features of varicella pneumonia, irrespective of whether or not they have respiratory symptoms, is important for the diagnosis and institution of early antiviral treatment to reduce the risk of a fatal outcome.^{1,2}

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References:

1. Mohsen AH, McKendrick M. Varicella pneumonia in adults. *Eur Respir J.* 2003;21:886–91.
2. Chiner E, Ballester I, Betloch I, et al. Varicella-zoster virus pneumonia in an adult population: has mortality decreased? *Scand J Infect Dis.* 2010;42:215–21.
3. Avnon LS, Smolikov A, Almog Y. Varicella pneumonia in southern Israel: clinical characteristics, diagnosis and therapeutic considerations. *Isr Med Assoc J.* 2009;11:261–5.



Obituary: Thomas Hocken

Published in N Z Med J. 1910;8(35):66.

The death of Dr. Hocken was briefly announced in our last issue. The following account of his career, the "Otago Daily Times" of May 18th, will be read with interest. It may be added that, from a professional point of view, he was loved and respected by all his colleagues; he always maintained a high standard of professional conduct, and was always ready to help his juniors:—

"It is with profound regret that we have to Dr. Hocken, whose name has for nearly half a century has been intimately associated with the progress of this city, passed away shortly after 8 o'clock last night. To his friends the news of will cause no surprise, for he had been in ill-health for several months, and his condition had been more or less critical for some weeks past. Both by them and by the public at large, with whom his name had been a household word, and by whom it had probably not been known that his illness was so grave, sincere sorrow will be expressed at the loss by Dunedin of a citizen like Dr. Hocken—one whose ideals of were so lofty and so inspiring—and at the realisation of the fact that his familiar, dapper figure will no more be seen in the streets of the city for which he entertained so great an affection.

Dr. Hocken has been for nearly half a century so essentially part and parcel of the life of our city that his familiar figure will be sadly missed. For all that long period his name has been the synonym for all that is kind, and gentle, and sympathetic. It needed but the unfolding to him of a tale of distress or misfortune to ensure an immediate response from his sympathetic nature.

He was indeed generous to a fault. Nor were his benefactions confined to such cases. His response was prompt and liberal to applications of all kinds where the public good was concerned. No worthy object was refused help if he were appealed to on its behalf, and he was never weary himself of taking his full share of active work in the promotion of worthy subjects.

"Dr. Hocken, who was born at Stamford on 14th January, 1836, studied for his profession at Durham University and at Dublin, and gained his diploma as a member of the Royal College of Surgeons of England in 1860. Subsequently he was for about two years surgeon on board the steamer Great Britain, well known as a passenger vessel between England and Australian ports.

It was in 1862 that he settled in Dunedin, and commenced the practice of his profession. " As the community grew his practice extended, and his material interests flourished. Ever recognising the duties of citizenship, he undertook many public duties. For 22 years he held the important post of coroner for the city, and discharged the duties of the this position with the greatest credit. He became one of the honorary surgeons with which his services were highly valued, and in the more important scholastic world was appointed the first lecturer on surgery to the Otago University on the establishment of that institution. Though his official connection with its teaching

staff ended many years ago, Dr. Hocken maintained a long and useful connection with the University by virtue of his appointment to a seat on the council.

In 1883 he was nominated by the Government as a life member of the governing body of the institution, and a few weeks ago, on the position of vice-chancellor becoming vacant through the elevation of Mr. Jas. Allen, M.P., to the chancellorship in succession to Judge Williams, Dr. Hocken was elected vice-chancellor—an honour at once well deserved by the recipient and delighted in, in its bestowal, by the council as a whole,

Dr. Hocken proved a most valuable worker in connection with the Otago Institute, and on three separate occasions his services were acknowledged by his election to the office of president.

In social work Dr. Hocken was ever active. At all times he took a very keen interest in philanthropic enterprises, and was a keen supporter of those most deserving social organisations—the Patients' and Prisoners' Aid Society, Society for the Prevention of Cruelty to Animals, and for many years he interested himself warmly in the affairs of St. Paul's Church and pro-Cathedral, and was one of the steady collectors of funds for the work of the church and for the cathedral, which some day it is intended shall add grace and dignity to the prominent site above the Octagon on which the present building stands.

"With one particular form of activity Dr. Hocken's name will be permanently associated in this community. He devoted himself indefatigably to the collection of manuscripts, maps, plans, pictures and all descriptions of literature relating to the early history of New Zealand. His energies were directed to this end for many years, and the result of his arduous labours is seen in the magnificent collection which, generously conferred by him upon the people of the dominion, is now permanently housed in the addition to the Otago Museum known as the Hocken Wing.

So much has been justly said and written concerning that noble gift, that it is unnecessary here to do more than mention the fact—the gift is in itself an enduring memorial of him. As a consistent contributor to the 'Transactions of the New Zealand Institute' and the records of the Australasian Association for the Advancement of Science of many papers of historic and general interest, Dr. Hocken achieved a merited fame.

As an author Dr. Hocken published in 1895 his valuable 'Contributions to the Early History of New Zealand,' which will always remain a standard authority upon events relating to the colonisation of the dominion. In 1903 he visited the Old Land to collect materials for a fuller edition of his work, while a little later he completed the publication of his 'Bibliography of New Zealand'—a work upon which he was engaged for many years, and which represents a notable monument of his industry, not only by reason of the comprehensiveness of the bibliography, but also by reason of the interesting annotations that are incorporated in it.

In 1884 Dr. Hocken's contributions to science were recognised by his election to the Fellowship of the Linnaen Society."



Proceedings of the Waikato Clinical School Biannual Research Seminar, Wednesday 13 October 2010

Trends of Chlamydia infection and related complications in New Zealand, 1998-2008

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Aim: To compare trends in Chlamydia testing and detection with trends in hospital discharge rates of Chlamydia-related diseases in the upper north island of New Zealand during 1998-2008.

Methods: Analysis of time trends in regional Chlamydia testing and detection rates and regional age-specific hospital admission rates per 100,000 females for pelvic inflammatory disease, female infertility, ectopic pregnancy and per 100,000 males for male epididymo-orchitis.

Results: Laboratory Chlamydia testing volumes increased steadily, from a total of 3732 tests per 100,000 population in 1998 to 9801 tests per 100,000 population in 2008. The highest detection rates and greatest increase over time were noted amongst women aged 15-24 years, at 773 reported cases per 100,000 in 1998, increasing to 8819 cases per 100,000 in 2008. Over the same period, for women aged 15-24 years, the rate of hospital admissions for PID and Chlamydia-related pelvic infections declined during 1998 to 2004 but rose from 2005-2008, the rate of publicly funded infertility admissions fell and the ectopic pregnancy rate was unchanged. The age-specific rate for epididymo-orchitis admissions amongst 15-44 year old men remained stable.

Conclusion: Chlamydia testing volumes from the upper north island have trebled since 1998, as have reported rates of Chlamydia infection, whilst disease complication rates do not appear to have increased. Ecological data must be interpreted with caution. Nonetheless, current high levels of chlamydia testing and detection appear consistent with greater detection of prevalent asymptomatic infection.

Presenting tumour features of Waikato women with newly diagnosed breast cancer from 2005-2008

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The Waikato Breast Cancer Register (WBCR) was established in 2005 to audit all Waikato women diagnosed with breast cancer. The primary goal is to establish the nature of breast cancer presenting in a defined regional population to examine inequalities in presentation and outcome. The population has the highest regional

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population of Maori women in New Zealand enabling detailed
comparisons and analysis.



All women residing in the Waikato region at the time of diagnosis are eligible for WBCR after informed consent. Detailed data of mode of presentation (screening or symptomatic), ethnicity, diagnostic and surgical procedures undertaken, pathological findings, adjuvant treatments and follow up are prospectively collected.

From 2005-2008, 998/1008 (95%) eligible women consented for entry into the WBCR. The majority of patients (~80%) were of European origin with Maori women making up approximately 15%. Of the women diagnosed with breast cancer who were within the screening age, only 54% were screen-detected cancers. Maori and Pacific Islanders were less likely to present with a screen-detected cancer. Invasive cancers comprised 86% of the total. Maori and Pacific Islander women had larger tumours and a higher proportion of node positivity. They also had a higher proportion of Her 2 positive tumours.

Significant variation in breast cancer presentation by ethnicity occurs in the Waikato. The extent of this variation is likely to lead to significantly worse cancer outcomes for these ethnic groups.

8 is Great! Cognitive Outcome of Very Low Birth Weight Infants at age 8

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Infants born very early and very small are at increased risk for development problems. Infants born weighing less than 1250g, and discharged from Waikato Hospital New Born Unit, are routinely followed up at the Child Development Centre, at 1 & 2 years (age corrected for prematurity) and at age 4. The aims of this study were to: (1) assess the cognitive outcome of these children at 8 years of age and compare to the normative data on the WISC IV, and (2) determine the potential value of the preschool cognitive assessments in predicting school-age outcomes.

Sixty-one infants born, weighing less than 1250g, in 1998 and 1999 were identified for the study. Of this group 4 children had been previously identified with an Intellectual Disability (ID) so were excluded, a further 21 were excluded for a variety of reasons. Thirty-six children were included in the final analysis (59%). Twenty-one (58%) were male. The mean age was 100.65 months (8yr 4mths) and the mean birth weight was 892.04gms (range 510g – 1202g). The Wechsler Intelligence Scale for children, Fourth Edition (WISC IV) was used to assess cognitive ability. The sample was normally distributed and individual scores were placed with in a normal distribution for comparison (WISC IV, mean 100, SD 15).

The mean full scale cognitive score of the 36 children in the final analysis was within the average range, but substantially lower than the mean on the WISC IV. The mean FSIQ was 86 (SD 18) and ranged between 48 and 117. Seventeen percent of children

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Journal of the New Zealand Medical Association

were within the extremely low range (2 SD below the mean and in the range consistent with Intellectual Disability), 28% were 1 Standard Deviation below the mean and 53% were within the average range (+/- 1SD of the mean). One child achieved an above average score. A *T* test for dependent samples indicated no significant difference in cognitive scores between 4 and 8 years.

Overall, our sample of VLBW infants achieved substantially lower cognitive scores compared to normative data on the WISC IV. In-fact taking into account the children that were excluded due to ID, 28% of the children in the cohorts of 1998/1999 (N=40) had an intellectual disability. This is compared to 2.5% expected within the normal population. Furthermore, cognitive scores at the 4 year assessment were consistent with cognitive scores at 8 years suggesting the 4 year assessment may be an important indicator of later cognitive achievement and can provide information to support school entry. Further results, limitations and clinical significance will be discussed.

Acknowledgements: This study was supported by Summer Scholarships by Waikato Clinical School 2006-2008. We would further like to acknowledge the families who participated in our study.

Evaluation of the CoaguChek XS system & INR online for Warfarin Management at Pharmacy 547.

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Warfarin is an oral anti-coagulant used to reduce the risk of blood clots forming in high risk patients. Warfarin dose needs to be closely monitored by international normalised ratio (INR) blood tests. The current system in the Waikato involves patients having a venous blood sample collected at their local laboratory, with delayed results being sent to their general practice. The dose is then assessed and any changes are relayed over the phone. The CoaguChek XS is a hand held INR monitoring device which gives an instant INR result. INRonline is an online decision support software developed to manage warfarin dosing. A small number of general practices in New Zealand are using the CoaguChek XS and INRonline to monitor their warfarin patients in an anticoagulation management service (AMS).

Our aim was to demonstrate that CoaguChek XS & INRonline could be used by community pharmacy to provide warfarin management. We also wanted to compare the pharmacy model to the laboratory model and general practice AMS and collect participants satisfaction responses.

A pharmacy AMS was developed and data was collected over a six month period. The results showed that the pharmacy AMS increased time in therapeutic range from 55% to 76%. Patients attended the pharmacy AMS on time 92% of the time. 80% of patients believed the pharmacy AMS was better than their existing service.

This study was able to show that a pharmacy AMS could successfully manage warfarin patients. The results gathered compared favourably with existing systems.

THE NEW ZEALAND MEDICAL JOURNAL

Journal of the New Zealand Medical Association

This study was only conducted at one pharmacy and further studies will be needed to evaluate the system at a greater number of pharmacies.



Acknowledgements: Dr Paul Harper, INOnline, Bronwyn Sheppard, Roche Diagnostics NZ, Prof John Shaw, University of Auckland School of Pharmacy, Elizabeth Plant, Pharmaceutical Society of NZ.

The Waikato Virtual Lesion Clinic: better, sooner and more convenient

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Skin cancer is very common in New Zealand and hospital lesion clinics struggle with the volume of referrals received. This results in long waiting times for diagnostic assessment leading to delayed treatment. Health Waikato is managing to reduce waiting times for skin lesion assessment and treatment using a private teledermoscopy service.

We analysed patient flow through the new service and compared it to traditional assessment clinics. Of the first 100 patients referred to the service, 97% did not require a hospital appointment to establish the diagnosis. Waiting times were reduced by two thirds. Eighteen patients with skin cancers or suspicious lesions were placed straight onto surgical waiting lists. Surveyed patients have been highly satisfied and confident with the service.

Virtual lesion clinics can allow hospitals to keep up with burgeoning referrals while providing a better, quicker and more convenient service. The new service will potentially provide cost savings, as teledermoscopy assessments can be cheaper than traditional assessments.

Use of device therapy in the outpatient management of congestive cardiac failure

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Device therapy in patients with severe systolic heart failure (HF), including cardiac resynchronization therapy (CRT) and implantable cardioverter-defibrillators (ICD), improves survival and functional status in selected patients¹⁻⁵. This study aimed to assess the number of patients fulfilling criteria for device prescription, as outlined in the ACC/AHA/HRS 2008 guidelines⁶, in an outpatient cardiology clinic setting.

We ascertained the following data from 321 consecutive patients attending cardiology clinic during a one month period: Aetiology of HF, New York Heart Association (NYHA) Class, Left Ventricular Ejection Fraction (EF), QRS Duration and Prescription of CRT/ICD.

THE NEW ZEALAND MEDICAL JOURNAL



Journal of the New Zealand Medical Association

Fifty-seven (18%) had a diagnosis of HF documented; 22 (39%) had an $EF \leq 35\%$ and 4 (7%) had no EF measurement. Of those with $EF \leq 35\%$, 9 (41%) patients had NYHA Class I symptoms, 6 (27%) Class II symptoms, 3 (14%) Class III symptoms and 4 (18%) had no functional class documented. Five (23%) patients had an ischaemic aetiology. Eleven satisfied criteria for an ICD on primary prophylaxis basis, 9 of whom were ≤ 75 yrs old; of these a single patient with known ventricular tachycardia had an ICD. For those with NYHA Class 3 or more, 1 patient had a QRS duration of 178ms with atrial fibrillation.

From our sample of HF patients, we identified a significant number of patients who may benefit from device therapy for prophylaxis of sudden cardiac death but had not been referred. Continuing education for physicians on the criteria and availability of device therapy is essential.

References:

1. Cardiac resynchronization therapy for patients with left ventricular systolic dysfunction: a systematic review. *JAMA*. 2007 Jun 13;297(22):2502-14.
2. MADIT-I, *N Engl J Med* 1996; 335:1933.
3. MADIT-II, Investigators. *N Engl J Med* 2002 Mar 21;346(12):877-83.
4. MUSTT, *N Engl J Med* 1999 Dec 16;341(25):1882-90.
5. SCD-HeFT, *N Engl J Med* 2005; 352:225.
6. ACC/AHA/HRS 2008 Guidelines for Device-Based Therapy of Cardiac Rhythm Abnormalities: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *Circulation* 2008; 117:e350.

Long-term outcomes of patients with hypothyroidism: an analysis of CVD morbidity and mortality over a decade (1997-2006).

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Some overseas observational studies have shown an increased risk of cardiovascular morbidity and mortality in subjects with subclinical hypothyroidism¹⁻⁴. This study aimed to examine CVD morbidity and mortality in a New Zealand population aged 20 years or older, comparing people with normal thyroid function with people with subclinical and overt hypothyroidism over a decade (1997-2006) by age, gender, ethnicity and deprivation score.

We utilised laboratory data of thyroid function tests to establish links with cardiovascular outcomes from the National Minimum Data Set for hospital events and National Mortality Collection. Data were linked by national health index (NHI) number. We defined subclinical hypothyroidism as having a TSH from 5-10 mIU/L with normal thyroxine levels.

A total of 61,935 individuals were included in the survival analysis, of whom 56,491 were classified as normal, 3,185 as having subclinical hypothyroidism and 2,259 as having overt hypothyroidism. 4,882 individuals had evidence of a cardiovascular event. The estimated overall unadjusted CVD event rate was 14.7 per 1000 person-

THE NEW ZEALAND MEDICAL JOURNAL



Journal of the New Zealand Medical Association

years (95% CI = 14.3 to 15.1 per 1,000 person-years). When adjusted by age at entry in a Cox regression model, the rate of a CVD event was 15% higher in SCH and 36% higher in OH when controlled for gender, ethnicity and deprivation compared to normal thyroid function.

In this laboratory defined cohort, age, gender, ethnicity and deprivation were important factors in CVD event rates for individuals with hypothyroidism. CVD outcomes in patients within a tightly defined range of subclinical hypothyroidism have worse outcomes than euthyroid individuals. Whilst these differences are small they may have implications when deciding on treatment in general practice.

References:

1. Razvi S, Weaver JU, Vanderpump MP, Pearce SHS. The Incidence of Ischemic Heart Disease and Mortality in People with Subclinical Hypothyroidism: Reanalysis of the Wickham Survey Cohort. *J Clin Endocrinol Metab.* 2010;95(4):1734-40.
2. Razvi S, Shakoor A, Vanderpump M, Weaver JU, Pearce SHS. The Influence of Age on the Relationship between Subclinical Hypothyroidism and Ischemic Heart Disease: A Metaanalysis. *J Clin Endocrinol Metab.* 2008;93(8):2998-3007.
3. Singh S, Duggal J, Molnar J, Maldonado F, Barsano CP, Arora R. Impact of subclinical thyroid disorders on coronary heart disease, cardiovascular and all-cause mortality: A meta-analysis. *Int J Cardiol.* 2008;125(1):41-8.
4. Imaizumi M, Akahoshi M, Ichimaru S, Nakashima E, Hida A, Soda M, et al. Risk for Ischemic Heart Disease and All-Cause Mortality in Subclinical Hypothyroidism. *J Clin Endocrinol Metab.* 2004;89(7):3365-70.

Use of transient elastography for non-invasive monitoring of methotrexate induced liver fibrosis

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One of the long-term complications of methotrexate use is liver fibrosis. Transient elastography (FibroScan®, Echosens, Paris) is a non-invasive technique to detect liver fibrosis. Recent meta-analysis comparing transient elastography with liver biopsy has concluded that transient elastography (TE) has excellent diagnostic accuracy in detecting cirrhosis (AUROC of 99% with TE score >13kPa)¹. Transient elastography can also be used to exclude liver fibrosis in patients on methotrexate (negative predictive value 88% for TE score <7.1kPa)².

All patients in the dermatology department on methotrexate were offered transient elastography. Transient elastography scores were divided into no detectable fibrosis (0-7kPa), detectable fibrosis (7.1-13kPa) and cirrhosis (>13kPa). Patients with transient elastography scores of more than 13kPa were to be assessed by the Gastroenterology department and considered for liver biopsy.

132 patients underwent scanning. Of the 132 patients, 32 were unsuccessful due to obesity as accurate readings could not be obtained. Mean age was 52 and 56% were male. Psoriasis (59%) was the most common indication for methotrexate followed by

THE NEW ZEALAND MEDICAL JOURNAL



Journal of the New Zealand Medical Association

eczema (25%). Mean methotrexate dose was 14mg per week, median cumulative dose was 510 milligrams and median duration on methotrexate was 9 months. 85 patients (85%) had TE scores of less than 7.1kPa (repeat scanning in 1 year). 15 patients (15%) had TE scores between 7.1-13kPa (repeat scanning in 3-5 years). No patients had TE scores higher than 13kPa. There was a slight correlation with TE scores and cumulative dose (Pearson correlation 0.233, p-value 0.03).

We successfully determined minimal fibrosis in the majority of patients obviating the need for liver biopsy. Longitudinal data are needed to observe the reliability of this test long-term.

References:

1. Friedrich-Rust M, et al. Performance of transient elastography for the staging of liver fibrosis: A meta-analysis. *Gastroenterology* 2008;134:960-974.
2. Laharie D, et al. Diagnosis of liver fibrosis by transient elastography (FibroScan) and non-invasive methods in Crohn's disease patients treated with methotrexate. *Alimentary Pharmacology and Therapeutics* 2006; 23:1621-1628.

Breast cancer treatments for Waikato women with newly diagnosed breast cancer, 2005-2008

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Women in New Zealand face a 20% greater chance of dying from breast cancer compared to women in Australia^(1,2), and Maori women fare worse still. The Waikato Breast Cancer Register (WBCR) is a comprehensive regional population based database of breast cancer diagnosed since 2005. Using the WBCR, this analysis seeks to examine patterns of care in Waikato women overall and by ethnicity.

The database encompasses the breast cancer population from both screening and symptomatic presentations. Data is also collected relating to surgical procedures and adjuvant treatments including any chemotherapy, radiotherapy or endocrine therapies prescribed. From 2005-2008, information on 817 women with invasive cancer and 124 women with DCIS is reported

50% of patients with invasive tumours had breast conserving surgery (BCS) as a primary surgical procedure compared to 65% of patients with Ductal Carcinoma In situ. BCS rates were higher for smaller breast cancers at 64% for T1 tumours. Maori and Pacific Islander women tend to present with more advanced tumours leading to a higher proportion of mastectomies (>60% for both, compared with 47% for European) and requirement for full axillary dissection. Consequently, they were also more likely to require adjuvant chemotherapy. 45% of Maori and 67% of Pacific Islander women required chemotherapy compared to 36% of European women. 50% of women who had a mastectomy received adjuvant radiotherapy compared to just over 90% of women who had BCS. Of women with endocrine responsive invasive cancers, 90% received endocrine therapy.

THE NEW ZEALAND MEDICAL JOURNAL

Journal of the New Zealand Medical Association

Waikato women are receiving the appropriate treatment for their cancer stage. This also applies to Maori women who despite having worse prognosis tumours are also receiving the appropriate treatment.



References:

1. Armstrong W, Borman B. Breast cancer in New Zealand: trends, patterns and data quality. *N Z Med J* 1996;109:221-224.
2. Robson B, Purdie G, Cormack D. *Unequal Impact: Maori and Non-Maori Cancer Statistics 1996-2001*. Wellington: Ministry of Health; 2005.

Identifying Person-Specific Factors Associated With Health Change in an Intervention Programme for Chronic Pain

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A multi-disciplinary persistent pain programme (MDPPP, funded by ACC compensation scheme) has been developed using a holistic model of practice based on Health Change Process Theory. The psychometric instrument QEHS was developed out of this theory and is used to determine clients' health status and locus on the health change pathway. Both a total score and a patient profile is generated. Other validated psychometric measures used are Kessler 10 (K10, psychological distress), the Pain Self Efficacy Questionnaire (PSEQ, a subjective measure of function) and the Return to Activities of Daily Living Scale (RADL, assesses return to activities usual for the client).

Aims of the Study. 1: To examine the internal consistency, content and concurrent validity of the QEHS. 2: To identify person-specific factors related to degree of change occurring during a chronic pain intervention programme.

Method: Aim 1: 88 data sets prospectively gathered from 55 participants in MDPPP between 2008-2009 were used. Internal consistency of the QEHS was assessed using multivariate analysis (SPSS v17.0). Correlational analysis between QEHS Total Score, the individual components of the QEHS, and K10, PSEQ and RADL scores was used to explore concurrent and content validity. Aim 2: Two groups were identified as having either high or low change in QEHS Total Score between admission and discharge. Grounded theory was used to identify factors associated with programme success or poor outcome.

The QEHS Total Score was found to have high internal consistency with each of the subscales ($p < 0.01$ for all); Anxiety (-0.946), Self Worth (0.956), Motivation to Change (0.872), Awareness to Possibility of change (0.951), Identity (0.949) and Sustainability (0.954). QEHS Anxiety score correlated strongly with K10 score (0.477, $p < 0.01$), and QEHS Self-Worth score with PSEQ (0.403, $p < 0.01$). QEHS Motivation to Change was negatively correlated with both K10 and QEHS Anxiety scores (-0.429 and -0.887 respectively, $p < 0.01$). Themes identified in promoting

THE NEW ZEALAND MEDICAL JOURNAL



Journal of the New Zealand Medical Association
programme success were 'Length of time off work', 'Considering a return to work' and 'Full engagement and participation in the programme'; low change was associated with 'Fixation upon a return to pre-injury functioning', 'Aims to return to previous work/a form of work that is too strenuous' and 'Unable to maintain new techniques learnt during the MDPPP'.

The QEHS is a valid index of health status and of change in health. Person-specific factors are predictive of change in health status in a multidisciplinary pain management programme.



Is incidence of type 2 diabetes related to fruit and vegetable intake?

This meta-analysis reviews data from 6 studies involving more than 220,000 subjects. The pooled results shows that increased consumption of fruit, vegetables, or both showed no benefit in incidence of type 2 diabetes. However, 4 of the studies had data on the intake of green leafy vegetables and those with high intake of these had a significant reduction (14%) in the incidence of type 2 diabetes.

An editorial commentary commends the study and notes that the finding is independent of weight loss. He also points out that there are other relevant lifestyle modifications—e.g. decreased caloric intake—that will decrease the incidence of type 2 diabetes and its consequences.

BMJ 2010;341:c4229 & c4395.

Psychiatric morbidity and hazardous alcohol use in Australian doctors

The mental health of doctors is an important issue for their patients. This study involves a postal survey of 2999 doctors (including all major specialty groups, trainees and general practitioners) insured with an Australian medical insurance company.

The authors report that the personality trait of neuroticism was most strongly associated with psychiatric morbidity. In addition, work-related factors including experiencing a current medicolegal matter, not having had a holiday in the past year, and working 60 or more hours per week were also significantly associated with psychiatric morbidity.

Hazardous drinking of alcohol was found to be related to demographic and personality traits rather than work-related, and included being male, having an Australian medical degree, neuroticism and extroversion.

We would not be surprised if these findings were also representative on our side of the Tasman.

Med J Aust 2010;193:161–6.

Rosiglitazone and the US Food and Drug Administration (FDA)

When this drug was first introduced it was widely proclaimed that it was a great innovation as it was capable of lowering the glycated haemoglobin by 1% and would obviously be beneficial to those with type 2 diabetes.

But in 2007, a meta-analysis of controlled clinical trials found increases in the risk of myocardial infarction and a near-significant increased risk of death from cardiovascular causes when rosiglitazone was compared with placebo or with standard diabetes drugs.

Since then, GlaxoSmithKline (the makers) and the FDA have had a dialogue on whether or not this drug is safe. Currently the FDA is restricting access to rosiglitazone by requiring the drug sponsor to submit a Risk Evaluation and Mitigation Strategy.

Others suggest that the drug should be withdrawn, or probably never have been licensed.

N Engl J Med 2010;363:1489–91.

Risk of birth defects after the use of antiviral agents in the first trimester of pregnancy?

The antiviral agents acyclovir, valacyclovir, and famciclovir are used in the treatment of herpes simplex and zoster. Generally speaking they are regarded as efficacious and safe.

The authors of this Danish study review their safety in terms of birth defects when used in the first trimester. Their study is a population-based cohort study involving over 800,000 live-born infants. They found a 2.2% incidence of major birth defects in those whose mother had had antiviral treatment in the first trimester. However the incidence rate was 2.4% amongst those not exposed to the antiviral drugs. Somewhat reassuring, however an editorial points out that the data mainly relates to acyclovir, which was the drug used in 86% of cases.

JAMA 2010;304(8):859–66 & 905–6.

Another disappointment in the treatment of Alzheimer's disease

Amyloid- β plaques are regarded as the defining lesions in Alzheimer's disease and the drug company Eli Lilly have produced a drug, semagacestat, which blocks the enzyme γ -secretase, which helps to produce amyloid- β . However, preliminary data from two large trials involving more than 2600 patients are disappointing as no clinical improvement have been noted.

Furthermore the drug seems to increase the risk for skin cancer, presumably because γ -secretase also processes several other important proteins, including epidermal growth factor receptor—the loss of which causes skin cancer in animal models.

The trials have been halted.

Nature 2010;466:1031.



Sample, send, screen, survive—simple. Rotary Club-subsidised community trial points way to simple screen for bowel cancer risk

During late February 2008 personal invitations were mailed to 400 patients between 55 and 75 years who had no previous history of bowel cancer. They were invited to call personally at either the Riccarton or Redcliffs Medical Centre in Christchurch, New Zealand to buy a \$5 self-test kit and return stool test samples during the following month.

The Rotary Club of Riccarton (district 9970) subsidised the purchase of the Hemosure™ IFOBT kits and wanted to confirm whether this test method is acceptable to the general public of New Zealand. It is already widely used in Australia.

Of the 387 test kits that trial participants bought, 221 (92.86%) were returned with samples to Redcliffs and 130 (87.24%) to Riccarton Medical Centre. The numbers of male and female respondents were approximately equal.

The researchers reporting on this project processed and presented all results anonymously. Participants were individually notified of their test results.

At Redcliffs 12.2% and at Riccarton 12.84% of the tests had blood positives.

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Screening for diabetes during and after an acute myocardial infarction: when and how?

The incidence of hyperglycaemia during an ST elevation myocardial infarction (STEMI) is significantly high.¹ Abnormal glucose tolerance during STEMI, diagnosed on a glucose tolerance test (GTT), is an important predictor for future cardiovascular outcomes.² Though previous studies have shown GTT to be reproducible a year after STEMI, this was more based on numerical data than the actual patient itself.³

In our study, all non-diabetic patients admitted with an STEMI were subjected to 75 grams oral GTT on day 5 after index event to diagnose abnormal glucose tolerance (AGT)[as diabetes (DM) and impaired glucose tolerance (IGT)].⁴ Patients with AGT were referred back to their family physicians for periodic monitoring and were prospectively called back for a repeat GTT (Figure 1).

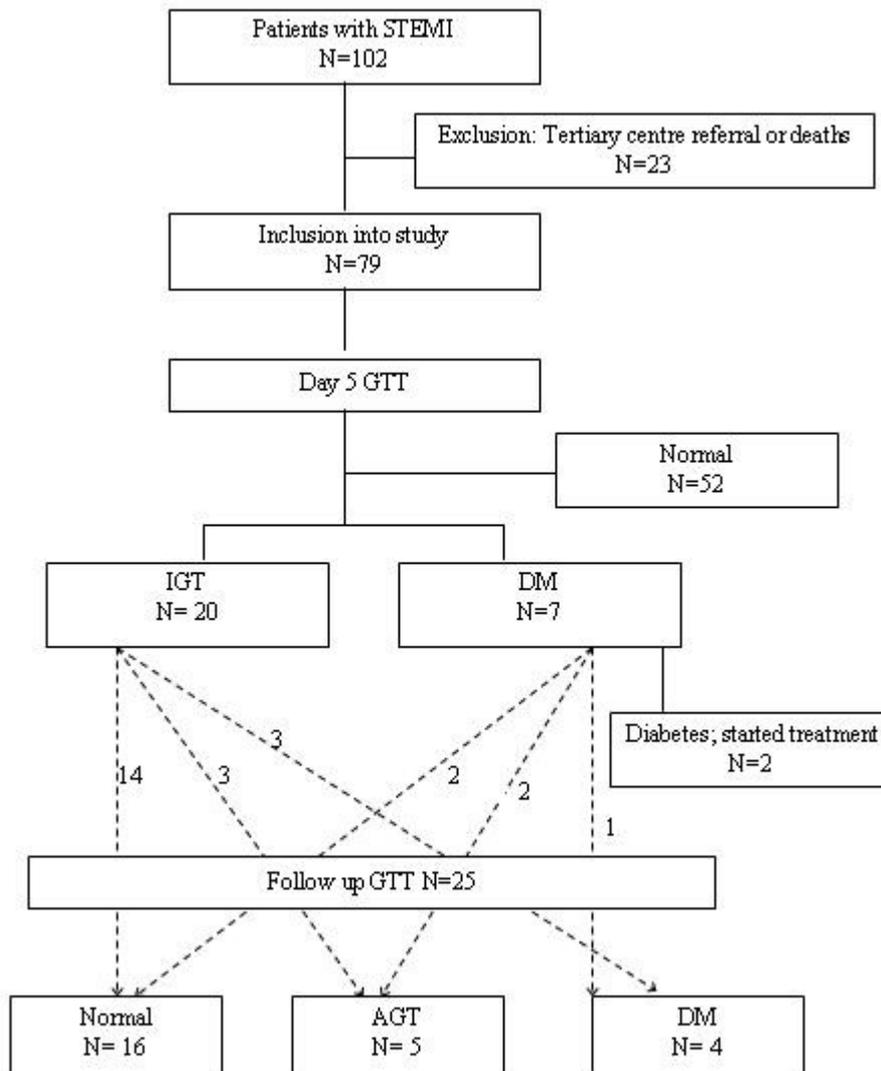
Seventy-nine consecutive patients were included for this study and 27(34%) had abnormal glucose tolerance based on day 5 GTT. Two patients were diagnosed with diabetes during the monitoring period with the family physicians and were initiated on treatment. 25 (2 patients diagnosed with DM during follow up period patients) had a repeat GTT done (mean 18 months; range 9–26) which showed 9(36%) to have persistent AGT, effectively being only 11% of the original cohort.

Our study again shows the high incidence of hyperglycaemia immediately after a STEMI and the phenomenon of stress hyperglycaemia. This study triggers a few vital issues: Firstly, HbA1c, in keeping with the current recommendations (ADA guidelines) would be a much better investigation to diagnose undiagnosed diabetes, as GTT does not differentiate between pre-existing diabetes and incident stress hyperglycaemia; however a GTT would be useful to guide immediate management.

Secondly, the link between AGT and future cardiovascular outcomes after STEMI are based on admission plasma glucose and GTT rather than HbA1c. Thirdly, GTT helps to diagnose more AGT compared to FPG alone (34% vs13% on day 5 GTT, 41% vs. 22% on follow up GTT in our study).⁵ However there is no clear consensus about the exact timing of the first or follow up GTT after a STEMI.

With the emphasis on using HbA1c as a diagnostic test for diabetes, clear guidelines are required regarding the most appropriate method and timing of screening for diabetes and the role of GTT in patients with acute myocardial infarction.

Figure 1. Flow diagram showing the results and the protocol of the study



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References:

1. Bartnik M, Ryden L, Ferrari R, et al. The prevalence of abnormal glucose regulation in patients with coronary artery disease across Europe. The Euro Heart Survey on diabetes and the heart. *Eur Heart J.* 2004 Nov;25(21):1880–1890.
2. Bartnik M, Malmberg K, Norhammar A, et al. Newly detected abnormal glucose tolerance: an important predictor of long-term outcome after myocardial infarction. *Eur Heart J.* 2004 Nov;25(22):1990–1997.
3. Wallander M, Malmberg K, Norhammar A, et al. Oral glucose tolerance test: a reliable tool for early detection of glucose abnormalities in patients with acute myocardial infarction in clinical practice: a report on repeated oral glucose tolerance tests from the GAMI study. *Diabetes Care* 2008 Jan;31(1):36–38.
4. American Diabetes Association. Standards of medical care in diabetes--2010. *Diabetes Care* 2010 Jan;33 Suppl 1:S11–61.
5. Jessani S, Gangopadhyay K, Patel JV, et al. Should oral glucose tolerance testing be mandatory following acute myocardial infarction? *Int J Clin Pract.* 2007 Apr;61(4):680–683.



Sugar consumption in New Zealand—with Thornley and McRobbie response

We write in response to Thornley et al's viewpoint article *The New Zealand sugar (fructose) fountain: time to turn the tide?* published in *The New Zealand Medical Journal* on 19 March 2010. The data quoted on sugar consumption in New Zealand are presented misleadingly and are not correctly referenced to primary sources.

The opening two sentences state: "In 2005 New Zealanders drank and ate, on average, over half a cup (158g) of sucrose (sugar) per day. In contrast, less than 40g a day (about 1½ tablespoons) are recommended by the World Health Organization (WHO) to prevent dental caries, obesity and chronic disease."

Firstly what is quoted is *total* sugar intake, not *sucrose* intake, as 158g per day. Total sugar intake is not equivalent to sucrose intake, which is only one type of dietary sugar, along with lactose, glucose, fructose and maltose.

Secondly, the WHO recommendation is for *added* sugars, not *total* sugars (which includes all sugars naturally present in foods). The generally accepted definition of *added* sugars is: "Sugars and syrups that are added to foods during processing or preparation. Added sugars do not include naturally occurring sugars such as those that occur in milk and fruits."

While it's not the same thing, data on *sucrose* intake are often used as a proxy for *added sugars* intake, since the majority of added sugars come in the form of sucrose in New Zealand. Comparing our *total* sugar (not sucrose) intakes with the WHO recommendation for *added sugar* (thereby inferring a nearly four fold difference), and using the terms sucrose and sugar interchangeably is both inaccurate and confusing. One would expect these terms to describe markedly different amounts, since the former encompasses sugar intake from all sources including fruit, vegetables, milk and honey, as well as added sugars.

In addition, the viewpoint article by Simon Thornley references a key New Zealand paper in this field, that by Parnell et al (Public Health Nutrition, 2007). The findings on beverages as source of sugar for New Zealand children are stated, but the author fails to acknowledge some of the key findings of this research. The paper titled *Exploring the relationship between sugars and obesity*, sought to discover the relationship (if any) between sugar intake and obesity, by analysing data from the 1997 National Nutrition Survey for Adults and the 2002 Children's Nutrition Survey. The researchers found no relationship between current sucrose intake from beverages (the predominant source of added sugar in children's diets) and obesity. Total current sugar intake (but not sucrose) was in fact significantly lower among obese children compared to children of a normal weight.

Adults and children with the lowest current intakes of sugar were actually significantly more likely to be overweight or obese. Subsequently, there was no relationship found between current sugar (sucrose) intake and body weight status in the New Zealand population. It is acknowledged that as a cross-sectional study we are

unable to equate these findings to sugar and sucrose intakes over time, however in the absence of longitudinal data for New Zealand this study is considered to provide credible and valuable information regarding current intake of sugar and body weight in both adults and children.

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Thornley and McRobbie respond

Parnell and colleagues draw attention to two items raised in our viewpoint article,¹ questioning our estimates of sugar consumption in New Zealand, and our fidelity in weighing the evidence linking sugar consumption with obesity.

The first point relates to our estimate of New Zealander's sugar consumption of 158g/day. This is derived from UN food balance sheet data which assesses the national "disappearance" of food, calculated from production, minus exports, plus imports. What is left is then considered to have been consumed, although this estimate does not account for wastage. Our figure was based on the disappearance of the item "sugar and sweetener's (Total)", which does not—as Parnell and colleagues claim—estimate total sugar intake, both added and intrinsic. This figure estimates the disappearance of *added* sugar and sweeteners in the food supply.

We concede that our report was slightly high, and that we should have restricted our estimate to sugar only, because other sweeteners may be glucose, fructose or a range of other mono or di saccharides. So our revised estimate, based on this source, is 50.5 kg/capita/year, or 138 g/day (**32 teaspoons** per day per person). Notwithstanding the lower figure, this still indicates that as a nation, we consume *added* sugar at a rate well above WHO recommendations and higher than indicated from survey data, based on self report, as in Parnell's study.

Parnell's assessment of New Zealander's sugar intakes are lower than food balance sheets.² However, what is not commonly reported in nutrition studies is that human memory of both the quantity and nature of food eaten is fallible, with recall estimates reporting about 20% less sucrose intake compared to more objective methods.³ This inaccuracy occurs non-randomly—obese people are more likely to under report consumption than normal weight people.⁴ Non-response also further underestimates food intake. Both sources, however, indicate that the majority of New Zealanders eat quantities of *added* sugar far in excess of the WHO guideline of 40g/day (**10 teaspoons** per day).

We are then accused of leaving out crucial analytical results of Parnell's study, which suggested that obese children *report* consuming less sugar than counterparts of normal weight. The study was not included for specific reasons. First, it employed a cross-sectional design. Second, it was funded by the sugar industry, and third, it

consisted of a secondary analysis of the data collected for other purposes. All characteristics have been identified in a meta-analysis which reports a positive association between soft drink consumption and obesity, as being less likely to report such a link.⁵ The weak study design, therefore, lead us not to include Parnell's analytical conclusions. Our article, instead, summarises the evidence of the adverse effects of sugar from either randomised controlled studies, or meta-analyses (of observational studies). Such designs are considered stronger than cross-sectional studies for assessing causation. The consistency of adverse effects, that we observe, linking added sugar, fructose and sugar-sweetened soft drink intake with obesity, dental decay, hypertension, insulin resistance and raised serum triglycerides remains.

We concur with Parnell that we have modestly overestimated national sugar consumption, but disagree that we have misrepresented research which links sugar intake with adverse health outcomes. In 2009, the American Heart Association reversed its earlier position,⁶ publishing guidelines that advise severe restriction of *added* sugar intake.⁷

We consider that the potential consequences of this ubiquitous exposure are too important to narrow our gaze to one cross-sectional study, sponsored by an industry with a lot to lose.

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References

1. Thornley S, McRobbie H, Jackson G. The New Zealand sugar (fructose) fountain: time to turn the tide? *N Z Med J*. 2010;123(1311):58–64. <http://www.nzmj.com/journal/123-1311/4030/content.pdf>
2. Parnell W, Wilson N, Alexander D, et al. Exploring the relationship between sugars and obesity. *Public Health Nutr*. 2008;11(08):860-6.
3. Karvetti RL, Knuts LR. Validity of the 24-hour dietary recall. *J Am Diet Assoc*. 1985;85(11):1437-42.
4. Zhang J, Temme EHM, Sasaki S, et al. Under- and Overreporting of Energy Intake using Urinary Cations as Biomarkers: Relation to Body Mass Index. *Am J Epidemiol*. 2000;152(5):453-62.
5. Vartanian LR, Schwartz MB, Brownell KD. Effects of Soft Drink Consumption on Nutrition and Health: A Systematic Review and Meta-Analysis. *Am J Public Health*. 2007;97(4):667-75.
6. Howard BV, Wylie-Rosett J. Sugar and Cardiovascular Disease: A Statement for Healthcare Professionals From the Committee on Nutrition of the Council on Nutrition, Physical Activity, and Metabolism of the American Heart Association. *Circulation*. 2002;106(4):523-7.
7. Johnson RK, Appel LJ, Brands M, et al. Dietary Sugars Intake and Cardiovascular Health: A Scientific Statement From the American Heart Association. *Circulation*. 2009;120(11):1011-20.



Ultrasound-guided nerve blocks are costly

I read the editorial *Regional Anaesthesia and pain relief after surgery* by Fredrickson et al¹ with interest. The current enthusiasm for ultrasound-guided nerve blocks needs to be tempered by a realisation of the cost of this procedure to the health system or health insurers.

In orthopaedics this procedure adds at least 15–30 minutes to the anaesthetic time which translates to an increase in theatre cost, and then a significant increase in rehabilitation time because the knee or hip doesn't work properly and patients mobilisation is delayed. In the hospitals in which I work if these techniques are used one can be sure the patient will require at least one extra day in hospital and often two, the cost of which is borne by the DHB or the health insurer.

What benefits are there for the patient? None that I can see given that last year's pain control worked fine, and of course lying around in bed may increase the risk of DVT/PE.

Like everything in medicine this current enthusiasm for regional blocks will wain. If you work in the finance department you will be praying it ends soon.

Geoffrey Horne
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Reference:

1. Fredrickson MJ, Kennedy RR. Regional anaesthesia and pain relief after surgery. *N Z Med J.* 2010;123(1326):9–11. <http://www.nzma.org.nz/journal/123-1324/4384/content.pdf>



Pelvic floor exercises

My doctor has encouraged me to write to you because this is a case of all the medical profession assuming that a patient automatically understands what is meant by a non-medical phrase, and he has pleaded guilty too.

In June 2009 I had a radical prostatectomy and, afterwards, was told to do the pelvic floor exercises to reform and strengthen the muscles that control the bladder. However, even though I read all the literature provided and listened to the hospital specialists and nurses, never was it explained that the exercises did not have to be done while sitting on the floor, but were actually a group of muscles at the base of the abdomen called “the pelvic floor”. Only when I received a pamphlet from ACC in September 2010 was it explained clearly!

Consequently I only did them intermittently because it was too painful when getting down to the floor. In June this year, because the incontinence had not stopped I had a loop inserted. To sit at all for some weeks was painful so the exercises again were only done intermittently. Later I was given a rubber-ring cushion that women apparently use when they have just had babies, because they have similar problems as I have recently had.

Although now retired I am considered reasonably intelligent (two post-graduate degrees) but am single, and do not know about these things that seem to be common knowledge to women and the medical/nursing profession.

As prostate cancer is so common, and the government is combatting it vigorously, it may help some other bachelor if the nurses ensure that he knows what is meant by the terms used. I have got used to the amusement this has occasioned amongst my retired nursing friends, so would ask you to publish this as it may help others in a similar position to myself.

David H B Speary
Auckland



Graham Collingwood (Mont) Liggins

Professor Sir Graham Collingwood Liggins or ‘Mont’—as he was known to family, friends and colleagues alike—died peacefully on 24 August 2010, after a long illness.



Mont began his life in Thames (NZ) on 24 June 1926 (slightly behind his twin sister Elizabeth), the last of five children born to James, the local GP surgeon, and Isobel.

His nickname Mont derived from his insistence at the age of 3 on being named after cartoon character Monty Mouse—it stuck for life, perhaps an early indication of the determination with which Mont continued to approach life. Mont described his childhood in Thames as magical. He spent his weekends exploring, with his mates, the old mines, on one occasion finding abandoned dynamite with which they proceeded to blast the hillsides.

He also discovered ‘inventing’—with the help of a friend’s uncle they would build steam, diesel and electric engines, and huge fireworks!

At the age of 15, Mont moved from Thames to board at Auckland Grammar for a final year at school before successfully completing medical intermediate at Auckland University, and so to Otago Medical School. Mont described his intermediate year as the hardest he ever worked—and declared that high marks would mean that he had worked too hard with not enough fun.

At Otago, he got the balance right, spending the years developing a life’s passion for the outdoors, playing rugby and learning to ski and rock-climb. One of Mont’s proudest achievements was winning a downhill, slalom and jumping championship on Mt Ruapehu.

Later these sports were replaced by fishing (all kinds), golf and planting trees. He loved spending time with his sons and grandsons on their annual deep-sea fishing trips. Closer to shore were regular weekends spent fishing around Auckland Harbour and Opahi Bay, and fly-fishing at Lake Rotoiti.

With house surgeon years in Auckland behind him, Mont spent 2 years working as a GP in Hamilton, before heading to England in 1953 to train in Obstetrics and Gynaecology. He returned 6 years later with his beloved wife Celia and three of their four children (Anne, Graham, and Jackie—Chris was born 2 years later).

Celia was from Whitehaven, a small fishing town in Cumbria, UK—family legend has it that they applied for the same O&G registrar position in Newcastle-upon-Tyne. One position became two and the rest was history. Mont always said that none of his achievements would have been possible without the love, care and support of Celia (a busy Obstetrician and Gynaecologist herself).

In 1959 Mont was offered a job at National Women's Hospital in the academic department, so began a 30-year career at NWH in clinical work, research and teaching. His life's work was to try and understand the initiation of labour, using the sheep as a model. He developed pioneering intrauterine surgical techniques and showed that the foetus initiated labour in the sheep. It was his lasting frustration that it wasn't quite so clear in humans.

In the process of this experimental work he serendipitously made the discovery that cortisol accelerated lung maturation in the premature foetus. This observation led to the ground-breaking RCT where he and Ross Howe showed that giving steroid hormones to mothers in premature labour dramatically reduced the incidence of respiratory distress in the newborn.

Mont was fascinated by science and unanswered questions. He made seven trips to Antarctica, camping on the ice with a team from Harvard University, chasing Weddell seals and investigating their diving physiology. He was involved in fertility research and first described foetal breathing. He and Celia made lasting friendships in the scientific community, opening their homes to many overseas visitors.

Mont received many honours. He was KBE, CBE, FRS (London), FRSNZ, FRCOG, FRCS, FRACS, PhD(Auck), MBChB(Otago), receiving awards and honorary degrees from around the world.

He will be remembered as a great New Zealand scientist, but also as a great father, grandfather, friend, fisherman, forester, teacher, doctor. He is survived by three of his children (Anne, Jackie and Chris) and nine grandchildren.

Written by Dr Jackie Liggins—daughter and Liaison Psychiatrist at Middlemore Hospital, Auckland.

THE NEW ZEALAND MEDICAL JOURNAL

Journal of the New Zealand Medical Association



Melvin Athol Brieseman

MB ChB 1959 NZ; FICS 1973; DPH 1977 Otago; DHA 1979; MCCM (NZ) 1980; FAFPHM (RACP) 1994

Melvin Brieseman fulfilled two lives, as a missionary doctor, and (for 31 years) the longest serving Medical Officer of Health in New Zealand. Mel, as he was known to all, was born in Stratford, Taranaki on 13 June 1934, the third of Francis and Ivy Brieseman's four children.



Mel was dux of Stratford High School. Mel's thoughts of becoming a doctor from an early age materialised when he entered Otago Medical School in the early 1950s.

In Dunedin he met Joan Daniels who was, at that time, secretary to the professor of microbiology. Mel and Joan shared a common bond in The Salvation Army. As their friendship matured and their future together seemed to be destined, Joan told Mel that she had, as a 7-year-old girl, heard a call to be a missionary, whether it be to India or to Africa she then did not know.

They were married in 1957.

Following his graduation in 1959, Mel became a junior medical officer at New Plymouth Hospital. In 1964, having completed the 4 years in hospitals which then entitled a recently-graduated doctor to a bursary for a year's overseas study, Mel and Joan travelled with four very young children to London.

In London, Mel studied at the Royal College of Surgeons and worked at Hillingdon Hospital. In 1966, following a residential period at The Salvation Army William Booth Memorial Training College, Captain Dr Mel Brieseman was appointed Chief Medical Officer of the Evangeline Booth Hospital. This hospital, one of six Salvation Army hospitals in India, is in Ahmednagar, Maharashtra, east of Mumbai. While here, Mel commenced outreach and public health programmes in surrounding villages.

In 1970, Mel returned to New Zealand, to further his knowledge and experience in medicine. In 1972, he was appointed Chief Medical Officer, Emery Hospital, Anand, Gujarat, 400km north of Mumbai. In 1974, Mel was laid low with a viral hepatitis, deeply jaundiced, that left him debilitated for many weeks.

For a 50-year reunion of classmates held in Queenstown early this year, Mel, with much encouragement, when his health was failing and his mind was not clear, wrote of his time in India:

I have enjoyed a widespread cover of a variety of medical fields—surgeon in every sphere apart from thoracic and cardiac—with obstetrics, including not a

few complications. Although I was not a specialist, I was needed in every situation. For example, a visiting New Zealand colleague, an obstetric GP, offered to assist if needed. A village mother with an impacted hydrocephalic dead foetus had been brought in one night. The colleague advised 'Can we call the local expert obstetrician?' My response had to be 'That's me!' So, having read up an obstetric textbook, I became the specialist needed to save the mother's life.

Joan often heard Mel say with regard to surgical matters, 'I did not know what to do, but my hands were directed.' There were times of great rejoicing, such as when a woman said to Mel, 'I thought I was going to die,' and Mel said, 'And so did I.' She had come in with a post-partum haemorrhage, bleeding profusely, and was nigh unto death with an extremely low haemoglobin level.

In 1976, Mel and Joan, when their older children graduated from secondary school, returned to New Zealand. Mel now gained the DPH and became Superintendent of Stratford Hospital. When the time came for Mel to return to India, the doors had been closed by the Indian Government for overseas workers.

Mel and Joan now moved to Christchurch, where Mel became Deputy Medical Officer of Health in November 1977. While Mel's work was immensely varied, the area he most made his own was communicable disease control. He was a member of the National Influenza Strategy Group from its inception and remained an honorary member after his retirement in recognition of his significant contribution to that work.

He was a member of the national Communicable Disease Control Advisory Committee for many years. He played a role in development of national strategies on immunisation and tuberculosis control amongst other things. He was also a foundation member of the Christchurch Infection Control Committee.

The 31 years that Mel worked in communicable disease saw the identification of campylobacteriosis, now New Zealand's most common notifiable disease, and the emergence of AIDS, SARS, and *E. coli* 0157. On the other hand, they saw New Zealand bring epidemics of invasive Hib disease and meningococcal disease under control. Mel was at the centre of the public health response to all of these issues. His name and face were for many years synonymous with public health in Canterbury.

Mel also took a great interest in the bigger picture of public health. He was a guest lecturer in the Diploma in Public Health and was frequently called upon to talk about the diversity of public health work that he had been involved with. He was a member of the Clinical Board of the Canterbury District Health Board. He was a Foundation member of the New Zealand College of Community Medicine in 1980. He also helped form the national Society of Medical Officers of Health, and always attended national MOH meetings and training.

Mel had a wealth of experience, and was generous in sharing it. He was also never afraid to be the one to take the responsibility and the flack that often came with working in the public eye.

If you asked Mel about what he did in his 31 years in public health, he'd tell you that he didn't do anything on his own, that he was always part of a team of public health people. Mel worked with patience, calmness, resilience and humour that were an

example to all. A caring colleague with broad shoulders and a warm heart, he will be fondly remembered by all those he touched.

Mel read widely, to a background of classical music. His spiritual home on earth was with The Salvation Army.

In early 2010, Mel was recognised to have a renal cell carcinoma metastatic to the brain. Mel died at his home in Christchurch on 25 October 2010, aged 76, surrounded by his family.

The celebration of his life was held in The Salvation Army Linwood Citadel, attended by some 300 people. Mel is survived by his wife Joan; children Nigel, Lyn, Sherry and Jo; and 10 grandchildren.

Dr Bramwell Cook wrote this obituary, with the assistance of Dr Daniel Williams and the family of Melvin Brieseman.