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This Issue in the Journal

Opioid poisoning deaths in New Zealand (2001–2002)

D Reith, J Fountain, M Tilyard

Researchers from the National Poisons Centre and the University of Otago performed a study to investigate the rates of narcotic deaths in New Zealand in the years 2001 and 2002. There were 92 poisoning deaths involving narcotics: morphine in 33, methadone in 31, dextropropoxyphene in 16, and codeine/dihydrocodeine in 12. There was a higher than expected rate of poisoning deaths associated with dextropropoxyphene, which is consistent with recent findings in the UK (see the letter in this issue at <http://www.nzma.org.nz/journal/118-1209/1294>). Restrictions in the availability of dextropropoxyphene, and increased monitoring of methadone, should be considered.

The impact of breast cancer screening on breast cancer registrations in New Zealand

A Richardson, B Cox, T Brown, Smale

The New Zealand national breast cancer screening programme, BreastScreen Aotearoa, was established in 1998. Since then, there has been an increase in the detection of breast cancer among women in the eligible age range, and a shift towards earlier diagnosis of breast cancer. These changes do not prove that BreastScreen Aotearoa will reduce deaths from breast cancer, but they provide an encouraging early indication. A breast cancer screening programme that did not show these early effects would be unlikely to reduce deaths from breast cancer in the longer term.

The epidemiology of breast cancer in Maori women in Aotearoa New Zealand: implications for screening and treatment

E Curtis, C Wright, M Wall

This paper describes the risk of developing breast cancer and dying from breast cancer in Maori and non-Maori women in New Zealand for 1996–2000. Despite having a similar risk of developing breast cancer, Maori women aged 25–59 years are more likely to die from breast cancer than non-Maori women. Further research is required to understand these ethnic disparities further (including the role of access to breast cancer screening and treatment, and deprivation).

The epidemiology of breast cancer in Maori women in Aotearoa New Zealand: implications for ethnicity data analysis

E Curtis, C Wright, M Wall

This paper describes new methods used to estimate the risk of developing breast cancer and dying from breast cancer in Maori and non-Maori women in New Zealand.

Four different methods were used to classify ethnicity from cancer registration and death data from 1996 to 2000. The findings confirm that the quality of ethnicity data in routinely collected datasets was poor. Suggestions of ways to improve the quality of ethnicity data estimates (for future breast cancer analyses) are suggested.

Anxiety before, during, and after participation in a population-based screening mammography programme in Waikato Province, New Zealand

M Brunton, C Jordan, I Campbell

Population-based programmes that screen for breast cancer have been criticised by some people for raising levels of anxiety in the population. As part of a larger research project, women (who had participated in a pilot screening programme in the Waikato region) were asked about the levels of worry they experienced at various stages of the process. Levels of anxiety were significantly related to ethnicity, and the majority of women ultimately experienced reassurance.

Use of early magnetic resonance imaging in the diagnosis of occult scaphoid fractures: The CAST Study (Canberra Area Scaphoid Trial)

S Kumar, A O'Connor, M Despois, H Galloway

The scaphoid bone is the most commonly broken wrist bone in young healthy individuals. As it is often difficult to diagnose the break on the X-ray, it is placed in a plaster cast for 10 days and re-X-rayed to accurately diagnose the break or to clear the injury. A magnetic resonance imaging (MRI) scan done in the first 24 hours after the injury (when the X-ray is normal) can diagnose the break thereby enabling early diagnosis and initiation of treatment. This also reduces the number of people who are lost in follow-up in 10 days and the over-treatment of patients with no break so that they can return to work early.

Reliability of magnetic resonance imaging for traumatic injury of the knee

K Winters, R Tregonning

This is a small study based in Wellington looking at the accuracy of magnetic resonance imaging (MRI) to evaluate the meniscal and ligamentous integrity of the injured knee.

Elevated serum prostate-specific antigen levels and public health implications in three New Zealand ethnic groups: European, Maori, and Pacific Island men

M Gray, B Borman, P Crampton, P Weinstein, C Wright, J Nacey

Available data show New Zealand European men have higher prostate cancer disease rates than both Maori and Pacific Islands men; however, by using a community-based rate of elevated serum Prostate Specific Antigen (a marker of prostate disease), this study found that the actual occurrence of prostate cancer between these three groups is likely to be at least equal. Because prostate cancer rates are determined by patients

seeking diagnosis and treatment for this disease, findings indicate cultural barriers in the health system for Maori and Pacific Islands men.



Prostate cancer screening—finding the middle road forward

David Lamb, Brett Delahunt

The viewpoint paper on prostate cancer screening in this issue of the *New Zealand Medical Journal* raises some interesting questions about the medical rights of individuals, and precisely what is unethical behaviour in medicine (Richardson A. *Prostate cancer screening: is it possible to explain diametrically opposed views?* URL: <http://www.nzma.org.nz/journal/118-1209/1289>).

The author immediately gets into difficulties by posing a question in the title of the paper, and then failing to address it in the subsequent text. The truth is that diametrically opposed views are rarely both right, and are often both wrong. The middle position, or moderate view, is usually shown in time to be the correct one. The extreme positions in prostate cancer screening are, at one end, that no one should be screened (the author's position); and at the other end, that all men over the age of 50 years should be enrolled in systematic population-based screening programmes.

This latter extreme is a position unsupported by nearly all health professionals active in the treatment of prostate cancer. The middle position, which currently available evidence suggests is a reasonable one to hold, is that screening should be accessible to those younger men most likely to have the disease, and to those men most likely to benefit from early diagnosis, such as men aged 70 years or less.

In adopting a paternalistic approach to all prostate cancer screening, the author fails to recognise that there are important differences between population-based screening (PBS) and self-requested screening (SRS).¹ The value of PBS is determined by measuring the net benefit for the population screened, and this requires levels I and II evidence from well designed randomised controlled trials (RCTs).

At present, such evidence is not available, but *absence of evidence is not evidence of absence*. Unless the biological behaviour of prostate cancer is completely different to that of other cancers, and we have no reason to believe that it is, then it is a reasonable premise that earlier detection and treatment of the disease will be associated with survival advantages for at least some patients. Backing this belief is the fact that, after treatment, the survival of patients presenting with locally advanced tumours extending through the prostate capsule falls to approximately 20% at 10 years, compared with 85% for early confined tumours.

Most clinicians do not believe that lead time bias can be the sole explanation for this difference, as the cancer control curves for patients presenting with non-metastatic tumours have reached a plateau by 10 years after treatment, indicating that patients who have not relapsed at this time are cured. If diagnosis as early as possible in the natural history is to be abandoned as one of the key goals for improving outcomes, RCTs will therefore need to produce some surprising results.

The situation with SRS is very different to PBS. Here it is the individual man who is making a value judgement on whether or not they perceive there are advantages in being screened. Given the uncertainties that currently exist, it is not appropriate for

others to make this decision on his behalf. The individual does require access to adequate educational literature to make an informed decision, and this material is currently not freely available to the public of New Zealand.¹ Most importantly, the public needs to know which people are most likely to develop prostate cancer, and which people are most likely to benefit from earlier diagnosis, as well as details of the screening process, and its side effects. Those who are found to have prostate cancer need additional information on the implications of their particular cancer,^{2,3} the management options open to them, and the possible side effects of treatment.⁴

The correct response is to provide this missing literature, not to withdraw all rights to screening. The suggestion that SRS is not 'ethical' is curious, as SRS is widely practised throughout New Zealand, and it is usually the majority opinion that determines what constitutes ethical behaviour.

Those men at particularly high risk of developing prostate cancer deserve a special mention. The risk of developing prostate cancer rises by up to 11 times that of the general male population when a man has one or more first-degree relatives with the disease.⁵ Surely these men with genetic risks comprise a group that should be actively encouraged to present for screening?

The utility of the prostate cancer screening process has been described in detail in a recent New Zealand publication.⁶ This paper addresses how the pathology report on prostate biopsies can be used to obtain a better understanding of whether or not the cancer is a clinically significant one. A recent Swedish report showed that unsuccessful treatment, or no treatment, of cancers considered 'low risk' at diagnosis led to a high mortality rate from cancer by the time 15 years follow-up had elapsed.⁷ The mortality rate was particularly high for those patients aged 70 years or less at diagnosis. The incidence of prostate cancer in the Finasteride Trial,⁸ referred to by the author of the opinion paper that follows, is certainly higher than might have been anticipated, but this is probably due to the inclusion of some low-grade tumours that most expert pathologists would regard as being of dubious clinical significance. The absence of any outcome data from this trial makes it impossible to interpret the data further.

The low mortality ratio (incidence/mortality) of prostate cancer seen in New Zealand⁹ and other Western countries is unlikely to be due solely to a high proportion of 'trivial' cancers allowing death from other causes to intervene. Effective treatment for early tumours is also probably a factor, but until the New Zealand Health Information Service (NZHIS) is able to monitor cohorts of patients with full clinical data, we will struggle to understand the relative contribution of treatment and natural history to patient outcomes.¹⁰

In conclusion, there are large gaps in our knowledge and understanding of prostate cancer, and we look to the results of large RCTs to fill these gaps. Until then, some assumptions have to be made based on the generic behaviour of cancer. Prostate cancer is a major cause of male death, so deserves the respect of all medical disciplines, as well as governments responsible for funding our health services.

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Opioid poisoning deaths in New Zealand (2001–2002)

David Reith, John Fountain, Murray Tilyard

Abstract

Aim To investigate the rates of opioid deaths in New Zealand relative to the utilisation of opioids.

Methods Deaths from opioid poisonings for New Zealand from 1 January 2001 to 31 December 2002 were identified from chemical injury cases that are routinely collected for surveillance purposes by the Institute of Environmental Science and Research from the Coronial Services Office in Wellington. Prescriptions for medicines containing morphine, methadone, and dextropropoxyphene were identified from the PharmHouse database from 1 January 2001 to 31 December 2002.

Results There were 92 poisoning deaths involving opioids: morphine in 33, methadone in 31, dextropropoxyphene in 16, and codeine/dihydrocodeine in 12. The rate (95% CI) of deaths per 100,000 prescriptions was 5.94 (4.09 to 8.34) for morphine, 1.34 (0.91 to 1.91) for methadone, and 2.5 (1.45 to 4.12) for dextropropoxyphene. The rate of deaths (95% CI) per 1,000,000 defined daily doses was 0.94 (0.65 to 1.32) for morphine, 0.40 (0.27 to 0.56) for methadone, and 0.14 (0.08 to 0.22) for dextropropoxyphene.

Conclusions Restrictions in the availability of dextropropoxyphene, and increased monitoring of prescription and dispensing of methadone, should be considered in order to reduce deaths due to opioids in New Zealand.

Opioids are amongst the most commonly implicated drugs in poisoning deaths.¹ This is related to the respiratory depressant and sedative effects of opioids, in addition to their potential for abuse. Opioids are also commonly used in the treatment of the terminally ill, and other patients with comorbidities, who have an increased risk of dying whilst under treatment. Opioids are also used in the treatment of substance abuse, such as in methadone maintenance programs. Patients with these conditions have a greater overall mortality than the general population.²

Recently there has been renewed interest in the role of dextropropoxyphene in opioid poisoning deaths.³ In England and Wales for the period 1997 to 1999, dextropropoxyphene-paracetamol combination medicines accounted for 5% of all suicides and 18% of drug-related suicides.³ Dextropropoxyphene was found in 7.5% of medicolegal autopsy peripheral blood specimens in Sweden between 1992 and 1996.⁴ Previously dextropropoxyphene has been described as having a disproportionate risk in comparison with prescription volumes.⁵ The hazard may be increased in patients with coexisting medical conditions such as renal failure.⁶ The aim of the present study was to investigate the rates of opioid deaths in New Zealand relative to the utilisation of opioids.

Methods

Deaths from opioid poisonings for New Zealand from 1 January 2001 to 31 December 2002 were identified from chemical injury cases that are routinely collected for surveillance purposes by the Institute of Environmental Science and Research (ESR) from the Coronial Services Office (CSO) in Wellington. The data used in the present analysis were current as of 28 January 2004.

From previous experience, there may be delay of over a year in the reporting of deaths from coroners and it is estimated 90%–95% of the poisoning deaths for 2002 were recorded by this date. Toxicology data were obtained from ESR toxicology reports that were present in approximately 95% of the coroner's files. Where this toxicology report was present, all substances detected were recorded in the chemical injury database with the exceptions of ethanol (where the blood level was less than 20 mg/dL) and lignocaine (a drug commonly given in resuscitation).

As the toxicological analysis did not usually analyse heroin separately, the heroin deaths were included with the morphine deaths. Whether the deaths were intentional (suicides or homicides) or unintentional (accidents) was determined according to the report of the coroner. The substance primarily involved in the fatality was determined using firstly the cause/circumstance recorded by the coroner, and secondly the primary and secondary substances identified in the ESR toxicology report.

Prescriptions for medicines containing morphine, methadone, and dextropropoxyphene were identified from the PharmHouse database from 1 January 2001 to 31 December 2002. The PharmHouse database is a subset of the New Zealand Health Information System database and contains records of all the claims for medicines dispensed within New Zealand. As codeine-containing preparations are available over the counter in New Zealand, prescription data were not obtained for codeine.

The records included the drug name, formulation, strength, type of prescriber, and the prescriber's New Zealand Medical Council number. The data were imported into Stata® for data management to enable tabulation of the prescription numbers by drug type.⁷ Analyses were also performed using defined daily doses (DDD) dispensed as the denominator.⁸ The defined daily doses used analgesia as the indication, and were 200 mg for dextropropoxyphene hydrochloride, 300 mg for dextropropoxyphene napsylate, 100 mg for morphine, and 25 mg for methadone. Rates and their 95% confidence intervals were calculated using the command 'cii' and the Poisson distribution in Stata®.

Results

There were 92 poisoning deaths involving opioids in New Zealand during 2001 and 2002 (Table 1). Morphine was the most frequently reported opioid reported in poisoning deaths, but there were almost as many methadone-related deaths.

Methadone and morphine deaths were predominantly considered to be unintentional with 28 of the 31 methadone deaths and 24 of 33 morphine deaths coded as unintentional, compared to 6 of 16 dextropropoxyphene deaths and 2 of 12 codeine/dihydrocodeine deaths. Fifty-two (56%) of the deaths occurred in males.

Sixty-two (67%) of the deaths occurred in the 25–44 year age group, 14 (15%) in the 45 to 64 year age group, 10 (11%) in the 15–24 year age group, and 6 (6%) in the over 65 year age group. There was an increased frequency of methadone deaths on Monday, Friday, and Saturday, compared with the other days of the week; no methadone deaths occurred on a Wednesday. It was not possible to determine whether in each case the deceased had been prescribed the opioid or had obtained the substance illicitly.

Table 1. Opioid deaths for 2001–2002 compared with usage (prescription volume or DDDs)

Drug	Prescriptions	Primary cause deaths	Primary cause deaths/100,000 prescriptions (95% CI)	Total related deaths	Total related deaths/1,000,000 prescriptions (95% CI)
Dextropropoxyphene/ dextropropoxyphene-paracetamol	630,655	8	1.27 (0.55 to 2.50)	16	2.5 (1.45 to 4.12)
Morphine/heroin	555,371	30	5.40 (3.65 to 7.71)	33	5.94 (4.09 to 8.34)
Methadone	2,308,646	30	1.30 (0.87 to 1.86)	31	1.34 (0.91 to 1.91)
Drug	DDDs	Primary cause deaths	Primary cause deaths/100,000 DDDs (95% CI)	Total related deaths	Total related deaths/1,000,000 units (95% CI)
Dextropropoxyphene/ dextropropoxyphene-paracetamol	11,682,679	8	0.07 (0.03 to 0.14)	16	0.14 (0.08 to 0.22)
Morphine sulphate	3,508,069	30	0.86 (0.58 to 1.22)	33	0.94 (0.65 to 1.32)
Methadone	7,830,402	30	0.38 (0.26 to 0.55)	31	0.40 (0.27 to 0.56)

DDDs=defined daily doses; CI=confidence interval.

Table 2. Usage of dextropropoxyphene, morphine, and desxpropoxyphene formulations (2001–2002)

Formulation	Prescriptions	DDDs
Cap dextropropoxyphene napsylate 100 mg	15,553	382,679
Cap dextropropoxyphene hydrochloride 32.5 mg with paracetamol 325 mg	29,155	549,369
Tab dextropropoxyphene napsylate 50 mg with paracetamol 325 mg	585,947	10,700,000
Cap morphine long-acting 100 mg	24,101	365,814
Cap morphine long-acting 10 mg	43,291	82,780
Cap morphine long-acting 20 mg	63,967	264,311
Cap morphine long-acting 50 mg	25,991	187,495
Injectable morphine 2 mg per ml, 1 ml	6	NA
Injectable morphine 5 mg per ml, 1 ml	2201	1215
Injectable morphine 10 mg per ml 1 ml	21,339	27,537
Injectable morphine 10 mg per ml, 5 ml	64	301
Injectable morphine 15 mg per ml, 1 ml	4091	6676
Injectable morphine 30 mg per ml, 1 ml	12,280	40,442
Suppository morphine 10 mg	323	NA
Suppository morphine 20 mg	125	NA
Suppository morphine 30 mg	235	NA
Suppository morphine 5 mg	281	NA
Tab morphine immediate release 10 mg	35,361	105,983
Tab morphine immediate release 20 mg	16,769	133,019
Tab morphine long-acting 100 mg	25,334	611,160
Tab morphine long-acting 10 mg	149,002	398,905
Tab morphine long-acting 200 mg	5474	227,016
Tab morphine long-acting 30 mg	80,502	513,828
Tab morphine long-acting 60 mg	44,634	541,588
Methadone	2,308,646	7,830,402

DDDs=defined daily doses; Cap=capsule; Tab=tablet; NA=data not available.

The most commonly prescribed formulation was dextropropoxyphene 50 mg with paracetamol 325 mg (Table 2), accounting for 49% of the analgesic opioid (dextropropoxyphene and morphine) prescriptions and 79% of the unit doses. The total number of prescriptions indicates that dextropropoxyphene was commonly prescribed in New Zealand during the study. When analysed by prescriptions, the death rate was similar for dextropropoxyphene and methadone, and both drugs had a lower rate of death than morphine (Table 1). When analysed by DDDs, dispensed dextropropoxyphene had the lowest rate of death followed by methadone, then morphine.

A total of 9255 prescribers issued at least one prescription for an opioid: 1064 prescribers issued at least one prescription for dextropropoxyphene, 7692 issued at least 1 prescription for dextropropoxyphene-paracetamol, 7172 issued at least 1 prescription for morphine sulphate, and 2779 issued at least 1 prescription for methadone.

The median number of prescriptions per prescriber of dextropropoxyphene 100 mg was 3, with a range of 1 to 342. The median number of morphine prescriptions was also 3 with a range of 1 to 1371. There was marked skewing of the prescription volume per prescriber for dextropropoxyphene-paracetamol. The top 5% of prescribers by volume were responsible for 44% of the dextropropoxyphene-paracetamol prescriptions, while the top 1% of prescribers were responsible for 17% of prescriptions and the top 0.1% of prescribers were responsible for 4.5% of

prescriptions. The top 5% of prescribers by tablet number were also responsible for prescribing 45% of the tablet volume for dextropropoxyphene-paracetamol.

Discussion

Dextropropoxyphene has previously been associated with a disproportionate rate of poisoning deaths in relation to prescription volume.^{3,5} The mode of death in dextropropoxyphene poisoning is a combination of respiratory and central nervous system depression, resulting from opioid effects; and cardiac arrhythmia, secondary to QT prolongation.⁹ Whilst tolerance may develop to the opioid effects, this would not be expected for the QRS prolongation. Hence patients who are on high doses of dextropropoxyphene, or are abusing dextropropoxyphene, may be at risk of cardiac arrhythmia. The majority of these deaths are reported as suicides, but the rate of accidental death is unexpectedly high and may be underestimated.¹⁰

Dextropropoxyphene has little advantage over paracetamol, and does not confer any benefit in combination with paracetamol, in clinical trials.¹¹ Although the present study was not able to measure the prescribing of paracetamol-codeine formulations, there were fewer deaths attributed to codeine over the time period of the study. Paracetamol-codeine preparations would be expected to have a better risk benefit profile than dextropropoxyphene-paracetamol, and should be preferred, except where there exists an allergy to codeine, or lack of response to codeine. Patients who are deficient in CYP2D6 (the enzyme that biotransforms codeine to morphine) may show no analgesic response to codeine and it may be preferable to prescribe morphine to these patients.¹²

Preliminary findings also suggest that by reducing the availability of dextropropoxyphene-paracetamol, poisoning deaths due to dextropropoxyphene-paracetamol can be reduced, but there is not necessarily a reduction in overall suicide mortality.^{13,14} However some of the dextropropoxyphene deaths appear to be accidental and restricting the availability of dextropropoxyphene may result in a decrease in these deaths.¹⁰ A difference in the representation of dextropropoxyphene in poisoning deaths in Scandinavian countries has previously been attributed to differences in availability of dextropropoxyphene.¹⁵

Methadone has previously been reported as a disproportionate contributor to poisoning deaths in New Zealand.¹⁶ This may be due to the practice of providing 'takeaways' for multiple doses that may be consumed as a single dose, and/or the illicit provision of these 'takeaways' to persons for whom they were not prescribed. Another factor may be the relatively low availability of heroin in New Zealand, reflected in a proportionately greater usage of methadone. The mode of death in methadone-related deaths is thought to be primarily respiratory depression, although in high doses torsade de pointes may occur.¹⁷

A contributing factor may be the time delay in achieving peak effect, which may be 4 hours in oral ingestion and 1 to 2 hours after subcutaneous or intramuscular administration.¹⁸ A significant proportion of the deaths appear to occur in naïve users, through the use of 'diverted' methadone.¹⁹ An inexperienced user may be falsely reassured by the lack of effect experienced immediately after administration, and doses/plasma levels tolerated by experienced users may be lethal to a naïve user.

Similarly, a period of abstinence in an experienced user may increase the risk of fatal opioid overdose due to loss of tolerance.^{20, 21}

The association of 'takeaways' with an increased rate of methadone-related deaths and diversion to naïve users has also been noted in Scotland.²² An increased rate of methadone-related deaths has also been noted on Saturdays and Sundays (but not Fridays), which is possibly related to pharmacy closures on Sundays.¹ With improved surveillance of methadone maintenance programs, this hazard has been ameliorated, with a consequent containment of methadone-related deaths.²² Hence, the practice of supplying 'takeaway' methadone doses in New Zealand appears to be a hazard and should be reviewed.

Indeed, a more robust monitoring of both the prescribing and dispensing of methadone could contribute to a decrease in methadone poisoning deaths. However, greater access to methadone maintenance programs appears to decrease overall opioid-related mortality and such programs should be maintained with appropriate controls.²³

Another contributor to opioid deaths is co-ingestion of ethanol and other sedative agents.² Opioids induce sedation via a different receptor mechanism to ethanol, benzodiazepines, and other sedatives, hence the effects of co-ingestion are additive. Therefore, caution should be exercised when prescribing opioids to patients with a history of ethanol abuse, or who are medicated with benzodiazepines.

The limitations of the present study include: an inability to measure illicit supply of opioids, an inability to determine intent, an inability to distinguish between heroin and morphine, and an inability to determine whether the opioids were diverted or prescribed. The DDD used were for analgesia, but the patients for which they were prescribed may have been tolerant to opioids and have therefore received higher doses. In addition, patterns of illicit drug use in New Zealand may differ from other countries in the region.

In New Zealand, there is a relatively high utilisation of methadone and morphine relative to illicit heroin use. This can be related to the production of morphine from codeine, obtained from commercially available codeine-containing products.²⁴ Hence morphine-related deaths in New Zealand may be due to illicitly produced morphine, in addition to illicit heroin, rather than morphine prescribed for therapeutic uses. New Zealand may also have a relatively high rate of amphetamine use compared with illicit opioid use;²⁵ so it may not be possible to extrapolate findings from New Zealand to other countries.

Conclusions

Restrictions in the availability of dextropropoxyphene, and increased monitoring of prescription and dispensing of methadone, should be considered in order to reduce deaths due to opioids in New Zealand.

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The impact of breast cancer screening on breast cancer registrations in New Zealand

Ann Richardson, Brian Cox, Thelma Brown, Paul Smale

Abstract

Aims To investigate the impact of the national breast cancer screening programme, BreastScreen Aotearoa, on breast cancer registrations in New Zealand.

Methods Age-specific breast cancer incidence rates for women aged 50–64 years were compared before and after the establishment of BreastScreen Aotearoa. The degree of spread of breast cancers diagnosed at screening was compared with the degree of spread of breast cancers registered before the introduction of population screening in New Zealand.

Results As expected, there was a marked increase in the age-specific incidence of breast cancer in New Zealand women aged 50–64 years in the first year of screening. There was a shift towards earlier diagnosis in women diagnosed with breast cancer at screening, compared with the diagnosis of breast cancers in women aged 50–64 registered before the introduction of population screening for breast cancer in New Zealand.

Conclusions BreastScreen Aotearoa has had the expected impact on breast cancer registration for a screening programme that detects breast cancer early.

BreastScreen Aotearoa started in December 1998. Up to June 2004, when the age-range was increased to 45–69 years, BreastScreen Aotearoa has offered 2-yearly two-view mammography to New Zealand women aged 50–64 years, with the aim of reducing the mortality from breast cancer in this population. Results from randomised controlled trials of breast cancer screening have shown that well-organised programmes can reduce deaths from breast cancer by about one-third among women aged 50 years and over who are offered screening.^{1,2}

Among women who accepted the offer of screening in these trials, breast cancer deaths were reduced by as much as 40%.³ The effect of screening in the routine healthcare setting is likely to be less, but the early results of at least one organised screening programme (albeit with very high participation of 85%) suggest a 24% reduction in breast cancer mortality is attainable.⁴ The benefits of screening for women aged under 50 years are uncertain.^{5–7}

Breast cancer screening should advance the time of diagnosis by detecting breast cancer earlier than it would otherwise be diagnosed. An indicator that a screening programme detects breast cancer early is an increase in breast cancer incidence in the first (prevalence) screening round. In the prevalence round of a new screening programme, most women have not previously had mammograms. If screening detects breast cancer early, it will identify tumours that may otherwise not have been diagnosed until some time later.

The early detection of these tumours causes an initial rise in the detection rate of breast cancers in the age group offered screening, to 2.5 to 3.0 times the pre-screening incidence. Some over-diagnosis of invasive breast cancer may occur. That is, some cancers may be detected which would not have presented as symptomatic disease because of their extremely slow growth. This may be about 6% of invasive cancers detected in a breast cancer screening programme.⁷

Another important indicator is the stage at diagnosis. If screening detects cancer early, a shift in the stage distribution towards earlier stage tumours would be expected, compared with the stage distribution of tumours diagnosed before the advent of widespread breast cancer screening in New Zealand.

If BreastScreen Aotearoa detected breast cancer early, there should have been an increase in the incidence of breast cancer in women aged 50–64 years during the prevalence screening round. Pilot breast cancer screening programmes had started earlier (during 1991 and 1992), but the pilot programmes screened only about 12% of New Zealand women aged 50–64 years.

Methods

The degree of spread of breast cancers at diagnosis was available from the New Zealand Cancer Registry. We have used the term 'degree of spread' rather than 'stage', to avoid confusion—because the information on stage of disease reported by the Cancer Registry is not the TNM stage. The stage reported by the Cancer Registry is closely related to the clinical stage of disease at diagnosis, and has been used to assess the effects of early detection in the breast screening programme.

Age-specific breast cancer incidence rates for New Zealand women aged 50–64 years were compared for the years before and after the establishment of BreastScreen Aotearoa.⁸ The degree of spread of breast cancer diagnosed in women screened in BreastScreen Aotearoa was compared with the degree of spread of breast cancers registered during 1979–1988 before the introduction of the pilot programmes or the establishment of BreastScreen Aotearoa.

Results

In the year (1999) following the establishment of BreastScreen Aotearoa, there was a marked increase in the age-specific incidence of breast cancer in New Zealand women aged 50–64 years (Figure 1).

Figure 2 shows a shift towards less spread of disease at diagnosis for screened women, compared with women in the same age range (50–64 years) before the introduction of population screening for breast cancer in New Zealand.

Discussion

The increase in the age-specific incidence of breast cancer in New Zealand women aged 50–64 years in the first year of screening suggests that BreastScreen Aotearoa detected breast cancer early in some women.

There was a shift towards earlier diagnosis of invasive cancer for screened women compared with women in the same age range (50–64 years) before the introduction of population screening for breast cancer in New Zealand. BreastScreen Aotearoa detected a higher proportion of localised invasive breast cancers compared with 1979–1988. Only a small proportion of this is likely to be due to over-diagnosis of extremely slow-growing invasive breast cancer.⁷ This comparison is between screened women and an unscreened population, and there may be selection effects, whereby

women who choose to be screened are those who would be more likely to present early in the absence of screening. Selection effects have been detected in randomised controlled trials of screening.³

Figure 1. Age-specific breast cancer incidence in New Zealand before and during the prevalence screening round of BreastScreen Aotearoa

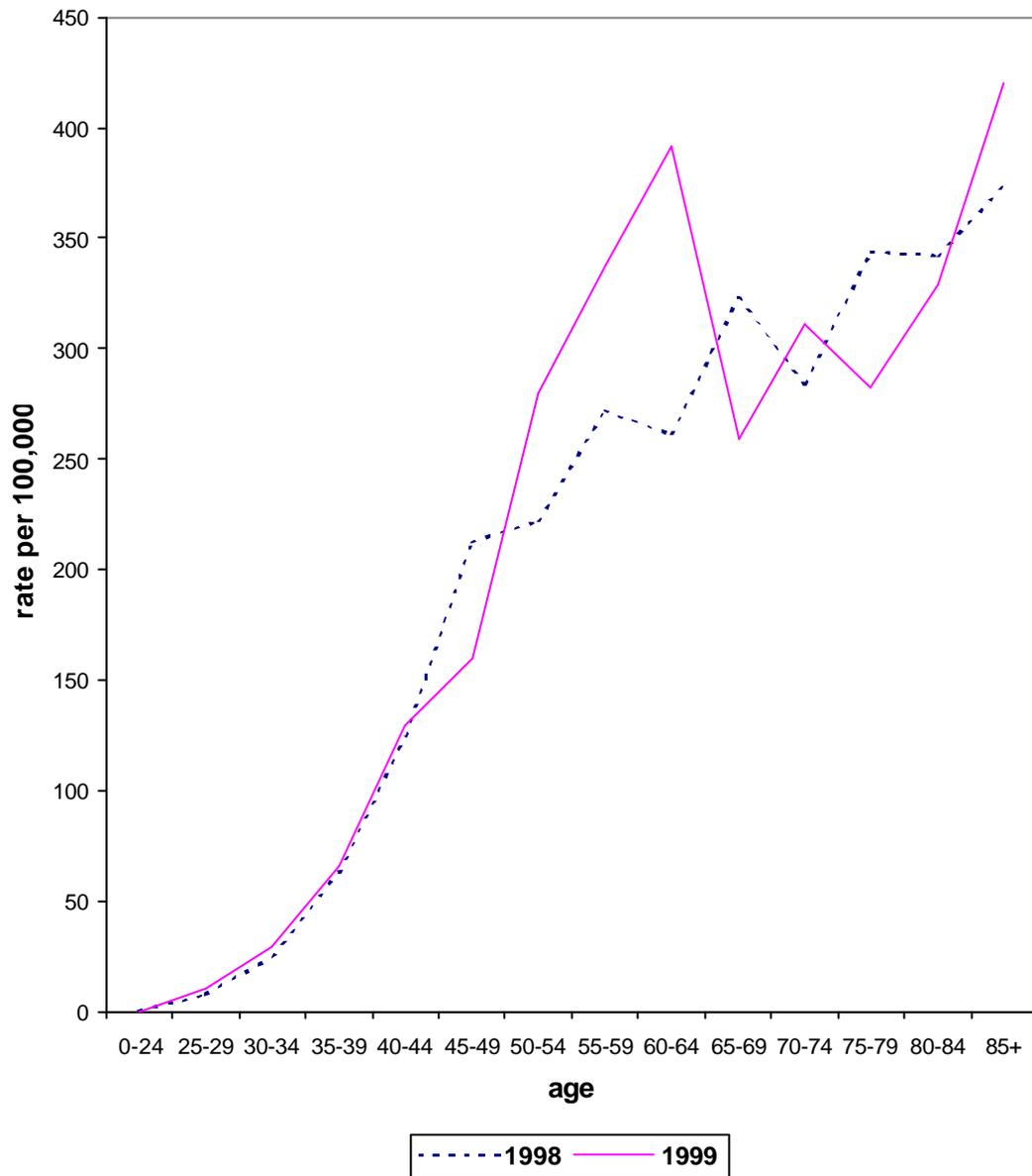


Figure 2. Stage distribution of invasive breast cancer diagnosed in BreastScreen Aotearoa during 1999-2001, compared with the stage distribution of invasive breast cancer diagnosed in New Zealand women aged 50–64 years during 1979-1988 (before the establishment of screening)

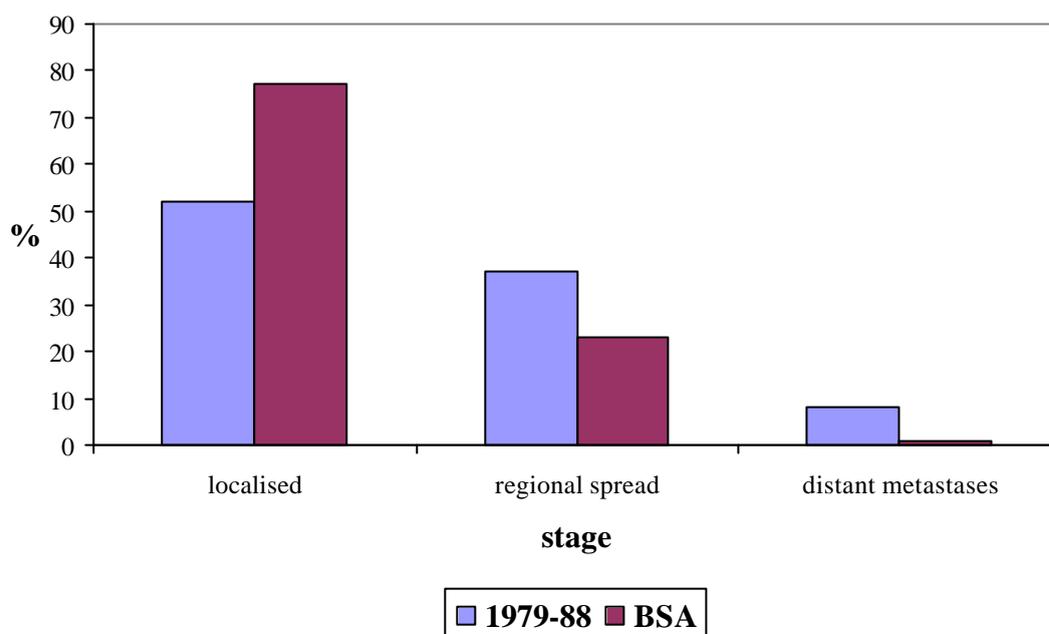


Figure 2. Continued

Stage (%)	Localised disease	Regional or node involvement	Distant metastases	Not stated
1979-88	52%	37%	8%	3%
BSA	77%	23%	<1%	0

$\chi^2=271.1$; $df=3$; $p<0.0001$; BSA= BreastScreen Aotearoa.

Unfortunately, adjustments for selection cannot be made for BreastScreen Aotearoa; there is no population register of women eligible for screening, and considerations of privacy have meant that no data are recorded about women who have declined an offer of screening.

During 1999–2001, 56% of eligible women were screened in BreastScreen Aotearoa. Only a major selection effect could completely explain the shift seen in Figure 2, if screening had no impact. BreastScreen Aotearoa also detected a similar proportion of node-negative invasive tumours to the Swedish Two-County Trial of breast cancer screening, with 75% of invasive tumours being node-negative.⁹ The Swedish Two-County Trial resulted in a significant reduction in breast cancer mortality among women offered screening, and the results from this trial have been used to develop international targets for breast cancer screening. The similarity in the nodal status of

invasive tumours detected by BreastScreen Aotearoa and the Swedish Two-County Trial is encouraging.

Other indicators of the impact of BreastScreen Aotearoa were being monitored for women screened up to June 2002, and have been published in the reports of the BreastScreen Aotearoa Independent Monitoring Group until early 2003.¹⁰

It is impossible to *prove* that any national screening programme reduces breast cancer mortality, since there is no control group. This is why internationally, screening programmes measure their progress against targets derived from randomised controlled trials. Because any impact of breast cancer screening on breast cancer mortality is not immediate, it is important to measure early indicators such as changes in breast cancer registration. The changes in patterns of breast cancer registration following the introduction of BreastScreen Aotearoa reported here, do not prove that the programme will reduce breast cancer mortality, but they provide an encouraging early indication. A breast cancer screening programme that did not show these effects would be unlikely to achieve a reduction in breast cancer mortality in the longer term.

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The epidemiology of breast cancer in Maori women in Aotearoa New Zealand: implications for screening and treatment

Elana Curtis, Craig Wright, Madeleine Wall

Abstract

Aim To describe the epidemiology of breast cancer in Maori and non-Maori women in New Zealand, and to identify the implications for breast cancer screening and treatment policy and practice.

Methods New Zealand Census Mortality Study (NZCMS)-adjusted age-specific incidence and mortality rates for breast cancer in total and sole Maori and non-Maori women were calculated using registration and mortality data obtained from New Zealand Health Information Service for 1996–2000.

Results Despite similar age-specific incidence rates of breast cancer in total Maori and non-Maori women under 50 years of age, total Maori women aged 25–59 years had higher age-specific mortality from breast cancer than non-Maori. A similar pattern is seen for sole Maori age-specific rates; however, the rates are even higher than total Maori rates.

Discussion Possible drivers of ethnic disparities in breast cancer mortality require investigation—particularly the role of access to breast cancer screening and treatment for Maori women compared to non-Maori. Specific initiatives are continually needed to ensure that Maori women are able to access breast cancer screening—otherwise ethnic inequalities in mortality will persist. The interaction between deprivation and ethnicity in breast cancer incidence and mortality analyses should be investigated in future analyses.

The aim of this paper is to describe the incidence and mortality of breast cancer in Maori and non-Maori women using New Zealand Census Mortality Study-adjusted estimates to assign ethnicity. The findings of this paper will help inform future policy development for breast cancer screening and treatment in New Zealand, with a particular emphasis on the identification and elimination of ethnic disparities between Maori and non-Maori. This emphasis is consistent with the Treaty of Waitangi, the indigenous rights of Maori and current Government policy.^{1–3}

Background

As is seen in many developed countries, breast cancer is the most common female cancer (apart from skin cancer) in New Zealand women, and the leading cause of female cancer deaths.^{4–6} There is consistent evidence of Maori vs non-Maori disparities in breast cancer mortality. In New Zealand, Maori women comprise 15.5% of the female population. In 1997, the overall age-standardised mortality rate was higher for Maori women (33 per 100,000) than ‘European and Other’ women (22 per 100,000).⁴

Age-specific analyses show that the mortality rate for Maori females aged 55–69 years in 1997 was higher than that of ‘European and Other’ women (118 versus 88 per 100,000).⁴ Recent New Zealand Census Mortality Study-adjusted estimates of ethnic-specific mortality found that (for 1996–1999) the Maori breast cancer mortality rate was nearly twice that of non-Maori non-Pacific women (36 versus 21 per 100,000).⁷

Estimates of the risk of developing breast cancer for Maori women in New Zealand are less consistent. Most analyses suggest that Maori women have a similar age-standardised incidence rate of breast cancer compared to other women in New Zealand.^{4,8–11} However, some analyses suggest that Maori breast cancer incidence may be higher or lower than that of non-Maori, depending on the classification of ethnicity in numerator and denominator data and study sample size.^{4,8,12}

The relationship between breast cancer incidence and mortality rates also varies by ethnicity internationally. In the United States, African-American women are noted to have a lower overall incidence of breast cancer than White women, but are disproportionately represented in breast cancer mortality statistics.¹³ A ‘black/white crossover’ in age-specific incidence rates has been documented, where African American women have a greater risk of developing breast cancer than White women below the age of 40 years, but a lower risk over age 40 years.¹⁴ The reasons for this pattern are not well understood, and hypotheses include variations in socioeconomic status, hormonal factors, genetics, nutrition, and healthcare access.^{15,16}

Methodology and Methods

This paper uses the methodological approach of Kaupapa Maori Research to analyse age-specific breast cancer incidence and mortality data for Maori and non-Maori women.^{17,18} Numerator data on breast cancer incidence and mortality for the years 1996–2000 were obtained from national cancer registration information (ICD9-CM code 174 Female Breast)¹⁹ and mortality information (ICD9-CM code 174 Malignant neoplasm of the female breast).²⁰

Based on the results discussed in an accompanying paper, ethnicity was assigned as either total or sole Maori and non-Maori by applying the New Zealand CMS adjustor to both breast cancer registration and deaths data.¹⁸ Denominator data were mean resident population estimates at the year ended 30 June (sourced from Statistics New Zealand estimates based on results from the 1996 and 2001 Censuses of Population and Dwellings).^{19–21}

Denominator ethnicity was identified as non-Maori and either:

- Total Maori—comprising sole Maori (those who identify Maori as their only ethnicity) and mixed Maori (those who identify Maori as one of their multiple ethnic groups), or
- Sole Maori.

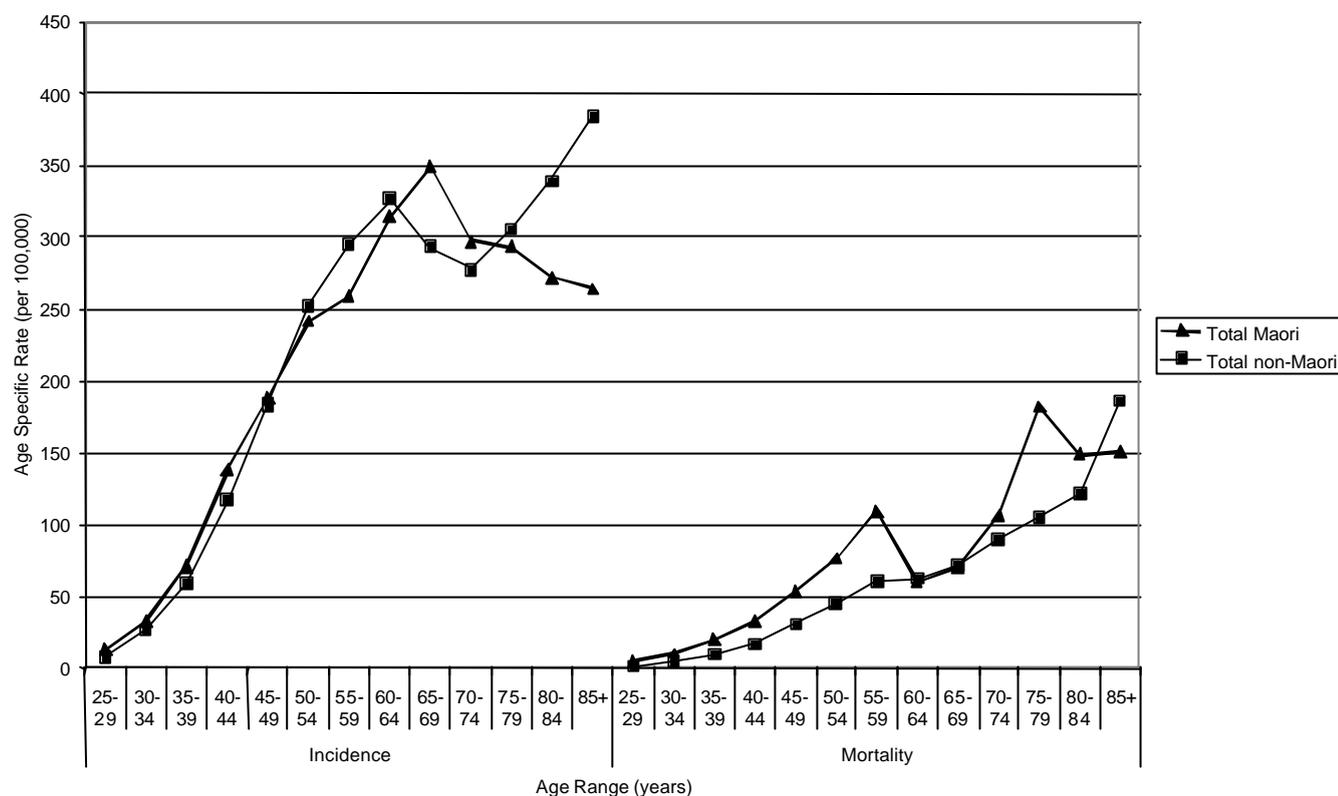
Results

Total Maori

A total of 10,424 women were identified from breast cancer registration data for the years 1996 to 2000. Of these women, 843 (8%) were identified as total Maori and 9,581 (92%) as non-Maori. A total of 2,560 women were identified from breast cancer mortality data for the years 1996–2000.

Of these women, 254 (10%) were identified as total Maori and 2,306 (90%) as non-Maori. Figure 1 presents the NZCMS adjusted age-specific breast cancer incidence and mortality rates for total Maori and non-Maori women in New Zealand.

Figure 1. NZCMS-adjusted age-specific breast cancer incidence and mortality rates for total Maori and non-Maori women in New Zealand (1996–2000)



The age-specific incidence rates of breast cancer are similar for Maori and non-Maori women, particularly in women aged under 55 years. Maori incidence rates drop below non-Maori rates between ages 55 and 64 years. Rates fluctuate from age 65 years, with the Maori incidence rate being greater than that of non-Maori from age 65 to 74 years, and then lower from age 75 years and over.

Breast cancer mortality rates are generally higher for Maori than non-Maori, particularly between ages 25 and 59 years. Of note, there is a drop in the Maori mortality rate between ages 60 and 69 years so that it is similar to that of non-Maori. Maori mortality then increases and is greater than non-Maori from age 70 to 84 years, peaking to approximately 180 deaths per 100,000 for ages 75 to 79 years, a rate not seen in non-Maori until age 85 years and over.

Sole Maori

A total of 10,680 women were identified from NZCMS breast cancer registration data for the years 1996 to 2000. Of these women, 675 (6%) were identified as sole Maori and 10,005 (94%) as non-Maori. A total of 2,560 women were identified from NZCMS breast cancer mortality data for the years 1996–2000. Of these women, 254 (10%) were identified as sole Maori and 2,306 (90%) as non-Maori.

Figure 2 presents the NZCMS-adjusted age-specific breast cancer incidence and mortality rates for sole Maori and non-Maori women in New Zealand for the years 1996 to 2000. Women classified as sole Maori ethnicity have higher age-specific breast cancer incidence and mortality rates than women in the total Maori group.

NZCMS-adjusted sole Maori mortality peaks at just over 200 breast cancer deaths per 100,000 for women aged 75 to 79 years, slightly higher than the peak seen for NZCMS-adjusted total Maori women.

Figure 2. NZCMS-adjusted age-specific breast cancer incidence and mortality rates for sole Maori and non-Maori women in New Zealand (1996–2000)

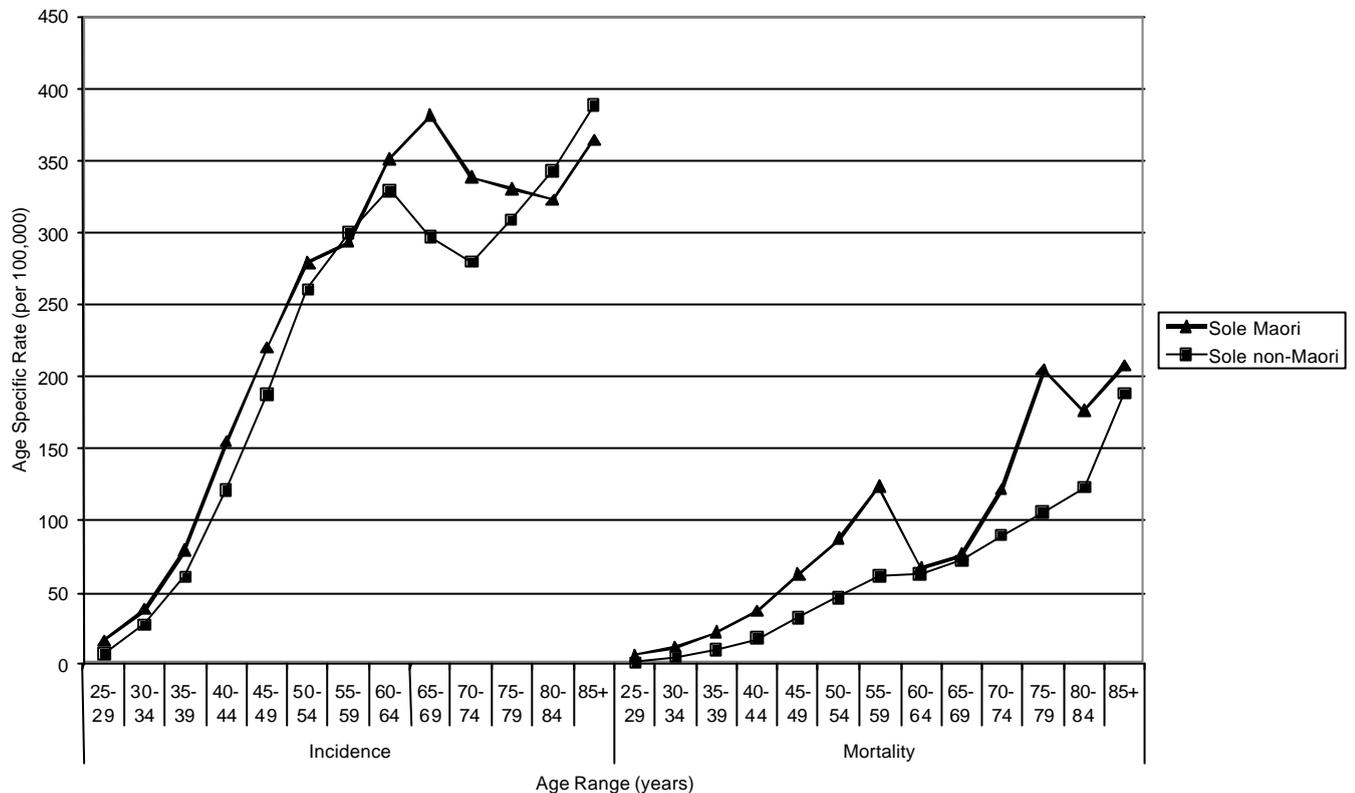
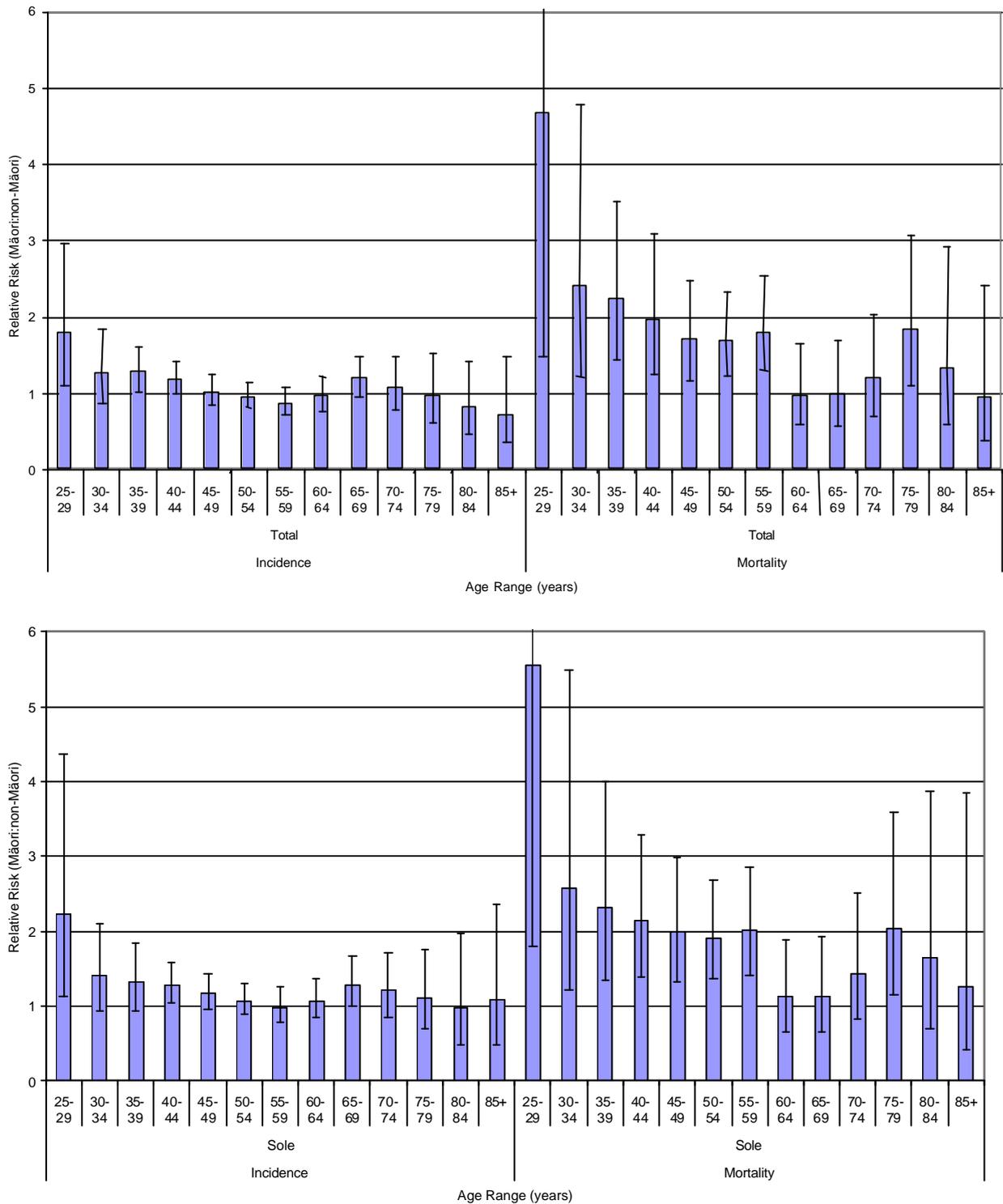


Figure 3 presents age-specific breast cancer incidence and mortality estimates as risk ratios for total and sole Maori and non-Maori women in New Zealand. Although incidence estimates suggest an elevated risk of breast cancer in total Maori women compared with non-Maori aged under 50 years, most of the confidence intervals for the 5-year age groups include one and therefore are not statistically significant at the 95% level of confidence. Only Maori women aged 25 to 29 years had a significantly higher risk of developing breast cancer than non-Maori women, but the numbers in this age group are small.

In contrast, the relative risk of death from breast cancer is generally greater for total Maori women than non-Maori, apart from the noted drop between ages 60 and 69 years and 80 years and over. Of note, the Maori vs non-Maori relative risk estimates for breast cancer mortality in women aged between 25 and 59 years are statistically significant, and therefore are unlikely to be due to chance.

Figure 3. NZCMS-adjusted age-specific breast cancer incidence and mortality relative risk for total/sole Maori and non-Maori women in New Zealand (1996–2000)



Overall the pattern found for sole Maori vs non-Maori risk ratios are similar to those for total Maori vs non-Maori risk ratios—i.e. ranging from 1.0–2.2 for sole incidence and 1.1–5.6 for sole mortality versus 0.7–1.8 for total incidence and 0.9–4.7 for total

mortality. Although the pattern is similar, the risk ratios for sole Maori are generally higher than those for total Maori.

Summary of key results

This analysis found that, despite similar age-specific breast cancer incidence rates, total Maori women had higher age-specific mortality rates from breast cancer than non-Maori women, particularly below the age of 60 years. A similar pattern is seen for sole Maori age-specific rates, but the mortality rates are even higher than those for total Maori. Total Maori women aged 65 to 74 years and sole Maori women aged 60 to 79 years had non-significant higher incidence rates of breast cancer than non-Maori women.

Discussion

This study represents the first time a Kaupapa Maori Research analysis of age-specific breast cancer incidence and mortality has been performed in New Zealand using NZCMS adjustment to compensate for ethnicity misclassification. Thus, the study provides better quality ethnicity data on which to base planning for breast cancer screening and treatment services than has been available previously.

A notable drop in mortality rates for both total and sole Maori women aged 60 to 69 years was observed. It is unclear why this drop in mortality occurs, but there are two possible explanations. First, there could be a 'cohort effect' for Maori women associated with reduced breast cancer mortality due to mortality from other causes, or better access to breast cancer screening and/or treatment services. Secondly, it may be due to a fluctuating mortality pattern in older age groups due to the relatively short study period and modest number of breast cancer events available for analysis. Therefore, further investigation is required.

Both total and sole Maori women aged 50 to 64 years have a lower breast cancer incidence rate than non-Maori women. This may be explained by the aggregation of 5 years' worth of data that includes 2 years in which BreastScreen Aotearoa was formally operating: 1998 and 1999. Coverage data show that non-Maori women were more likely to have been screened in this 2-year period than Maori women, producing a relatively higher incidence rate for non-Maori. It is possible that, in the absence of screening, non-Maori incidence rates between ages 50–64 years would have been similar to or below those for Maori women. This hypothesis could be investigated by comparing Maori vs non-Maori incidence rates for non-screen detected cancers only.

There are three broad explanations for the finding that Maori women have a similar risk of developing breast cancer but a greater risk of death from breast cancer than non-Maori women. Firstly, Maori women may have a biologically different disease that is more aggressive than non-Maori leading to a poorer prognosis. Secondly, Maori women may experience a greater delay in diagnosis of their breast cancer as a result of differential access to screening and/or primary health care. Thirdly, Maori women may experience poorer outcomes from breast cancer treatment associated with differential access to treatment, patterns of referral, and/or quality of care within the care pathway.

There is currently no conclusive evidence that ethnic differences in breast cancer mortality reflect biologically different disease processes occurring for Maori women in comparison to non-Maori.

There is evidence that late diagnosis occurs for Maori women. Armstrong and Borman reviewed cancer registry data between the years 1972 to 1992 and found that Maori women had higher rates of regional and metastatic stages than non-Maori women.⁹ Lawes et al also reviewed cancer registry data from 1987 to 1994 and found that Maori women were diagnosed with a more advanced stage of breast cancer than 'Other' women.⁸

Lethaby et al found that (in the Auckland region between 1976 and 1985) Maori women were diagnosed with larger tumours and were more likely to have metastases at presentation than non-Maori women.²² Interestingly, they note that a delay in seeking treatment for initial symptoms did not differ significantly by ethnicity in their study, implying that the delay was not in presentation but in diagnosis. Similar findings were found by Newman et al, who noted that Maori women were significantly more likely to be diagnosed with large tumours (31%), have 1–3 axillary lymph nodes involved (33%), and have metastases (14%) at time of presentation than European women (19%, 20%, and 11% respectively).²³

Maori women are also less likely to receive breast cancer screening services. Between 1999 and 2001, BreastScreen Aotearoa data show that participation in breast cancer screening was significantly lower for Maori (39%) and Pacific (34%) women than for non-Maori/non-Pacific women (59%).²⁴

There is no conclusive information on whether Maori women may be less likely to receive radiotherapy, chemotherapy and/or surgery necessary to effectively treat breast cancer once it has been diagnosed. Feek et al used NZHIS data to estimate five-year relative survival rates for breast cancer using 1996 to 1999 mortality data.²⁵ They found that only 64% of Maori women survived 5 years after diagnosis compared with 81% of 'Other' women. However, this analysis did not control for stage of diagnosis, nodal status, or tumour type, so it is not possible to determine whether the disparity in survival reflects differential access to treatment.

Lethaby et al reviewed breast cancer survival and controlled for stage of diagnosis and found that ethnicity was not an independent factor influencing survival. However, the authors acknowledge that the small sample size of Maori women included in their study meant that their findings were inconclusive.²²

Investigating where and how ethnic differences may be occurring on the entire breast cancer care pathway is warranted.

Implications for screening

The finding of Maori vs non-Maori disparities in age-specific breast cancer mortality supports the need to deliver accessible and appropriate breast cancer screening services to Maori women. Indeed, the successful screening of Maori women has the potential to produce even greater benefit for Maori women than non-Maori women, particularly for women under the age of 60 years.

This is particularly important with the recent announcement by the Ministry of Health of an extended age-range down to 45 and up to 69 years.²⁶ If screening in both the

original age range and the proposed extended age range of 45 to 69 years is not successful for Maori women, it is possible that Maori vs non-Maori inequalities may worsen. Given this context, on the basis of high health need, Maori women are clearly a priority group for access to breast cancer screening in New Zealand.

It is important to note that the presence of ethnic inequalities in screening programmes is not a unique problem to New Zealand and is a challenge that other countries are attempting to address.^{27,28}

The role of deprivation

To date, international incidence data and case-control studies have led to the typical portrayal of breast cancer as a disease of affluence.¹⁵ However, there is increasing recognition that the incidence of breast cancer in poorer countries and among women of lower socioeconomic status is 'catching up'.¹⁵ In New Zealand, Ministry of Health data suggest that breast cancer incidence increases with increasing deprivation.⁴ This pattern is not reported for mortality, although this may be because few data were available for this analysis (1996/97).⁴

Given these findings, the relationship between breast cancer and socioeconomic status appears to be more complex than previously thought, and analysis of breast cancer epidemiology stratified for deprivation would be valuable. In addition, ethnic disparities in the distribution of deprivation are likely to be contributing to Maori disparities in breast cancer screening and treatment, and should be specifically investigated in future analyses. The possibility that ethnicity may have an independent effect on whether or not Maori women receive access to appropriate breast cancer screening and treatments warrants further investigation, especially as previous New Zealand studies have documented differential access to health services by ethnicity.^{29,30}

Conclusion

This study highlights the need to ensure that breast cancer screening is effectively delivered to Maori women so that ethnic inequalities are reduced rather than increased by the breast screening programme. The presence of age-specific disparities in breast cancer mortality between Maori and non-Maori (despite similar breast cancer incidence rates) is very concerning. Additional research is required urgently to understand these disparities, develop effective interventions, and therefore eliminate their existence.

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The epidemiology of breast cancer in Maori women in Aotearoa New Zealand: implications for ethnicity data analysis

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Abstract

Aim To describe the methods used to estimate breast cancer incidence and mortality in Maori and non-Maori women using multiple adjusters to assign ethnicity.

Methods Age-specific incidence and mortality rates for breast cancer in Maori and non-Maori were calculated using registration and deaths data obtained from New Zealand Health Information Service (NZHIS) for 1996–2000. Four different methods were used to assign total and sole ethnicity: New Zealand Census Mortality Study (NZCMS)-adjusted, ever Maori-adjusted, National Health Index (NHI)-adjusted, and unadjusted source information.

Results Unadjusted and NHI-adjusted estimates were least similar to the NZCMS-adjusted estimate used as the ‘gold standard’ in this study. Ever Maori-adjusted results closely approximated NZCMS-adjusted results in both incidence and mortality data. Sole Maori breast cancer incidence and mortality estimates were generally higher than total Maori estimates.

Discussion Using four different estimates to assign ethnicity confirms previous findings showing poor quality of ethnicity data in routinely collected datasets. Future calculations of breast cancer incidence and mortality rates should assign total and sole ethnicity and reduce ethnicity misclassification by using NZCMS or ever Maori-adjusted estimates. This paper supports the need to collect better quality ethnicity data in order to identify and monitor Maori vs non-Maori cancer inequalities.

The epidemiology of breast cancer in New Zealand women has been well described from a total population perspective, but Maori specific analyses have produced inconsistent results.^{1–8} This paper presents the methods used to analyse breast cancer incidence and mortality in Maori and non-Maori women for a 5-year period (1996–2000). A full discussion of the ethnic disparities identified by this analysis is presented in an accompanying paper.⁹

Background

Breast cancer is a major cause of death for women both internationally and in New Zealand.^{2,10} Ethnic disparities in breast cancer mortality have been consistently documented with Maori women having a higher mortality rate than non-Maori.^{2,11,12} In contrast, ethnic differences in breast cancer incidence rates between Maori and non-Maori are less consistent with conflicting estimates stating Maori risk as similar, higher and/or lower than non-Maori.^{2–5,7} Explanations for these findings may include: variations in the analytical frameworks used to explore ethnic differences, such as sole versus total ethnic group analyses (the latter includes people who identify with

only one ethnic group plus those people who identify with more than one ethnic group, where ethnicity is assigned by application of a prioritisation rule—i.e. Maori first, then Pacific Island, European and Other)^{2,4,7}; blood quantum versus cultural affiliation definitions of ethnicity,⁴ and a lack of equal explanatory power—i.e. small sample sizes for Maori compared to non-Maori.^{3,5}

In addition, there are problems with ethnicity data within cancer registration and death records in New Zealand. Cancer registration data come primarily from laboratory information or hospital records. Although ethnicity is supposed to be self-defined in these records, reviews have shown that this is not always the case, particularly in the hospital setting.¹³ Furthermore, although it has been possible to record multiple ethnic groups since July 1996, less than 2% of hospital records had ethnicity recorded as including more than one ethnic group during 1997–1998, suggesting that this option was either not given or not recorded by hospital staff.¹⁴ Approximately 10% of breast cancer registrations in 1999 had ethnicity recorded as ‘not stated’.¹⁵

There are similar issues with cancer mortality data. Prior to September 1995, deaths were officially registered as Maori, usually by funeral directors, if the deceased was determined to be of half or more Maori ancestry based on the descent of their parents.¹³ This led to an under-reporting of Maori deaths prior to 1995.^{16,17} Since 1995, the classification of ethnicity on death records has changed to determine the ethnicity (rather than ancestry) of all deceased with multiple options available as in the census.¹³ Although this has led to an increased recording of Maori in mortality datasets, there is still evidence of undercounting.^{12,13}

This paper recognises the need to review the quality of ethnicity data within cancer registration and mortality datasets when considering Maori vs non-Maori disparities in breast cancer incidence and mortality. This is consistent with the recently released *New Zealand Cancer Control Strategy*, which aims to reduce both the incidence and impact of cancer and inequalities with respect to cancer.¹⁸

Methodology

This paper uses the methodological approach of Kaupapa Maori Research (KMR)¹⁹. To date, KMR has been used primarily within a qualitative setting. However, a number of more recent studies have described the use of KMR within quantitative analyses, particularly within health.²⁰⁻²² This paper reflects a KMR approach because: it puts Maori at the centre of enquiry (i.e. focuses on Maori data and identifies the best quality information available); performs a Maori vs non-Maori analysis (i.e. ensures that Maori are not treated simply as a subgroup of the total population); maximises the quality of ethnicity data (i.e. uses multiple methods to assign ethnicity), and maximises statistical power (i.e. by aggregating five years of data).

This approach reflects the principle of ‘equal explanatory power’, which promotes the need to gather data to the same breadth and depth for the Maori population as it is collected for the non-Maori population in order to appropriately analyse Maori vs non-Maori inequalities.²³

Methods

Numerator data

Numerator data on breast cancer incidence and mortality were obtained from national cancer registration information (ICD9-CM code 174 Female Breast)¹⁵ and mortality information (ICD-9-CMA-II code 174 Malignant neoplasm of the female breast).²⁴ To assess the extent of the Maori undercount associated with routinely collected datasets, four different methods were used to calculate breast cancer incidence and mortality. These methods were chosen because they reflect current practice

in New Zealand, and they allow recently developed analyses (i.e. New Zealand Census Mortality Study [NZCMS] and ever-Maori adjustments) to be examined and compared with unadjusted datasets.

NZCMS mortality-adjusted estimates—These estimates draw on adjustors from the NZCMS, which is a record linkage study in which death registration data are anonymously and probabilistically linked to census data.²⁵ Ethnic specific rates are calculated by multiplying the Census Mortality adjustor with either the incidence or mortality rate for each age group. As this corrects the estimate for numerator-denominator bias, the NZCMS adjustment is used as the ‘gold standard’ against which other methods are compared in this paper.¹² It is uncertain how valid it is to apply this method to morbidity data, such as cancer registrations, because ethnicity information in morbidity data has not yet been matched to census ethnicity information. An assumption has been made that the problems with ethnicity data collection are similar between routinely collected data sets. Thus, applying a mortality adjustor to morbidity data is likely to produce more accurate estimates, and this approach is consistent with recent Ministry of Health cancer analyses.²

Ever Maori-adjusted estimates—Ethnicity is adjusted according to whether or not any previous admission for patients (as identified by their unique NHI identifiers) had been recorded as Maori, either sole or total, in any admission record at any time or on the death certificate. All remaining records, including those with no ethnicity specified in the NHI unique identifier, were classified as non-Maori. Other researchers have used this method to reduce the undercount of Maori in routinely collected datasets.^{20,21} We hypothesised that the ever-Maori approach would approximate the NZCMS adjustment, but probably corrects Maori rates for older age groups better than for younger age groups. This reflects the fact that older people are more likely than younger people to have had previous hospital admissions, and are therefore more likely to have an NHI unique identifier by which to assign an ever-Maori ethnicity classification.

National Health Index (NHI)-adjusted estimates—Ethnicity is adjusted according to the most recent NHI ethnicity recorded in hospital admission data. We expected that this estimate would underestimate total Maori and over-estimate sole Maori rates compared with NZCMS-adjusted rates. This effect may be even greater than that seen in the unadjusted source information because NHI data only record ethnicity on the most recent publicly-funded hospital admission.

Unadjusted source information—Ethnicity is identified according to the classification recorded at either the cancer or death registration event. This estimate depends on the quality of data in routinely collected cancer registration and mortality datasets. The New Zealand Census Mortality Study indicates that this approach is likely to under-estimate total Maori and over-estimate sole Maori rates.¹²

Denominator data

Denominator data were mean resident population estimates at the year ended 30 June, sourced from Statistics New Zealand estimates based on results from the 1996 and 2001 Censuses of Population and Dwellings.^{15,24,26}

Denominator ethnicity was identified as non-Maori and either:

- Total Maori comprising sole Maori (those who identify Maori as their only ethnicity) and mixed Maori (those who identify Maori as one of their multiple ethnic groups), or;
- Sole Maori.

Data Analysis

All statistical analyses were conducted using the SAS system for Windows version 8.2. As this study utilised data collected from cancer registry and mortality records, it was assumed that all cancers and cancer deaths in New Zealand had been obtained. Therefore, the confidence limits provided reflect the uncertainty due to misassignment of ethnicity to each cancer incidence or death event. A Delete-A-Group Jackknife method was used to estimate the confidence intervals around the risk ratios.²⁷ This involves randomly allocating each individual in the female population to one of one hundred groups. The risk ratios are then recalculated one hundred times after deleting only those women assigned to each group. The variation in the distribution of Delete-A-Group Jackknife estimates were used as a measure of the error in the risk ratios due to misassignment of ethnicity, and the resulting confidence intervals were then log-transformed.²⁸

Results

Incidence

Table 1 presents the total number of breast cancer registrations in New Zealand for the years 1996–2000 by total and sole ethnicity. A total of 10,524 records were analysed for unadjusted, NHI-adjusted, and ever Maori-adjusted estimates; 10,424 for NZCMS-adjusted total; and 10,680 for NZCMS-adjusted sole. The difference in total numbers reflects the fact that NZCMS adjustors have been smoothed to allow 5-year age group adjustors. While the total numbers are not exactly the same, the relative proportions are correct based on the smoothing function used.²⁶ The percentage of total Maori women ranged from 6.2% (NHI) to 8.6% (ever-Maori). Sole Maori percentages were slightly lower with findings ranging from 5.7% (NHI) to 7.5% (unadjusted).

Table 2 presents age-specific relative risk (Maori vs non-Maori) of breast cancer incidence for unadjusted, NHI, ever-Maori and NZCMS-adjusted estimates. Overall, the relative risk of breast cancer incidence is generally higher for sole Maori ethnicity compared with total Maori ethnicity analyses. With respect to total findings, the NHI-adjusted estimate varies most from the NZCMS-adjusted estimate. Both the unadjusted and ever-Maori estimates appear to be relatively similar to the NZCMS-adjusted estimate. When comparing sole Maori findings, the unadjusted estimate is the least consistent with the NZCMS-adjusted one, over-estimating sole Maori risk of developing breast cancer. The NHI-adjusted sole Maori estimate is comparable with the NZCMS-adjusted estimate overall; however, it also appears to overestimate Maori breast cancer incidence at younger ages (i.e. 15–29 years).

Mortality

Table 3 presents total female breast cancer deaths by total and sole ethnicity in New Zealand during the years 1996–2000. A total of 2577 deaths were analysed for unadjusted, NHI-adjusted and ever Maori-adjusted estimates and 2560 deaths for NZCMS-adjusted. The proportion of total Maori women ranged from 7.3% to 9.9%. Sole Maori proportions were similar with estimates ranging from 7.1% (NHI) to 9.9% (NZCMS).

Table 1. Cancer incidence by total number and percentage Maori and non-Maori for total and sole NZCMS, ever-Maori, NHI, and unadjusted estimates (1996–2000)

	Total				Sole		
	NZCMS	Ever-Maori	Unadjusted	NHI	NZCMS	Unadjusted	NHI
Maori number	843	906	792	649	675	788	600
(%)	(8.1)	(8.6)	(7.5)	(6.2)	(6.3)	(7.5)	(5.7)
non-Maori number	9581	9618	9732	9875	10005	9736	9924
(%)	(91.6)	(91.4)	(92.5)	(93.8)	(93.7)	(92.5)	(94.3)
Total number	10,424	10,524	10,524	10,524	10,680	10,524	10,524

NZCMS= New Zealand Census Mortality Study; NHI=National Health Index.

Table 2. Age-specific relative risk of total and sole, Maori vs non-Maori breast cancer incidence for NZCMS, ever-Maori, NHI, and unadjusted estimates (1996–2000)

Age group (years)	25-29	30-34	35-39	40-44	45-49	50-54	55-59	60-64	65-69	70-74	75-79	80-84	85+
Total													
NZCMS	1.8 (1.1-3.0) H	1.3 (0.9-1.8)	1.3 (1.0-1.6)	1.2 (1.0-1.4)	1.0 (0.8-1.2)	1.0 (0.8-1.1)	0.9 (0.7-1.1)	1.0 (0.8-1.2)	1.2 (1.0-1.5)	1.1 (0.8-1.5)	1.0 (0.6-1.5)	0.8 (0.5-1.4)	0.7 (0.4-1.5)
Ever-Maori	1.7 (1.0-2.8)	1.2 (0.8-1.7)	1.2 (1.0-1.5)	1.2 (1.0-1.5)	1.1 (1.0-1.3)	1.1 (0.9-1.3)	1.0 (0.8-1.2)	1.1 (0.9-1.3)	1.2 (1.0-1.5)	1.2 (0.9-1.6)	1.0 (0.6-1.6)	0.8 (0.5-1.4)	0.8 (0.4-1.6)
Unadjusted	1.6 (0.9-2.8)	1.1 (0.8-1.5)	1.1 (0.9-1.5)	1.1 (0.9-1.3)	0.9 (0.8-1.1)	0.9 (0.7-1.1)	0.8 (0.7-1.0)	0.9 (0.7-1.2)	1.1 (0.9-1.4)	1.0 (0.7-1.4)	0.9 (0.6-1.3)	0.7 (0.4-1.5)	0.6 (0.3-1.4)
NHI	1.3 (0.7-2.4)	0.7 (0.4-1.0)	0.9 (0.6-1.2)	0.8 (0.7-1.1)	0.7 (0.6-0.9) L	0.7 (0.6-0.9) L	0.7 (0.5-0.9) L	0.8 (0.7-1.1)	0.9 (0.7-1.1)	0.9 (0.6-1.3)	0.6 (0.3-1.2)	0.7 (0.4-1.2)	0.5 (0.2-1.3)
Sole													
NZCMS	2.2 (1.1-4.4) H	1.4 (0.9-2.1)	1.3 (0.9-1.8)	1.3 (1.0-1.6)	1.2 (1.0-1.4)	1.1 (0.9-1.3)	1.0 (0.8-1.2)	1.1 (0.8-1.4)	1.3 (1.0-1.7)	1.2 (0.9-1.7)	1.1 (0.7-1.8)	1.0 (0.5-2.0)	1.1 (0.5-2.4)
Unadjusted	3.0 (1.6-5.3) H	1.9 (1.3-2.7) H	1.7 (1.3-2.3) H	1.6 (1.4-2.0) H	1.4 (1.2-1.7) H	1.3 (1.0-1.6)	1.1 (0.9-1.4)	1.2 (1.0-1.5)	1.5 (1.2-1.9) H	1.4 (1.0-1.9)	1.2 (0.8-2.0)	1.1 (0.6-2.0)	1.1 (0.5-2.3)
NHI	2.5 (1.2-4.9) H	0.9 (0.6-1.5)	1.2 (0.9-1.6)	1.2 (0.9-1.5)	1.0 (0.8-1.2)	0.9 (0.8-1.2)	0.9 (0.7-1.1)	1.1 (0.9-1.3)	1.1 (0.9-1.5)	1.1 (0.8-1.6)	0.8 (0.4-1.5)	0.9 (0.5-1.8)	0.9 (0.4-2.2)

H=significantly higher risk (alpha=0.05); L=significantly lower risk (alpha=0.05); NZCMS= New Zealand Census Mortality Study; NHI=National Health Index.

Table 3. Cancer mortality by total number and percentage Maori and non-Maori for total and sole NZCMS, ever-Maori, NHI, and unadjusted estimates (1996–2000)

	Total				Sole		
	NZCMS	Ever-Maori	Unadjusted	NHI	NZCMS	Unadjusted	NHI
Maori number	254	252	239	188	254	239	182
(%)	(9.9)	(9.8)	(9.3)	(7.3)	(9.9)	(9.3)	(7.1)
non-Maori number	2306	2325	2338	2389	2306	2338	2395
(%)	(90.1)	(90.2)	(90.7)	(90.7)	(90.1)	(90.7)	(92.9)
Total number	2560	2577	2577	2577	2560	2577	2577

NZCMS= New Zealand Census Mortality Study; NHI=National Health Index.

Table 4. Age-specific relative risk of total and sole, Maori vs non-Maori breast cancer mortality for NZCMS, ever-Maori, NHI, and unadjusted estimates (1996-2000)

Age group (years)	25-29	30-34	35-39	40-44	45-49	50-54	55-59	60-64	65-69	70-74	75-79	80-84	85+
Total													
NZCMS	4.7 (1.5-14.8) ^H	2.4 (1.2-4.8) ^H	2.2 (1.4-3.5) ^H	2.0 (1.3-3.1) ^H	1.7 (1.2-2.5) ^H	1.7 (1.2-2.3) ^H	1.8 (1.3-2.5) ^H	1.0 (0.6-1.7)	1.0 (0.6-1.7)	1.2 (0.7-2.0)	1.8 (1.1-3.1) ^H	1.3 (0.6-2.9)	0.9 (0.4-2.4)
Ever-Maori	4.0 (1.2-13.0) ^H	2.0 (1.2-3.4) ^H	2.0 (1.2-3.4) ^H	1.8 (1.2-2.8) ^H	1.8 (1.2-2.6) ^H	1.6 (1.1-2.3) ^H	1.7 (1.3-2.3) ^H	1.0 (0.7-1.6)	1.1 (0.7-1.8)	1.3 (0.8-2.1)	1.6 (0.9-2.8)	1.1 (0.5-2.7)	0.9 (0.4-2.5)
Unadjusted	4.0 (1.1-15.1) ^H	2.0 (1.0-4.0)	2.0 (1.2-3.5)	1.7 (1.1-2.7) ^H	1.6 (1.1-2.3) ^H	1.6 (1.1-2.3) ^H	1.7 (1.3-2.3) ^H	0.9 (0.6-1.5)	0.9 (0.5-1.5)	1.1 (0.6-2.0)	1.6 (0.9-2.8)	1.1 (0.5-2.6)	0.7 (0.2-2.3)
NHI	2.9 (0.9-9.9)	1.7 (0.9-3.3)	1.2 (0.7-2.0)	1.6 (1.1-2.4) ^H	1.3 (0.8-1.9)	1.0 (0.7-1.5)	1.2 (0.8-1.7)	0.7 (0.4-1.4)	0.8 (0.4-1.3)	0.9 (0.5-1.6)	1.1 (0.6-2.1)	1.1 (0.5-2.6)	0.6 (0.1-2.1)
Sole													
NZCMS	5.6 (1.8-17.1) ^H	2.6 (1.2-5.5) ^H	2.3 (1.3-4.0) ^H	2.1 (1.4-3.3) ^H	2.0 (1.3-3.0) ^H	1.9 (1.4-2.7) ^H	2.0 (1.4-2.9) ^H	1.1 (0.7-1.9)	1.1 (0.7-1.9)	1.4 (0.8-2.5)	2.0 (1.2-3.6) ^H	1.6 (0.7-3.9)	1.3 (0.4-3.8)
Unadjusted	7.6 (2.6-22.5) ^H	3.4 (1.7-6.7) ^H	3.1 (1.9-5.1) ^H	2.7 (1.8-4.0) ^H	2.4 (1.6-3.4) ^H	2.3 (1.6-3.2) ^H	2.3 (1.7-3.1) ^H	1.2 (0.7-2.0)	1.2 (0.7-2.2)	1.6 (0.9-2.8)	2.2 (1.2-4.1) ^H	1.7 (0.6-4.7)	1.3 (0.4-3.9)
NHI	5.6 (1.6-18.8) ^H	3.0 (1.4-6.1) ^H	1.8 (1.0-3.2)	2.3 (1.4-3.6) ^H	1.7 (1.1-2.7) ^H	1.5 (1.0-2.2)	1.5 (1.0-2.3)	1.0 (0.6-1.7)	1.1 (0.6-1.9)	1.2 (0.7-2.2)	1.5 (0.8-3.0)	1.4 (0.5-3.6)	1.0 (0.2-3.7)

H=significantly higher risk (alpha=0.05); NZCMS= New Zealand Census Mortality Study; NHI=National Health Index.

Figure 1. Age-specific relative risk (Maori vs non-Maori) of breast cancer incidence for total and sole NZCMS, ever-Maori, NHI, and unadjusted estimates (1996-2000) (NZCMS=New Zealand Census Mortality Study; NHI=National Health Index)

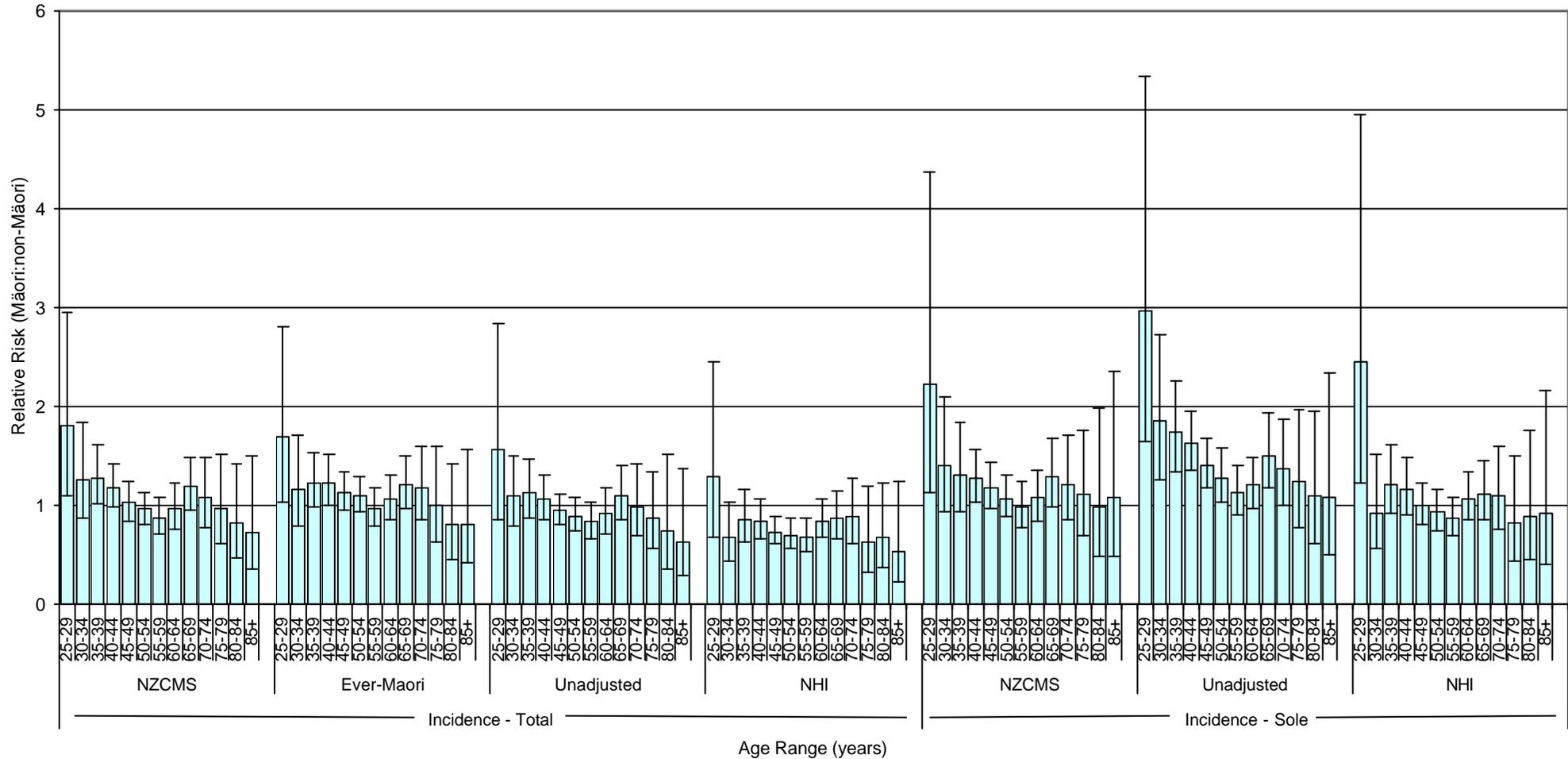


Figure 2. Age-specific relative risk (Maori vs non-Maori) of breast cancer mortality for total and sole NZCMS, ever-Maori, NHI, and unadjusted estimates (1996-2000) (NZCMS=New Zealand Census Mortality Study; NHI=National Health Index)

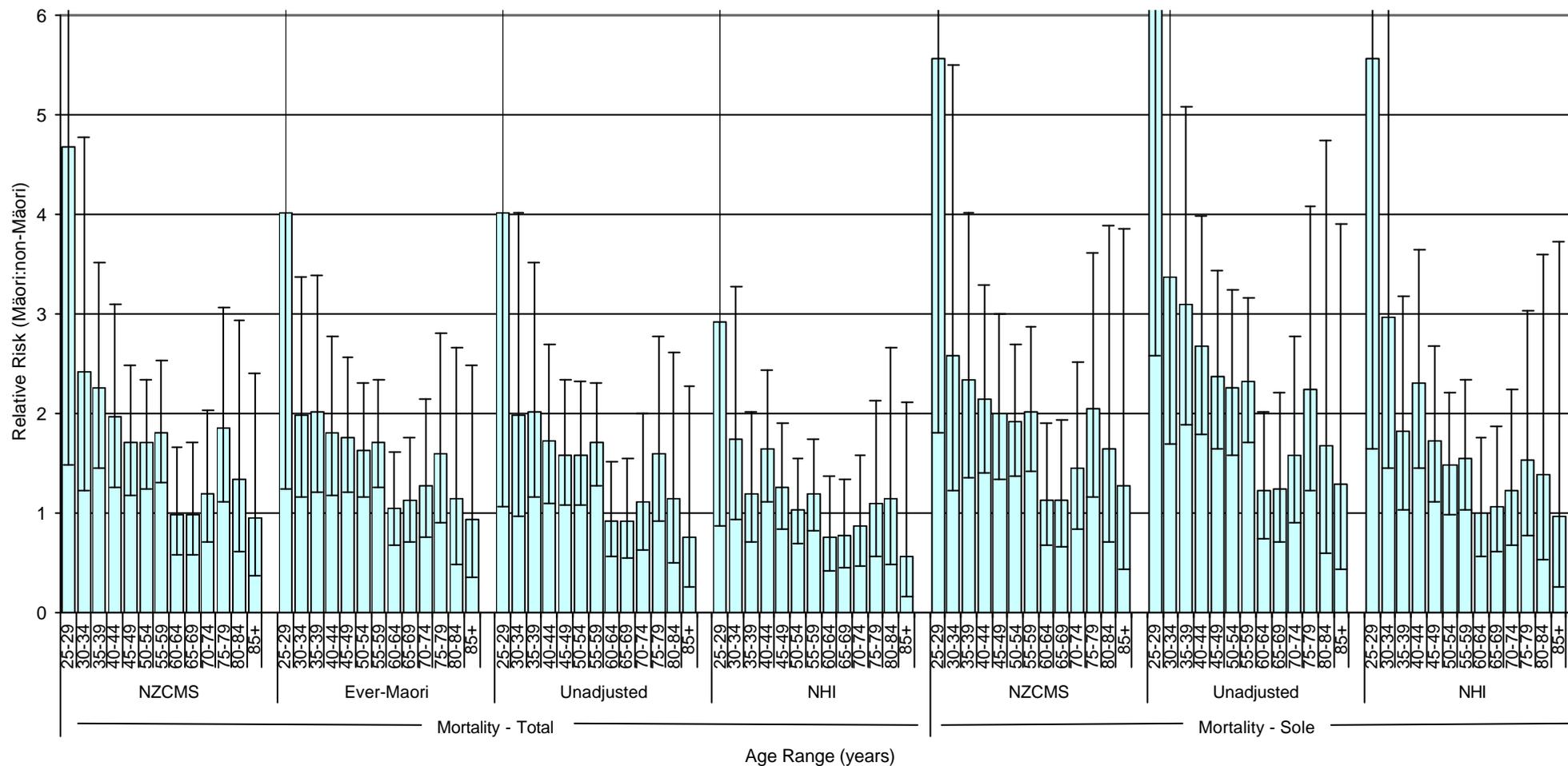


Table 4 presents age-specific relative risk (Maori vs non-Maori) of breast cancer mortality for the unadjusted, NHI, ever-Maori, and NZCMS-adjusted estimates. Similar to breast cancer incidence findings, the relative risk estimates for mortality are greater for sole Maori than for total Maori ethnicity.

With respect to total Maori mortality findings, the NHI-adjusted results are least consistent with the NZCMS-adjusted estimates. This is followed by unadjusted and ever-Maori estimates that both have a similar risk pattern to the NZCMS-adjusted estimates.

With respect to sole Maori mortality findings, the unadjusted results are least consistent with the NZCMS-adjusted estimate, overestimating the sole Maori risk of death when compared with the NZCMS-adjusted estimate. Although the NHI sole estimate still differs from the NZCMS 'gold standard' one, this difference is less than the unadjusted sole Maori estimate. This is to be expected because data obtained from the National Health Index underestimate the number of Maori. Therefore (in the case of sole Maori estimates) NHI adjustment reduces the overestimation of sole Maori numbers found in unadjusted estimates. This results in sole Maori NHI findings being more closely aligned to the NZCMS 'gold standard' than unadjusted sole Maori findings (the opposite to what was found for total Maori results).

Figures 1 and 2 present the age-specific relative risk of total Maori and sole Maori breast cancer incidence and mortality for each estimate. As noted previously, NHI-adjusted and unadjusted estimates are least similar to NZCMS-adjusted estimate, with ever Maori-adjusted risk estimates closely approximating NZCMS-adjusted estimates for both incidence and mortality data.

Overall, the majority of confidence intervals across the seven incidence estimates include one, and therefore the age-specific relative risks are not significantly different at the 95 % level of confidence. The risk ratios for the under-30 age groups should be interpreted cautiously given the low incidence rates in these age groups. In contrast to breast cancer incidence findings, confidence intervals for Maori vs non-Maori mortality relative risks in women aged 25-59 years did not include one and therefore are statistically significant at the 95% level of confidence

Discussion

This study found different breast cancer incidence and mortality rates when different methods were used to estimate Maori and non-Maori ethnicity. These results confirm previous findings of the unreliable quality of ethnicity data in routinely collected datasets.

With respect to total findings for mortality, the NHI-adjusted and unadjusted estimates were least similar to NZCMS-adjusted estimates. These findings were predicted and are consistent with other analyses.¹² However, it is concerning that ethnicity data obtained from mortality datasets after 1995 continue to undercount Maori despite attempts to align ethnicity collection with that of the census during this period. This highlights the ongoing presence of numerator/denominator bias and reinforces the need to assume an undercount of Maori in mortality datasets. Accordingly, the method used in this paper to reassign ethnicity using NZCMS derived adjustors should be considered routinely. Of note, the ever-Maori estimate

closely approximates the NZCMS-adjusted estimate representing an alternative option for adjustment.

Consistent with other ethnic analyses, this study found that the mortality risk was higher for women who identified solely as Maori compared with women who identified themselves as belonging to more than one ethnic group.²⁹ One proposed explanation for this finding is that people identified as Maori (assumed to be more likely for the sole Maori ethnic group) are at increased risk of institutional racism and differential health care access, and therefore differential health outcomes.²⁹ This hypothesis supports the ongoing examination of breast cancer incidence and mortality rates by both sole and total Maori ethnicity.

Concerns about the validity of applying the NZCMS adjustor to cancer registration data remain. While this analysis supports the application of mortality adjustors to morbidity data, the findings are suggestive rather than conclusive. In particular, we cannot conclude whether the use of NZCMS or ever Maori-adjusted morbidity data is appropriate in other contexts—e.g. data collected prior to 1996, fewer years of data, or data reviewing rarer diseases or health outcomes with lower incidence.

The relatively short study period and modest number of breast cancer events may affect the pattern observed between the different estimates, and a similar analysis on a larger dataset would be worthwhile. Ideally, cancer registration data should be linked with census information to quantify the ethnicity misclassification as in the New Zealand Census Mortality Study. This analysis is currently underway at the Wellington School of Medicine (personal communication, Dr Martin Tobias, 2004) and will aid future ethnic analyses of breast cancer incidence.

Despite these limitations, this study represents the first time an analysis of cause-specific incidence and mortality has been performed in New Zealand using and comparing multiple methods of ethnicity adjustment. In addition, these estimates are the best available for age-specific Maori rates of breast cancer incidence and mortality, and they represent a significant advance in the quality of ethnicity data on which to base breast cancer screening policy and treatment interventions. The use of a Kaupapa Maori Research approach within a quantitative setting has provided new information that will guide the approach to future analyses of breast cancer data in Maori and non-Maori women.

In conclusion, NZCMS or ever Maori-adjusted breast cancer incidence and mortality estimates are the measures of choice for analysing Maori data. If unadjusted or NHI data have to be used, then the undercount of Maori in the data should be acknowledged. This paper reinforces the need to improve the collection and analysis of both sole and total ethnicity data in New Zealand so that Maori vs non-Maori disparities in breast cancer can be appropriately identified, monitored, and eventually eliminated.

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Anxiety before, during, and after participation in a population-based screening mammography programme in Waikato Province, New Zealand

Margaret Brunton, Claire Jordan, Ian Campbell

Abstract

Aim This study investigated anxiety levels before, during and after mammography in the Waikato breast cancer screening pilot.

Method A sample of 1085 women on the Waikato database were sent survey questionnaires, which included questions about the anxiety experienced. Data from 584 completed questionnaires were obtained.

Results Two significant findings were identified. The first was that population-based screening can ultimately reduce anxiety for participants who receive a clear result from their mammogram. The second finding was that levels of worry throughout were related to ethnicity. Maori and Pacific Island women reported higher levels of worry than New Zealand European and Asian women about developing breast cancer ($p < 0.001$), while awaiting their appointment ($p = 0.041$) and results ($p = 0.046$). Across all groups, levels of worry about developing breast cancer were also related to level of education ($p = 0.018$), a family history of breast cancer ($p = 0.002$), stress levels during screening mammography ($p < 0.001$), and experience of pain during the procedure ($p < 0.001$). At least some months following receipt of their results, 67% (95% CI 63-71) of all women experienced reassurance from receiving a clear result.

Conclusions The results show that the population-based screening programme demonstrates greater potential to ultimately relieve (rather than increase) anxiety for participants who receive a clear result from their mammogram.

This research investigated how participation in a free-of-charge, population-based, breast-cancer-screening pilot programme influenced levels of anxiety in a sample of New Zealand women in the Waikato region. Breast cancer is the most common cancer among women in New Zealand. Ten percent of women will experience breast cancer over their lifetime, and incidence increases with age. Eighty-four percent of cases will occur in those over 54 years old.

The Ministry of Health's most recent report illustrates that 25% of all cancer registrations and 20% of all cancer deaths among New Zealand women result from breast cancer.¹ Mortality rates are higher in the ethnic minority groups of Pacific Island women, who tend to present later with advanced disease.² Furthermore, there is a similar trend for indigenous Maori.³ In the continuing absence of an effective means of primary prevention or a 'statistical cure of symptomatic invasive breast cancer',⁴ screening mammography currently offers the best means to reduce mortality in women aged fifty years and over.⁵

However, one of the potentially adverse effects of population-based screening mammography programmes is that they can contribute to anxiety in the population,

and this may even extend to women who are not eligible (e.g. because of age).⁶ For eligible women, although anxiety tends to be associated with outcome risks of screening mammography such as false positives,⁷ anxiety has also been associated with the invitation process⁸ and with exposure to radiation during screening mammography.^{9,10} It has even been argued that screening mammography programmes are undesirable because they can induce anxiety.¹¹

Accordingly, as the success of the programme requires ongoing participation, and anxiety associated with the process or outcome can inhibit participation,¹² it is important to understand how women experience or perceive anxiety when participating in a screening mammography programme.

Method

There were 14,392 women registered on the database of the Waikato screening mammography pilot programme, who had participated in the third round of screening between November 1995 and April 1998. Participants had consented to participate in evaluation surveys of the pilot programme. They had also been asked to nominate their ethnicity on the enrolment form.

A sample of 1,100 women (grouped by region, age, and ethnic group) were randomly selected from the database by programme staff. To ensure adequate numbers, Maori were oversampled. Specific ethnic samples were selected to represent 35% of Maori and 4% of the New Zealand European groups of women on the database. Because of the small numbers registered on the database, all women of ethnic groups other than New Zealand European and Maori were selected. Women who developed cancer were excluded from the screening database, and so were not included in the sample. However, women who had been recalled in the past (6%), and whose mammograms had not indicated the presence of cancer, were included.

A questionnaire was developed to assess how worried participants had felt at various stages of the screening mammography process. The level of anxiety was measured using a 4-point Likert scale. Following approval from the Waikato Ethics Committee, a two-stage pre-test was conducted among seven groups of a total of 60 women.

In October 1999, a total of 1,085 survey questionnaires were distributed. Excluding the 34 returned as 'not available' (8 deceased, 4 living overseas, 1 male) or with 'gone, no address' the net number of questionnaires distributed was 1051. A reminder letter was sent to those who had not responded within 5 weeks. To enhance data recovery from the limited number of Pacific Island women on the database, most of their questionnaires were hand delivered and collected by health workers employed by the screening programme to promote participation among eligible women.

Of the 639 (61%) questionnaires returned, 611 were completed satisfactorily. Of the 639 (61%) questionnaires returned, 611 were completed satisfactorily. Of these 611 women, 584 were in the sample frame of four ethnic groups—i.e. aged between 50 and 64 years (the eligible age screening mammography at the time). (The group of 15 women categorised as 'other', and the group of 12 women over the age of 64 were not included in any ethnic or age-based analyses carried out, which provided a usable response rate of 56%).

Demographic information about the respondents, including ethnic origin, is illustrated in Table 1 below. Statistical analysis was performed using SPSS (Release 8.0.0 22/12/97). Tests of significance were applied using chi-squared tests for independence in contingency tables.

Table 1. Demographic information obtained from the 584 survey respondents

Age		Domicile	
50-54	224	In a city	182
55-59	204	In a rural town	313
60-64	156	In the country	89
Ethnic origin		Occupation	
Maori	153	Wages or salary	269
New Zealand European	339	Unpaid work in home	121
Pacific Island	51	Self employed	72
Asian	41	Retired	122
Level of education		Annual income (NZ dollars)	
Primary School	28	Less than \$15,000	132
Secondary School	366	\$15,000 to \$30,000	147
University	68	\$30,001 to \$50,000	105
Trade or Polytech	43	Greater than \$50,000	66
Other sources	79	Don't wish to answer	134

Table 2. Frequencies of levels of worry at various stages of mammogram

Level of worry	General level of worry about breast cancer	Level of worry awaiting mammogram	Level of worry awaiting results of mammogram	Level of reassurance	After mammogram
Not at all	135 (23%)	258 (44%)	186 (32%)	Felt reassured	393 (67%)
A little bit	288 (49%)	265 (45%)	294 (50%)	Felt the same	184 (32%)
Quite	80 (14%)	38 (7%)	75 (13%)	Felt more worried	7 (1%)
Very	81 (14%)	23 (4%)	29 (5%)		
Total	584	584	584		584

Results

The respondents were experienced in mammography screening, with over 80% having had two or more screening rounds. Most respondents (71%) reported that their most recent mammogram had been within the previous 12 months. When asked about the worry or level of stress they experiences at various stages of the screening process, women volunteered 1248 additional comments, resulting in several pages of open and detailed feedback.

General level of anxiety about breast cancer

Respondents were asked about how worried they normally felt about getting breast cancer (Table 2). The number of respondents expressing either no or little worry was significantly greater than the number expressing that they were either quite or very worried ($p < 0.001$). There were no significant differences in higher levels of worry about breast cancer between women participating in initial (prevalent) and subsequent (incident) rounds of screening ($p = 0.587$). Demographic variables of age ($p = 0.649$), domicile ($p = 0.210$), occupation ($p = 0.460$), and annual income ($p = 0.308$) did not have a significant effect on the level of general anxiety about breast cancer.

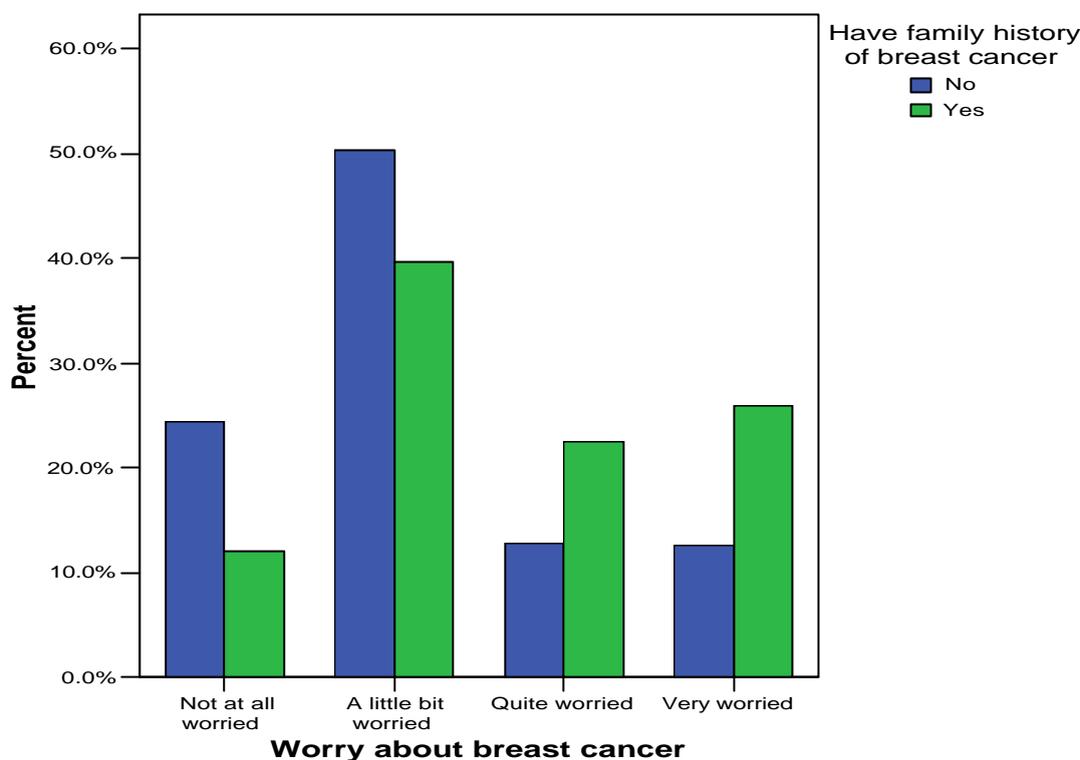
However, ethnicity proved to be highly significant ($p < 0.001$) with Maori and Pacific Island women showing much higher levels of worry than their New Zealand European or Asian counterparts. Level of education also proved significant ($p = 0.018$), with those having a higher level of education indicating lower levels of worry about breast cancer. The 68 women who had a higher level of education (university educated) reported lower levels of worry about breast cancer (19% were 'quite' or 'very' worried). This compared to 26% of the 366 women educated to secondary school level and 39% of the 28 women who had been educated only to primary school level.

Fifty-eight women reported a family history of breast cancer, and levels of worry for this group were significantly greater ($p = 0.002$) than those who reported no family history of breast cancer (Figure 1). Twenty-five percent of those with no family history of breast cancer reported feeling 'quite' or 'very' worried about the possibility of breast cancer, compared with 48% of those who did report a family history of breast cancer. However, this association between level of worry and family history was only significant for women aged 50-54 years ($p < 0.001$) (55-59 years, $p = 0.068$; 60-64 years, $p = 0.345$).

Anxiety awaiting appointment for screening

Women were asked about the level of worry they experienced prior to the appointment (Table 2), once they had made a decision to participate in a screening mammogram. For most respondents, waiting for their appointment was not a time of high levels of worry ($p < 0.001$) with 89% of respondents indicating that they were 'not at all' or 'a little bit' worried. This level of worry was not associated with the number of mammograms previously undertaken ($p = 0.595$). Ethnicity was the only significant demographic variable, with Pacific Island women (10%) and Maori women (6%) expressing higher levels of feeling 'very worried' than Asian (2%) or New Zealand European women (2%, $p = 0.041$). Several women commented that the difficulties they experienced in obtaining suitable appointments contributed to their level of anxiety at this stage of the process.

Figure 1. Worry about breast cancer by family history of breast cancer



Anxiety during the process

To assess how many respondents found the physical process of screening mammography stressful, they were asked what level of relaxation or stress they experienced during their mammogram. Most women reported feeling relaxed or quite relaxed (87%), and the numbers of mammograms experienced was not significant ($p=0.383$). However, the experience of pain or discomfort during the mammography was directly related to the level of stress reported during screening ($p<0.001$).

The 161 women who reported they were either 'quite' or 'very' worried about the possibility of breast cancer did experience higher levels of stress during their mammogram (20%), compared to the 423 women who had only minor, or no level of worry about breast cancer (10%, $p<0.001$). Twelve percent of women who reported feeling stressed and 2% of women who reported feeling relaxed during screening mammography, said they would either reconsider or refuse further screening ($p<0.001$). The level of anxiety experienced during the process was not significantly related to any demographic variables, but was related to the level of worry experienced while awaiting the appointment and the level of worry about breast cancer in general, as shown in Table 3.

Table 3. P values for significance of relationship between experience of relaxation or stress during mammography and other variables

Demographic information	Ethnic Origin	Domicile	Age	Occupation	Annual Income	Level of Education
P value	0.432	0.160	0.201	0.488	0.509	0.707
Feelings during mammogram						
	Worry while awaiting appointment			General level of worry about breast cancer		Total
	None/Little	Quite/Very		None/Little	Quite/Very	
Relaxed	475 (93%)	34 (7%)		381 (75%)	128 (25%)	509
Stressed	48 (64%)	27 (36%)		42 (56%)	33 (44%)	75
Total	523	61		423	161	584
	P < 0.001			P = 0.001		

Anxiety while awaiting results

Eighty-two percent of women reported feeling ‘not at all’ or ‘a little bit’ worried while awaiting the results of the mammogram (Table 2). This was related to the way women felt during the mammogram ($p < 0.001$), with 91% of those who felt relaxed during the mammogram feeling ‘not at all’ or ‘a little bit’ worried while awaiting the results. Demographic variables demonstrated no significant relationship with the number of mammograms undertaken ($p = 0.663$), age ($p = 0.564$), domicile ($p = 0.316$), income ($p = 0.180$), occupation ($p = 0.168$), or level of education ($p = 0.140$).

Once again, however, a significant relationship was observed with ethnicity ($p = 0.046$). Twenty-three percent of Maori and Pacific Island women reported feeling ‘quite’ or ‘very’ worried, compared with 15% of New Zealand European or Asian women. The 58 respondents with a family history of breast cancer also experienced higher levels of worry while awaiting their results (28%), than those without a family history of breast cancer (17%, $p = 0.035$). The level of anxiety at this stage was not significantly related to the length of time spent waiting for the results ($p = 0.450$).

Effect of experience on plans for future mammography

Respondents were asked if they planned to have a mammogram when next called by the programme. A significant relationship was identified between the level of anxiety during the process and plans for future screening ($p \leq 0.001$), with 96% of those feeling relaxed during the process stating they would agree to take part in future screening, compared with 79% of those who reported feeling stressed during the process.

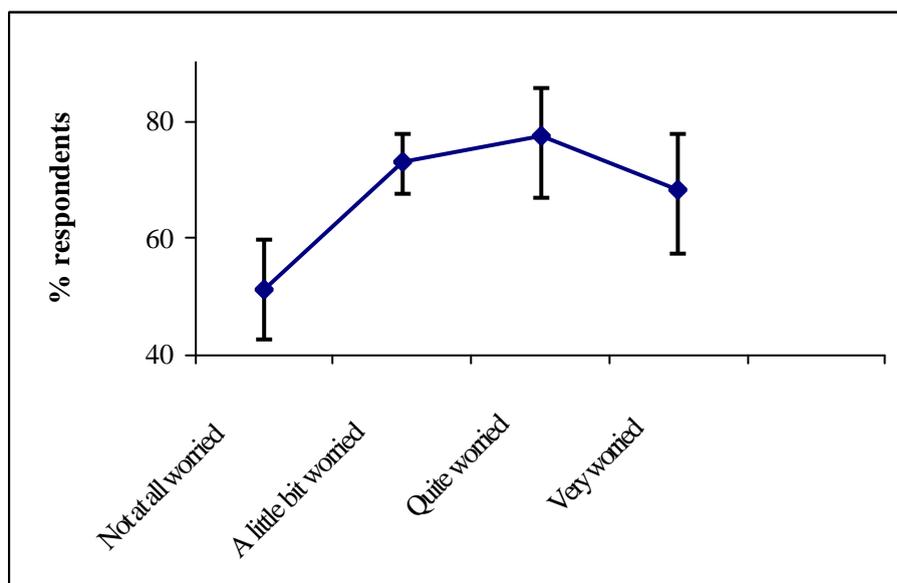
Long-term anxiety following mammogram

It was also important to assess what effect the outcomes of screening mammography may have on population levels of anxiety about breast cancer. Respondents were therefore asked whether having a screening mammogram had influenced their level of

worry about breast cancer (Table 2). Significantly more women (67%) reported they felt reassured, or less worried by the process; 32% felt the same and only 1% felt more worried ($p < 0.001$). No demographic variables had a significant effect on level of worry at this stage of the process; however, those who felt 'more' worried all reported that some abnormality had been identified on their mammogram.

When responses were compared with the reported level of worry about breast cancer prior to screening, even 51% (95% CI: 43–60) of those who women were 'not at all worried' still obtained reassurance from their mammogram (Figure 2). There were no significant differences in level of reassurance obtained across experience of mammography, with 67% of women who had participated in one round of screening experiencing reassurance, compared with 69% of those women who had participated in two or more rounds of screening ($p = 0.875$).

Figure 2. Percentage of respondents (with 95% confidence intervals) who obtained reassurance from screening mammography related to their initial level of worry about breast cancer



Discussion

It has been suggested that fear of cancer is 'one of the most powerful sources of anxiety during screening mammography.'¹³ In this study, none of the women surveyed were currently scheduled or invited for a mammogram, however 77% of them reported that they normally felt some level of anxiety about the possibility of breast cancer. It is suggested that women with a family history of breast cancer may be so worried that they are less likely to participate in regular screening mammography.¹⁴

Women with a family history of breast cancer in this research were more likely to report higher levels of worry about breast cancer (including the procedure, and

awaiting results) than those women without a family history of breast cancer. Also, lesser-educated women, as well as Maori and Pacific Island women, reported higher levels of worry about the possibility of breast cancer than more highly educated Asian or New Zealand European women. Nonetheless, despite experiencing some level of anxiety at various stages of the process, most women felt reassured on completion of their mammogram. Overall, the majority of respondents (67%) felt reassured once their mammogram was completed and they received a clear result.

The low percentage of women experiencing any level of stress during the physical process of mammography confirms the findings from an earlier Waikato survey that fear of the procedure itself appears to be a minor factor for respondents.¹⁵ However, the significant relationship between higher levels of anxiety at this time and reconsideration of further participation in screening mammography demonstrates the importance of addressing anxiety during the process. Furthermore, although the level of recalled pain in this research is within the expected range of 5%–15% of women who report severe pain on mammography,¹⁶ the stress that was reported during the physical process of screening mammography was significantly related to pain ($p < 0.001$), providing support for earlier findings.^{17,18}

The same women who reported higher levels of anxiety while waiting for appointments also reported higher levels of worry while awaiting results. This result is similar to that in other New Zealand studies, which reported that there were no significant differences between Waikato respondents' level of worry while waiting for appointments and results,¹⁹ as was also the case with participants in the Southland-Otago Pilot Programme.²⁰ This research revealed that Maori and Pacific Island women reported higher levels of anxiety than New Zealand European or Asian women both while awaiting appointments ($p = 0.041$) and results ($p = 0.046$).

Ultimately, however, the level of reassurance that women received from their mammograms, provided the result was clear, appeared to outweigh any anxiety they experienced beforehand. The women who participated in this study reported a high level of support for screening mammography. The majority viewed screening mammography as very important (88%) or important (11%). Even the 32% who said that their level of worry was unchanged still regarded the programme as an important part for their health management. Their ongoing participation and high level of commitment suggests that the benefits, including reassurance, far outweigh any risk of anxiety that women may experience during the process.

The process of screening mammography is associated with varying levels of worry for women. As every woman in this study had prior experience of mammography, the levels of anxiety in relation to screening may be underestimated in this survey. Because high levels of worry about breast cancer may already be prevalent in the 'at risk' population regardless of whether they are currently undergoing screening mammography, and this may deter some women from attending, further research is required to identify specific and appropriate interventions to deal with this anxiety. Similarly, the link between pain, anxiety and future intentions to attend warrants further investigation, as this research is limited to women who have chosen to participate in a pilot programme.

Although it is a potential source of bias in this study that it is based on 56% of those surveyed, to address the variable of non-responders in the research design, the 213

surveys returned after the reminder letter was sent in late November were coded as 'late returns' and compared with those of earlier returns. There were no significant differences among variables between respondents in the two categories, such as experience of anxiety, pain or reassurance obtained from mammography, which suggests that the sample was likely to be representative of the remaining population of non-responders in this study.

As the first New Zealand study of the screening mammography programme to identify ethnic subgroups, this research has indicated that Maori, Pacific Island, and lesser-educated women demonstrate significantly higher levels of worry both about breast cancer and some aspects of screening mammography. The propensity of the same ethnic groups to respond differently to invitation strategies from the programme may also be an outcome of their increased levels of anxiety.²¹ Accordingly, it is important to identify the specific needs of these women to address their current under-representation in the screening programme, so that they may obtain the benefits of reassurance and early detection.

Overall, however, contrary to suggestions from other researchers,²²⁻²⁵ this study does not demonstrate that screening mammography raises the ongoing level of anxiety in this population of women. The reverse had been shown. The majority of women felt reassured following their mammogram, and levels of anxiety about breast cancer were diminished.

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Use of early magnetic resonance imaging in the diagnosis of occult scaphoid fractures: The CAST Study (Canberra Area Scaphoid Trial)

Sashi Kumar, Alan O'Connor, Mervyn Despois, Howard Galloway

Abstract

Aim To establish the effectiveness of early magnetic resonance imaging (MRI) in the diagnosis of scaphoid fracture in patients with suggestive clinical findings (but a normal initial X-ray) in an Australian major-referral emergency department

Methods A prospective study of patients who presented within 24 hours after trauma with clinical findings suggestive of a scaphoid fracture but no evidence of fracture on the initial X-ray. MRI was performed within 24 hours of presentation. Clinical review in patients with normal initial MRI was carried out in 10 days and repeat MRI was carried out when clinically indicated.

Results A total of 22 patients were enrolled. Early MRI within 24 hours revealed 6 scaphoid fractures, 2 distal radial fractures and a hamate fracture. Thirteen patients had no fracture on the initial MRI. Upon clinical review in 10 days, 5 of these patients were clinically cleared and discharged. Eight of these patients underwent repeat MRI none of which revealed a fracture.

Conclusions In an Australian tertiary hospital with MRI facility, the early use of MRI is a sensitive and practical way to diagnose occult scaphoid fractures and saves unnecessary immobilisation.

Scaphoid fractures occurs in young healthy individuals,¹ and is the most commonly fractured bone of the wrist (50%–80% of all carpal bone injury).² It is also the most common undiagnosed fracture³ due to two factors:

- Tenderness in the anatomical snuff-box has a sensitivity of 90% but a specificity of 40%.⁴ The positive predictive value of the clinical examination (21%) implies that four out of five patients will not have a scaphoid fracture.⁵
- Scaphoid fractures commonly present as occult fractures (20% to 25%^{2,6}) whereby the initial X-rays are negative. The fracture may not be evident on X-ray until 10 to 14 days after injury when there is resorption of the fracture margins.

Early diagnosis is essential to avoid complications of delayed union, non union (5%–12%),^{12,13} avascular necrosis and resultant long-term disability.

Several studies have confirmed the value of MR imaging to diagnose scaphoid fractures which are not visible on the initial plain X-ray.^{3,5,8} Most studies have shown MRI to be 100% sensitive when performed after 2.8 days.^{1,3} Hunter performed MRI 7 days post-trauma and found it to be 100% sensitive in 36 patients.⁸

Bretlau et al showed that MRI was sensitive when performed at a mean of 4 days after trauma, and showed that no new fractures were found on a follow-up MRI at a mean

of 11 days¹ in 52 patients. Gaebler performed MRI on 32 patients 2.8 days post trauma with a sensitivity of 100% and a confidence interval of 95%³.

Methods

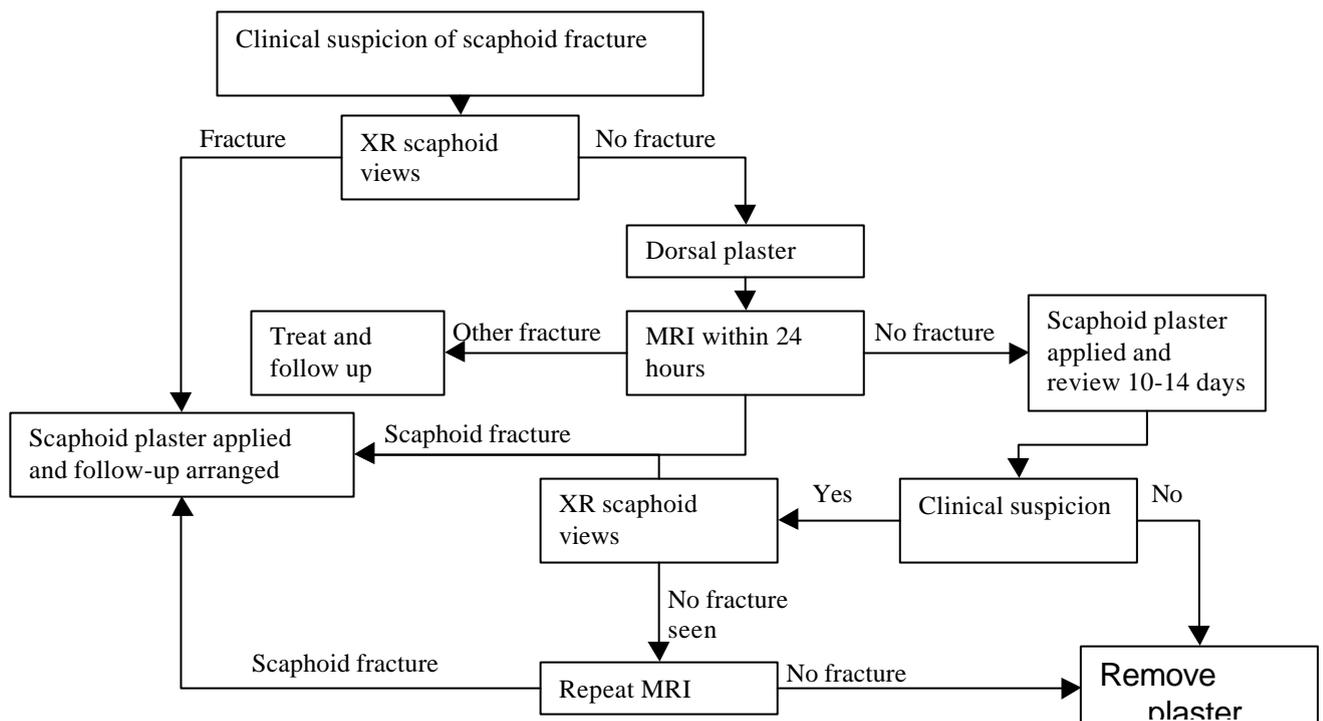
This was a prospective study conducted as a joint collaboration between the Departments of Emergency Medicine and Medical Imaging at The Canberra Hospital from May 2001 to September 2002. Patients who presented following a fall on the outstretched hand (with localised pain in the anatomical snuff-box and no evidence of a fracture on the initial scaphoid X-ray series) were recruited to the CAST study following an informed consent. Ethics approval was granted by the ACT Health and Community Human Research Ethics Committee for this study. An MRI was organised within 24 hours of the trauma and was performed between the daily scheduled MRI patients.

The patients were scanned on a 1.5 T Siemens Vision MRI with small flex coil using a shortened wrist MRI protocol consisting of a coronal T1W SE and coronal STIR sequences: thickness 2 mm with a gap of 0–0.1 mm, field of view 160 mm, and matrix 220 x 512. The scanning time was 7 minutes and the examination lasted in all less than 20 minutes.

The patients who had a negative MRI scan were placed in a scaphoid cast and were reviewed in 10 days. Patients who no longer had clinical signs on review were discharged with no further imaging or treatment, while those with persistent snuff box tenderness underwent a repeat MRI.

The following clinical pathway describes the flow of patients through this process and identifies the steps in which the diagnosis was made or when the patient was discharged.

Figure 1. Management of suspected scaphoid fracture



Results

Over the 17 months of the study, 22 patients were recruited with suspected occult scaphoid fracture with normal initial X-ray. There were 5 female and 17 male patients with a mean age of 27 years.

MRI diagnosed six scaphoid fractures in this group of patients. The distribution of these fractures was: three through the waist and three through the distal pole. Thirteen patients did not have a fracture on the initial MRI.

MRI criteria for an occult fracture was a linear zone of low signal intensity on T1W or STIR on a background of marrow oedema. Bone bruising was diagnosed when marrow oedema was present but no fracture line seen.

Findings in other patients included: two fractures of the distal radius, one fracture of the hamate, and one lytic lesion of the distal radius (which was later diagnosed as gout). Three of these patients (one scaphoid fracture, one hamate fracture, and one gout case) had a repeat MRI at 10 days which confirmed the previous MRI findings. Twenty-two clinically suspected occult scaphoid fractures all had MRI within 24 hours (Table 1):

Table 1. Diagnosis of 22 clinically suspected occult scaphoid fractures

N=22	Diagnosis
6	Scaphoid fractures
2	Distal radius fracture
1	Hamate fracture
1	Gout
12	No fractures or bone bruise only

No fracture was seen on the first MRI in the remainder 12 patients. Three of the latter who did not return for their repeat MRI were contacted by telephone and confirmed they were totally symptom free and declined to have another MRI. In two patients, a clinical decision was made not to proceed with further imaging as they were totally asymptomatic and did not have any snuff-box tenderness upon clinical review at 10 days. The seven remaining symptomatic patients had a repeat MRI which did not reveal a fracture.

Table 2. Follow up (first MRI) of the 12 patients without fractures

N=12	Observation
7	No fracture seen
3	Failed to return for the repeat MRI but symptom free when contacted by phone
2	Clinical review at 10 days did not reveal any tenderness and no further imaging was required

Repeat MRI at 10 days did not detect any new scaphoid fractures and so did not alter the management.

The sensitivity of the 24-hour MRI is 100% (confidence interval 72.3–100) with a specificity of 100% (confidence interval 75.8–100). The wide confidence interval reflects the small sample size.

Case 1

History—16-year-old male presented after fall with tenderness in the snuff-box.

Findings—MRI: marrow oedema through the scaphoid on STIR with fluid around the distal pole and linear hypo intense line on T1W.

Diagnosis—scaphoid fracture.

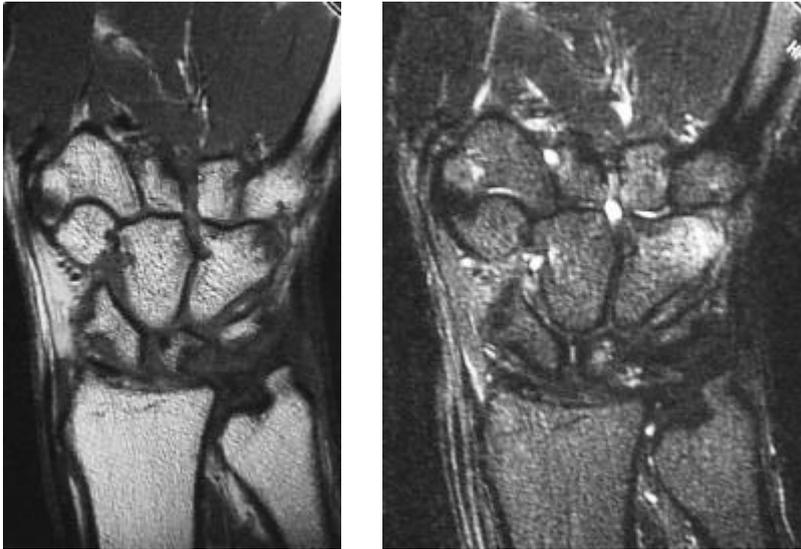


Case 2

History—23-year-old male; fall; tenderness in snuff box.

Findings—MRI: irregular hypo intense line on T1W in the hamate with surrounding oedema on STIR.

Diagnosis—fracture of hamate distally.



Case 3

History—17-year-old male; fall on outstretched hand.

Findings—MRI: marrow oedema distal radius on STIR. Hypo intense line on T1W in distal metaphysis

Diagnosis—Fracture of distal radius



Discussion

The current treatment algorithm of occult scaphoid fractures is applying a scaphoid plaster cast, X-ray in 10 to 14 days, and organising an MRI or a bone scan if focal tenderness persists³ with no fracture on the X-ray. This algorithm raises the issue of unnecessary immobilisation. The negative predictive value of negative initial radiographs (74%) implies that three out of four patients will be needlessly immobilised.⁵

This leads to over treatment in patients who do not have a scaphoid fracture on follow-up studies.^{10,11} These patients are also young and active which leads to loss of productivity and adds to the economic costs of this injury. Another problem with this approach is the loss of follow up of a large percentage of these patients who fail to return for review in 10 days.

A retrospective review of patients seen in the Emergency Department at Canberra Hospital between April 1999 and March 2000 (with suspected scaphoid fracture and a negative initial X-ray) revealed that 55% of patients who were placed in a cast for suspected occult scaphoid fractures failed to return for review as requested. They were either reviewed elsewhere in private clinics (locally and interstate) or not at all. Sixty-three percent of those that were reviewed had no scaphoid fracture and were immobilised for an average of 11.3 days. Thorpe et al reported 52% of patients failed to attend for final follow-up in their series.⁷ This highlights a weak link in the

management and follow-up of these patients and the need of early diagnosis at presentation .

Fractures of the scaphoid bone can be difficult to diagnose. There has been no previous study showing that early MRI within 24 hours of injury is either practical or is sufficiently sensitive and specific to become a gold standard test immediately after the injury.

Over the last few years there has been an increase in MRI scanning of suspected scaphoid fractures and has replaced bone scan as the gold standard in many institutions¹³.MRI has fewer false positive results and better inter and intra observer agreement than bone scan.³

A few previous studies have shown that early MRI at presentation within 24 hours of trauma was sensitive.⁵ This study demonstrates that such an approach is sensitive and specific and can easily be implemented in a major hospital with on site MRI facility. The sample size of this study is admittedly small (22 patients over 17 months) although not too dissimilar to other series such as Van Gelderen et al in New Zealand who recruited 16 patients over 21 months.⁹

Although a cost analysis of this approach has not been done in this study, its cost effectiveness has previously been shown to be not too different from the traditional approach to management.^{5,6} This of course does not take into account the loss of productivity of patients who are placed in a cast unnecessarily.

Conclusion

Our study demonstrates that MRI can be used for early assessment of occult scaphoid fractures within 24 hours of injury. In a tertiary level hospital containing a MRI facility operating at least 5 days a week, it is possible to accommodate an MRI of the wrist with a modified shortened protocol . This would lead to early initiation of treatment at the time of presentation and decrease the number lost to follow-up. The proposed clinical pathway would also reduce the over treatment of patients allowing scaphoid fracture to be ruled out at presentation—thus enabling early return to work instead of the usual 10 days in a plaster cast.

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Reliability of magnetic resonance imaging for traumatic injury of the knee

Keith Winters, Russell Tregonning

Abstract

Background: Magnetic resonance imaging (MRI) has become an important modality in the assessment of traumatic soft-tissue injury of the knee.

Methods: This prospective study was performed to evaluate the accuracy of MRI used at Wellington Hospital—by comparing the result of the scans of 67 patients with subsequent arthroscopy.

Results: The imaging studies provided a diagnostic accuracy of 90% for the medial meniscus, 82% for the lateral meniscus, 94% for the anterior cruciate ligament, and 96% for the posterior cruciate. The sensitivity was 87% for the medial meniscus, only 46% for the lateral meniscus, 92% for the anterior cruciate, and 80% for the posterior cruciate. The specificity was relatively high at 92%, 91%, 94%, and 97%, respectively.

Magnetic resonance imaging (MRI) has now been accepted as the best imaging for non-invasive evaluation of knee injuries. MRI can show osseous and soft-tissue structures without the use of ionising radiation, and has been reported to have a high diagnostic sensitivity, specificity, and accuracy. Arthroscopy, however, remains the 'gold standard' and acts as the reference in the validation of other diagnostic tools.

Other authors have compared the findings of MRI with those of diagnostic arthroscopy. In series ranging in size from 28 to 1400 knees, the accuracy of meniscal imaging ranged from 45 to 98%; and that of imaging the cruciate ligaments, from 90 to 100%.¹⁻⁶

The purpose of this study was to determine the accuracy of magnetic resonance imaging performed at Wellington Public Hospital in patients who have an injury to the soft-tissue of the knee—by comparing the findings of the imaging technique with those that were found with subsequent arthroscopy.

Methods

The series consisted of 67 patients who underwent an MRI of a traumatic knee and subsequent arthroscopy at Capital Coast Health between January 1999 and January 2003. The patients in this group often underwent the preoperative MRI because the diagnosis of meniscal or cruciate ligament trauma was not obvious on clinical evaluation. The study was approved by the local ethics committee. A computer check was performed by the Medical Records Department of all patients that underwent an MRI of the knee, and their clinical record was then examined to determine if they had undergone a subsequent arthroscopy. All patients in this group with traumatic soft-tissue injuries to the knee were included in the study.

The magnetic resonance imaging studies were completed between 2 weeks and 3 months before the operation. No patient had another injury to the knee between the time of the scan and the time of the operation. The scans could be performed by any one of three consultant radiologists who specialise in musculoskeletal imaging, and who had access to both clinical details and plain-films.

A Picker scanner with 1.5 Tesla magnet and Quadrature lower extremity coil was used. The knee was kept as close to the iso-centre as possible. Three routine sequences were performed, including Axial 2D FE with 5 mm thickness at 1 mm interval, Sagittal Dual Echo SE, and Coronal DE FSE with 3 mm thickness at 0.6 mm intervals using standardised parameters for T1-weighted images. T2-weighted images were made at the discretion of the radiologist. The total time for each scan ranged from 30 to 45 minutes.

A meniscal tear was identified on the magnetic resonance image by one of three criteria:

- A linear or complex intrameniscal signal extending to the superior or inferior surface of the meniscus,
- Gross disruption of the normal meniscal contour with obvious foreshortening, or
- Complete absence of any meniscal structure.

A cruciate ligament was considered to be intact if a homogenous low-intensity signal spanned the intercondylar notch from the origin to the insertion of the ligament.

A tear was diagnosed if:

- The normal signal of the ligament was discontinuous or absent,
- The ligament was poorly visualised because the intensity of the signal was variable or non-homogenous, or
- The ligament was identified but it did not proceed from or to its normal anatomical attachments.

Eight different orthopaedic consultants at either Wellington or Kenepuru Hospitals performed the arthroscopic procedures. The surgeons had full knowledge of the clinical findings and radiographic imaging. Arthroscopy was usually performed under general anaesthetic through up to three ports. A superolateral drainage port was established followed by an anterolateral portal for the arthroscope and an anteromedial portal for the insertion of the probe.

A routine sequence that included the evaluation of the patellofemoral joint, the medial and lateral compartments, and the intercondylar notch (to include the cruciate ligaments) was followed. Any pathology was usually photographically recorded. A meniscus was considered torn if any pattern of cleavage (complex, horizontal, flap, parrot-beak, vertical, or radial) produced a mechanical abnormality within the meniscus, which could be displaced by probing. A cruciate ligament was considered to be torn if it was completely disrupted in its substance or if laxity could be demonstrated with a probe or on anterior drawer.

We considered arthroscopy to be the standard for the accurate diagnosis of pathology in the menisci and cruciate ligaments, and used this as the basis for comparing the reliability of MRI. An image was considered a true positive when it indicated a torn structure, which was confirmed at arthroscopy. It was a true negative when it indicated no tear and this diagnosis was confirmed at arthroscopy. An image was a false positive when it indicated a torn structure and arthroscopy revealed no tear, and it was a false negative when it indicated no tear but arthroscopy revealed a torn structure.

Five parameters were calculated:

- **Accuracy**—the percentage of patients for whom the diagnosis based on magnetic resonance imaging was correct,
- **Sensitivity**—the percentage of patients in whom an arthroscopically confirmed tear had been preoperatively diagnosed on the basis of MRI,
- **Specificity**—the percentage of patients who had no tear at arthroscopy who had been found to have no tear on the basis of MRI,
- **Negative predictive value**—the percentage of patients that were diagnosed as having no tear on the basis of MRI and were subsequently found to have no tear at arthroscopy, and
- **Positive predictive value**—the percentage of patients that were diagnosed as having a tear on MRI and were subsequently seen to have a tear at arthroscopy.

Results

Thirty-seven patients were male and 30 were female. The ages ranged from 10 to 70 years (average 37 years). The average age of females in this group were over a decade higher at 45 years compared with males at 30 years. Forty percent of the patients were in their third and fourth decade; and of this group, only 33% were female.

The results of magnetic resonance imaging and those of arthroscopy are listed below (Table 1). There were 21 normal arthroscopies and no abnormalities were reported in 15 MRI scans. Arthroscopy revealed 31 tears to the medial meniscus, 13 tears of the lateral meniscus, 13 tears of the anterior cruciate ligaments, and 5 torn posterior cruciate ligaments.

Table 1. Findings of arthroscopy and magnetic resonance imaging (MRI)

Modality	Medial meniscus	Lateral meniscus	Anterior cruciate ligament	Posterior cruciate ligament
Arthroscopy	31	13	13	5
MRI	30	11	15	6

The comparison between arthroscopy and MRI are recorded in Table 2 and Table 3. Imaging of the medial meniscus yielded 27 true-positives, 33 true-negatives, 3 false-positives, and 4 false-negatives. Accuracy was 90%, sensitivity 87%, specificity 92%, negative predictive value 89%, and positive predictive value 90%, respectively.

Table 2. Reliability of magnetic resonance imaging as determined at arthroscopy

Result	Medial meniscus	Lateral meniscus	Anterior cruciate ligament	Posterior cruciate ligament
True positive	27	6	12	4
True negative	33	49	51	60
False positive	3	5	3	2
False negative	4	7	1	1

Table 3. Percentage validity of the diagnoses from magnetic resonance imaging

Validity	Medial meniscus	Lateral meniscus	Anterior cruciate ligament	Posterior cruciate ligament
Accuracy	92	82	94	96
Sensitivity	87	46	92	80
Specificity	92	91	94	97
Negative predictive value	89	88	98	98
Positive predictive value	90	55	80	67

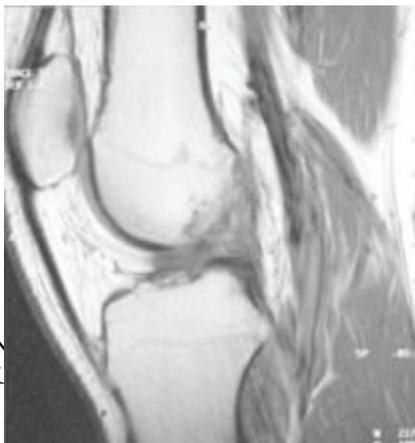
An example of an MRI showing a meniscal tear is seen in Figure 1. For the lateral meniscus, the imaging revealed 6 true -positives, 49 true-negatives, 5 false-positives, and 7 false-negatives. Accuracy was 82%, sensitivity 46%, specificity 91%, negative predictive value 88%, and positive predictive value 55%, respectively.

Figure 1. Sagittal T1-weighted imaging showing a tear of the posterior horn of the medial meniscus



For the anterior cruciate ligament, 12 true positives were found as well as 51 true negatives, 3 false positives, and 1 false negative. The accuracy was 94%, sensitivity 92%, specificity 94%, negative predictive value 98%, and positive predictive value 80%. A rupture of the anterior cruciate ligament is shown in Figure 2.

Figure 2. Sagittal T1-weighted imaging showing a rupture of the anterior cruciate ligament



Finally, for the posterior cruciate ligament, 4 true positives were found, 60 true negatives, 2 false positives, and 1 false negative. This equated to an accuracy of 96%, sensitivity of 80%, a specificity of 97%, a negative predictive value of 98%, and a positive predictive value of 67%.

Meniscal tears were grouped into three types at arthroscopy, which included flap, bucket-handle and radial tears. There were 25 flap tears, 4 bucket-handle, and 2 radial tears of the medial meniscus. Seventeen of these tears were in the posterior horn. The lateral meniscus was more evenly distributed with 5 flap tears and 4 in the other groups. Arthroscopy revealed 5 posterior horn tears of the lateral meniscus.

Discussion

Arthroscopy is accepted as the ‘gold standard’, and in experienced hands can reach a diagnostic accuracy between 69% and 98%,^{7,8} with correspondingly high levels of specificity and sensitivity in the detection of meniscal and cruciate ligament injury. However, arthroscopy is invasive and carries the risk of complications such as infection, pain, deep venous thrombosis, blood loss, and anaesthetic problems.⁹

The inherent advantage of the arthroscopic approach is that it can act as both a diagnostic and treatment modality. Our study has confirmed the ability of the MRI to reliably identify internal derangement of the knee. Its multiplanar imaging capabilities, outstanding resolution, absence of artefacts caused by superimposition of osseous structures, cost benefit, and non-invasiveness make MRI an important diagnostic modality.

Table 4. Sensitivity, specificity and accuracy (%) of the MRI for menisci and cruciates

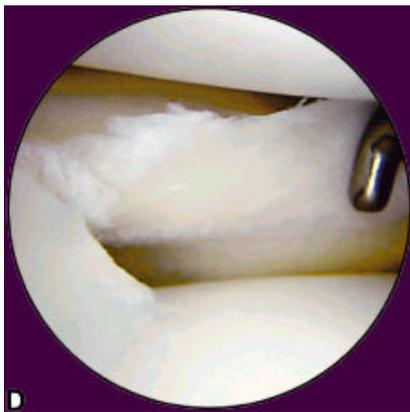
Reference ¹¹⁻¹⁷	Medial meniscus			Lateral meniscus			ACL/PCL		
Kinnunen et al 1994	88	80	82	25	97	88	83	85	85
Grevitt et al 1992	92	90	91	88	98	96	100	80	82
Kersting et al 1995	73	76	75	33	98	85	95	87	90
Glashow et al 1989	77	71	74	93	94	94	61	82	72
Raunest et al 1991	94	37	72	78	69	72			
Rappeport et al 1997	86	73	77	40	98	91	89	97	96
Polly et al 1988	96	100	98	67	95	90	100/100	97/100	97/100
Fischer et al 1991	93	84	89	69	94	88	93/93	93/99	93/100
Spiers et al 1993	100	71		99		92	100	98	

MRI=magnetic resonance imaging; ACL= anterior cruciate ligament; PCL=posterior cruciate ligament.

In our small study, the accuracy of the MRI was 92% for the medial meniscus, 82% for the lateral meniscus, 94% for the anterior cruciate ligament, and 96% for the posterior cruciate ligament. These findings were consistent with other studies in the literature, although the accuracy in the diagnosis of lateral meniscal injury is in the lower levels of previous reports. MRI very easily detected intact lateral menisci with a specificity of 91%. However only 6 of the 13 arthroscopically proven tears were detected at MRI. The lower sensitivity of detecting tears of the lateral meniscus by

MRI is well known, with sensitivity ranging from 25% to 99% (Table 4). Four of the seven false negatives were radial tears. We suspect that these tears are easily missed because they tend to lie in neither the sagittal nor coronal plane, which are best visualised on magnetic resonance imaging. An example of a radial tear missed on MRI is seen in Figure 3. The remaining tears were flap and bucket-handle type that we would have expected to be well visualised.

Figure 3. A radial tear of the lateral meniscus in a 19-year-old rugby player missed on magnetic resonance imaging



We also found that the MRI seemed to over-diagnose tears of the lateral meniscus resulting in a low positive predictive value. Of the five false-positives, four were diagnosed in the posterior horn of the meniscus. This phenomena also applied to the medial meniscus, with the MRI diagnosing posterior horn tears in two of the three cases not found to have tears on arthroscopy. Mink et al reported (in a series of five studies) a total of 47 false-positive results with MRI, 70% of which were in the posterior horn.¹⁰ This may occur because the lesion is missed at arthroscopy given the difficulty in visualising the posterior compartments. This calls into question the assumption that arthroscopy is the most reliable method for assessment of tears of the posterior horn of the menisci. Meniscal degeneration has also been suggested to explain over-diagnosis because of the increased signal intensity. Interestingly, in our study, five of the eight patients incorrectly diagnosed with meniscal tears on MRI were in their fourth and fifth decade. We were, however, strict on our selection criteria and only patients with a definite history of injury were included in the study. None of these patients were noted to have degenerative pathology of the menisci on arthroscopy.

MRI is also very good in detecting tears in the cruciate ligaments with sensitivity and specificity in the nineties, although the actual numbers are quite small in this study. Once again, the MRI showed a tendency to overdiagnose tears with five false-positives giving an overall positive predictive value of only 76%. This probably reflects the difficulty in distinguishing between complete and partial tears on MRI and the fact that arthroscopy is not the best tool for diagnosing cruciate ligament tears. Clinical evaluation and the detection of a positive anterior drawer remains the 'gold standard'.

One of the main concerns with this study inherent within the design is verification bias, where the result of the scan influences the decision to perform an arthroscopy—this tends to overestimate the ability of the MRI to detect lesions. This, however, is tempered by the clinician's approach that if the clinical signs are strong or if the symptoms fail to resolve, then an arthroscopy was performed despite the result of the scan.

The validity of MRI would have been better proven with a prospective study, with all patients having a preoperative scan, and a double-blind element introduced. We were able to demonstrate a high degree of accuracy in the diagnosis of medial meniscal and cruciate trauma. It is a very effective diagnostic tool in ruling out an injury when the clinical examination is equivocal. Furthermore, we identified 15 patients (22%) who could have avoided arthroscopy if the result of their MRI had been accepted.

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Elevated serum prostate-specific antigen levels and public health implications in three New Zealand ethnic groups: European, Maori, and Pacific Island men

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Abstract

Aims To predict differences in prostate cancer rates between New Zealand's major ethnic groups using community-based levels of elevated serum prostate-specific antigen (PSA).

Methods This study was undertaken in the Wellington region of New Zealand. 1425 subjects with no clinical history of prostate cancer had serum PSA levels measured—728 New Zealand European, 353 Maori, and 344 Pacific Island men. Age-standardised elevated PSA prevalences were calculated by standardising for population proportions. Prostate cancer prevalence ratios were predicted using a previously published method.

Results There was no significant difference between New Zealand's ethnic groups in the prevalence of elevated PSA ($p>0.05$). The overall age-standardised elevated PSA prevalence (3.9%) was lower than for all other community-based studies that were compared. Predicted cancer prevalence ratios were 1.1 across all New Zealand ethnic comparisons.

Conclusions The prevalence of elevated PSA in New Zealand men is lower than found in other community-based studies, and not significantly different between the three New Zealand ethnic groups. However, levels of elevated PSA may be useful for predicting prostate cancer incidence rates in ethnic groups. Available incidence data show New Zealand European men to have a higher prostate cancer incidence rate than both Maori and Pacific Islands men; however, this study found that prostate cancer incidence ratios between these groups are more likely to be closer to 1. Findings may indicate cultural barriers in the health system for Maori and Pacific Islands men; highlighting the need for clinicians to further consider cultural appropriateness in practice, and to target prostate health promotion for these groups.

There are notable ethnic and geographic variations in the incidence of prostate cancer.¹ The highest reported incidence is from Scandinavian countries, while there is an intermediate incidence in America and the United Kingdom, and the lowest incidence occurs in the Far East, especially Mainland China and Japan.^{2,3}

New Zealand's population consists of three major ethnic groups, New Zealand European, Maori, and Pacific Islands people and, for several years, New Zealand has ranked among the six highest incidence countries in the World.⁴ World Health Organization (WHO) age-standardised prostate cancer rates in 1998–1999, showed that incidence at 86.1 per 100,000, Maori males had the lowest incidence, followed by Pacific Islands males at 115.2 per 100,000, and 'other males' (chiefly New Zealand

European) had the highest incidence (118.9 per 100,000).⁵ Conversely, the prostate cancer WHO age-standardised mortality rates for Pacific Islands and Maori males in 1998–1999 (52.3 and 39.3 per 100,000 respectively) were higher than rates for ‘other males’ (22.8 per 100,000).⁵ However, there are reported inaccuracies in ethnic health data collection in New Zealand.⁶

In the United States, there is a 30% higher incidence and 120% higher prostate cancer mortality rate for African-Americans, compared to European Americans.⁷ Indeed, most clinical studies indicate that African-American men have a two-fold higher prevalence of metastatic disease at diagnosis. Differences in tumour stage have been attributed to differences in access to and utilisation of healthcare in African-American men.⁸ Similarly, higher prostate cancer incidence among Japanese-American men in comparison with native Japanese men is thought to be due to the more intensive prostate cancer screening within America.⁹ Healthcare access and utilisation is also an issue in New Zealand for Maori and Pacific Islands people^{10–13} and may be reflected in the difference between prostate cancer incidence and mortality statistics shown for these groups.

When used in conjunction with a digital rectal examination, elevated PSA levels (>4.0 ng/mL) aid in the detection of organ confined prostate cancer.¹⁴ Specifically, it has been found that a PSA value between 4.0–10.0 ng/mL carries a 22% probability of prostate cancer, while a PSA value of >10.0 ng/mL increases the cancer risk to more than 60%. PSA levels can also be raised in cases of benign prostatic hyperplasia and prostatitis.¹⁵

Because of the lack of specificity and sensitivity, PSA screening for asymptomatic men in New Zealand is not recommended by the National Health Committee.¹⁶ Regardless, the risk of prostate cancer parallels serum PSA levels and it has been suggested that elevated PSA could be used in the identification of high prostate cancer risk cohorts.¹⁷ A Japanese study (Shibata et al) compared the numbers of undetected prostate cancers between two populations, using the prevalence of elevated PSA.⁹ Shibata et al showed that even though prostate cancer incidence is currently four-fold to six-fold higher in Japanese-American men than native Japanese, it is likely to be lower (1.9 times) when undetected cancers are accounted for.⁹

Determining the prevalence of elevated PSA within New Zealand's major ethnic groups may provide some indication of the extent to which inadequate access to and under-utilisation of healthcare services influences New Zealand prostate cancer incidence statistics.

In the course of examining PSA levels in a non-random population with no clinical history of prostate cancer, we were able to evaluate the cross-sectional prevalence of elevated PSA at enrolment. Our study provides estimates of elevated PSA prevalence, prostate cancer incidence, and predicted cancer prevalence ratios among the three major New Zealand ethnic groups.

Methods

Subjects—Wellington Ethics Committee approval was gained. Written informed consent was obtained from each subject. Between January 2000 and February 2002 inclusive, a total of 1617 males were recruited into the Wellington Regional Community Prostate Study (Wellington Study), coordinated through the Wellington School of Medicine and Health Sciences of the University of Otago. To ensure an adequate representation of Maori and Pacific Islands men, subjects were non-randomly enrolled by

two separate means. Initially males aged between 40 and 69 years (within selected census area units containing at least 5% Maori and 5% Pacific Islands populations) were invited to attend a local clinic. A blood sample was taken and a detailed questionnaire was completed (Phase 1). The total number recruited into Phase 1 was 698.

The second mode of recruitment was through invitation to all eligible individuals who had been screened as part of the Wellington hepatitis and diabetes-screening programme for Maori and Pacific Islands populations. Upon recruitment, blood samples were retrieved from the Hepatitis Foundation of New Zealand and subjects completed the study questionnaire (Phase 2). The total number recruited into Phase 2 was 919.

Questionnaires utilised tick boxes and were self-administered. Ethnicity was determined on a self-identification basis (as in the 1986 New Zealand Census of Population and Dwellings ethnicity question; NZHIS 2001). Subjects were categorised as Maori or Pacific Islands if they indicated this affiliation as well as other ethnic affiliations. Each respondent was asked to grade the severity of urinary symptoms (using the International Prostate Symptom Score),¹⁸ and to declare if they have ever had any evidence of prostate disease. Subjects with total PSA levels of >4.0 ng/mL were referred back to their general practitioner (GP) for further evaluation for prostatic malignancy.

To ensure subjects were affiliated to New Zealand European or Maori or Pacific Islands groups and aged between 40-69, age and ethnicity selection criteria were applied to the combined Phase 1 and Phase 2 group (1617). Twenty-eight subjects were excluded on the basis of indicating Asian affiliation, and three subjects were excluded on the basis of prior prostate cancer. There were 1425 eligible subjects—728 New Zealand European, 353 Maori, and 344 Pacific Islands men.

A literature review undertaken during the planning of this study demonstrated that in population-based studies across all ages (40–90 years), 9%–13% of subjects had PSA levels elevated above 4.0 ng/mL.^{8,14,19,20} The power calculation determined a sample size of 343 per ethnic group, assuming elevated PSA levels would occur in 9%–13% of subjects and allowing an 80% chance of finding a significant difference at the $p < 0.05$ level in a t-test comparing geometric means between ethnic groups. Sera from each subject were tested for total PSA and free PSA. A 10mL tube of blood was collected, and centrifuged within 4 hours of sampling. Serum was stored at -70°C until assayed.

Laboratory blood analysis for PSA was carried out using an Elecsys 2010 assay (Roche Diagnostics, Mannheim, Germany). Assays were performed according to manufacturer's specifications.

Statistical analyses—Statistical analyses were performed with SPSS version 10.1 for the PC (SPSS Inc, Chicago, IL). Two separate sets of analyses were developed, first to investigate the overall and ethnic-specific prevalence of elevated PSA, as based on the currently used cut-off of over 4.0 ng/mL,²¹ and second to examine the use of elevated PSA as a means of estimating potential levels of undetected cancer and prostate cancer burden in New Zealand's ethnic groups.

Prevalence estimates for the larger Wellington target population were calculated by standardising by population proportions using the most recent Census (2001) data.²²

Chi-squared tests were performed to determine whether the prevalence of elevated PSA varied significantly by ethnicity. A logistic regression model was developed to test whether the prevalence of elevated PSA was associated with a number of measured demographic and clinical characteristics, including ethnicity and socioeconomic position (using NZDep96).²³ Comparison of age structure of elevated PSA by ethnic group was made using an interaction term (ethnicity * age) in the model. Tests of statistical significance were two-sided.

NZDep96 provides a deprivation score for each meshblock in New Zealand. Meshblocks are geographical units defined by Statistics New Zealand, containing a median of 90 people. The NZDep96 scale runs from 1 to 10 so that a value of 10 indicates that the meshblock is in the most deprived 10% of areas in New Zealand.²³

1998-1999 prostate cancer registration data from the New Zealand Health Information Service were utilised. Comparisons of levels of elevated PSA and prostate cancer incidence, as well as predictions of prostate cancer detection rates, were undertaken by graphically modelling crude age-specific prostate cancer incidence rates and prevalences of elevated PSA by ethnic group in New Zealand and other selected countries. The proportion of prostate cancers in relation to the prevalence of elevated PSA was calculated.

Shibata et al's methodology provided an estimate of cancer burden based on elevated PSA and was used as a guide for the analysis of cancer prevalence ratios between New Zealand's three main ethnic

groups. We initially followed Shibata et al's method, which used age-specific reference ranges, based on the upper PSA cut-off level for each age 10-year group in their study population, to determine the age-specific prevalence of elevated PSA (P) in two populations.⁹ After this point, we modified Shibata et al's original methodology (see Appendix 1).

The utilisation of total PSA as a predictor of malignancy may be enhanced by the use of age-specific reference ranges, which increase the sensitivity of PSA screening in younger males and the specificity in older males.²⁴ The age-specific reference range used to determine the prevalence of elevated PSA in the Wellington Study group was calculated using a best-fit regression of the 95th percentile cut-offs, devised on the subjects showing no evidence of clinical prostate cancer.²⁵

We estimated the prevalence of undetected cancer in all three New Zealand ethnic groups with elevated PSA, by using the age-specific positive predictive value (PPV) based on the age-specific reference range for elevated PSA from a large population of American European volunteers.¹⁴

Estimates of undetected cancer for each ethnic and 10-year age group were calculated by the following formula:

The estimated proportion of undetected cancers = the expected PPV x (number with elevated PSA/total number of subjects) x 100.

To determine the crude prostate cancer incidence rates that coincided most closely with the Wellington Study's timeframe, ethnic and age-specific incidence rates were calculated for men aged between 40–69 years, for the years 1998–1999. To avoid the possible inclusion of study participants who were found to have prostate cancer, prostate cancer registration data after this date were not included.

Estimates of the prevalence of undetected cancer were combined with the incidence of detected cancer to find the cumulative risk of having an undetected cancer. These risk estimates provided estimates of the total cancer burden.⁹

The fraction of undiagnosed cancers was estimated by the formula:⁹

% undiagnosed cancer = 100 - (%detected cancers / %cumulative risk of undetected cancer) x 100.

Predicted cancer prevalence ratios were calculated using the % cumulative risks.⁹ For example;

Predicted cancer prevalence ratio for New Zealand European compared with Maori =
%cumulative risk New Zealand European / %cumulative risk Maori.

Cancer incidence ratios were calculated on 1998-99 prostate cancer registration statistics. For example;

Cancer incidence ratio for New Zealand European compared with Maori = New Zealand
European age-specific cancer incidence rate/Maori age-specific cancer incidence rate.

The predicted cancer prevalence ratios were compared with cancer incidence ratios.⁹

The PPV is likely to differ by ethnicity, as the risk of getting cancer at elevated PSA levels is different between ethnic groups.^{8,21} We explored the effect of an ethnic difference in PPV on results by recalculating predicted cancer prevalence ratios. For example, we applied 18.2% PPV for Maori men (as for Japanese men; to illustrate an extreme low PPV)²⁶ and had New Zealand European's PPV remain at 31.8%.

Results

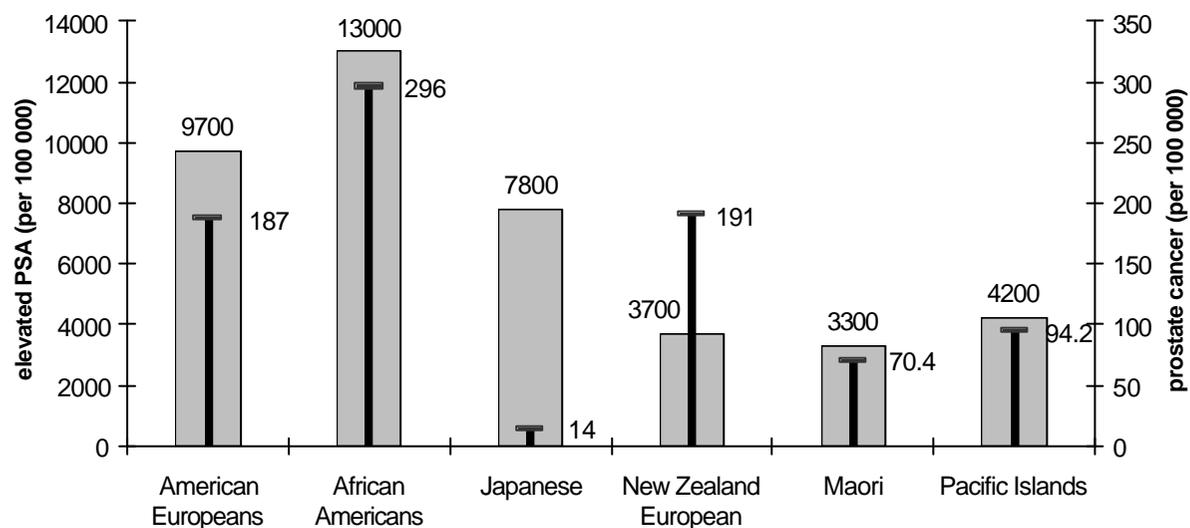
Table 1 gives the New Zealand prevalence of elevated PSA compared to other ethnic groups. It shows that the age-standardised elevated PSA prevalences for New Zealand European, Maori and Pacific Islands men (3.7, 3.3, and 4.2% respectively) are lower than all other community-based studies show.^{8,14,26–29} The chi-squared tests confirmed that the difference in the prevalence of elevated PSA between the New Zealand ethnic groups and the other groups was statistically significant ($p < 0.05$).

Table 1. Prevalence of elevated PSA (>4.0 ng/mL) in the Wellington Study Group by ethnicity and age, compared with ethnic groups from other community-based studies.

Study	Wellington Study			Imai <i>et al.</i> ²⁶	Bunker <i>et al.</i> ²⁷	Crawford <i>et al.</i> ¹⁴	Tay <i>et al.</i> ²⁸	Smith <i>et al.</i> ⁸	Horninger <i>et al.</i> ²⁹
	<i>NZ Euro.</i> %	<i>Maori</i> %	<i>Pacific Islands</i> %	<i>Japanese</i> %	<i>Afro-Caribbean</i> %	<i>American European</i> %	<i>Singaporean Asian</i> %	<i>African-American</i> %	<i>Austrian European</i> %
<i>n</i>	728 ^{*a}	353 ^{*a}	344 ^{*a}	2502	2156	31 953 ^{*a}	799 ^{*a}	949 ^{*a}	21078
Overall	3.7 ^{*b}	3.3 ^{*b}	4.2 ^{*b}	7.8 ^{*c}	31	9.7	13.1	13	8
40-49 years	0.4	0.6	1.3	0	10	N/A	N/A	N/A	N/A
50-59 years	3.5	3.2	3.7	5.0	28	N/A	N/A	N/A	N/A
60-69 years	11.0	13.2	12.7	8.8	45	N/A	N/A	N/A	N/A

^{*a} Study populations were non-randomly selected. ^{*b} These are age-standardised estimates for the Wellington target population. ^{*c} These elevated PSA levels are > 3.7ng/mL. ^{*d} The positive predictive values (PPVs) are calculated by the formula (number of prostate cancers with a positive test)/(number of subjects with a PSA > 4.0ng/mL).

Figure 1. Prostate cancer incidence rates compared to the prevalence of elevated PSA (per 100 000) by ethnic and 40–69 year age group, as determined by various studies.



Bars indicate age-specific prevalence of elevated PSA. Drop lines indicate age-specific prostate cancer incidence rates. To relate incidence data with corresponding available elevated PSA data; the incidence rate for African-Americans was calculated from 1991-1996 data,^{1,8} American Europeans from 1993-1994 data,^{1,14} and Japanese from 1988-1992.^{9,26} New Zealand incidence data is based on men aged 40-69 from the years 1998-1999. To avoid subjects being counted twice, New Zealand incidence data did not correspond with the study period 2000-2002.⁵

Table 2. Percentage of men in the Wellington Study with a PSA concentration value above the age-specific reference range by age group

	New Zealand European %	Maori %	Pacific Islands %
	<i>Age specific range ^{*a}</i>	<i>Age specific range ^{*b}</i>	<i>Age specific range ^{*c}</i>
Overall	7.0	6.5	7.0
40-49 years	4.3	6.8	4.6
50-59 years	6.9	5.6	7.4
60-69 years	11.7	7.5	12.7

^{*a} The recommended age-specific reference ranges for New Zealand European men are as follows: 40-49 years, 0.0 to 2.1ng/mL, 50-59 years, 0.0 to 2.8ng/mL, 60-69 years, 0.0 to 3.8ng/mL.²⁵; ^{*b} The recommended age-specific reference ranges for Maori men are as follows: 40-49 years, 0.0 to 1.5ng/mL, 50-59 years, 0.0 to 2.8ng/mL, 60-69 years, 0.0 to 5.0ng/mL.²⁵; ^{*c} The recommended age-specific reference ranges for Pacific Islands men are as follows: 40-49 years, 0.0 to 1.9ng/mL, 50-59 years, 0.0 to 2.7ng/mL, 60-69 years, 0.0 to 3.8ng/mL.²⁵

Table 3. Estimated prevalence of undetected prostate cancer by age and ethnic group

	New Zealand European				Maori				Pacific Islands			
	% <i>elev. PSA</i> ^{*a}	% <i>undetected. Ca</i> ^{*b}	% <i>detect. Ca</i> ^{*c}	% <i>cumulat. risk</i> ^{*d}	% <i>elev. PSA</i> ^{*a}	% <i>undetected. Ca</i> ^{*b}	% <i>detect. Ca</i> ^{*c}	% <i>cumulat. risk</i> ^{*d}	% <i>elevat. PSA</i> ^{*a}	% <i>undetected. Ca</i> ^{*b}	% <i>detect. Ca</i> ^{*c}	% <i>cumulat. risk</i> ^{*d}
Overall	7.0 (51/728)	2.2 (16.2/728)	0.2 (191 ^{*5})	2.4	6.5 (23/353)	2.1 (7.3/353)	0.1 (70.4 ^{*5})	2.2	7.0 (24/344)	2.2 (7.6/344)	0.1 (94.2 ^{*5})	2.3
40-49 years	4.3 (12/277)	0.9 (2.4/277)	0.0 (11.2)	0.9	6.8 (12/176)	1.4 (2.4/176)	0.0 (3.8)	1.4	4.6 (7/153)	0.9 (1.4/153)	0.0 (0.0)	0.9
50-59 years	6.9 (20/288)	1.8 (5.3/288)	0.1 (136.2)	1.9	5.6 (7/124)	1.5 (1.9/124)	0.1 (55.1)	1.6	7.4 (10/136)	2.0 (2.7/136)	0.1 (69.6)	2.1
60-69 years	11.7 (19/163)	3.6 (5.9/163)	0.6 (582.2)	4.2	7.5 (4/53)	2.5 (1.3/53)	0.3 (292.3)	2.8	12.7 (7/55)	4.0 (2.2/55)	0.4 (443.8)	4.4

Expected PPVs are based on age-specific reference ranges were as follows: Overall, 31.8%, 40-49 years, 20.0%, 50-59 years, 26.7%, 60-69 years, 31.3%.¹⁴ ^{*a} % of elevated PSA was determined on the age-specific reference ranges in Table 2. ^{*b} Estimated proportion of undetected cancers = the expected PPV x (number with elevated PSA/number of subjects) x 100. ^{*c} Percentage of detected cancers are based on the 1998-99 cancer registration statistics.⁵ ^{*d} Cumulative risk of prostate cancer = estimated % of undetected cancers + % incidence of detected cancers. ^{*e} Calculated age-specific incidence rates are per 100 000. The 'Overall' age-specific rate is calculated for the age group 40-69, other rates are calculated separately for each 10 -year group. All figures have been rounded to one decimal place.

Table 4. Cancer incidence ratios and predicted cancer prevalence ratios between New Zealand European, Maori, and Pacific Island men.

	New Zealand European compared with Maori		New Zealand European compared with Pacific Islands		Pacific Islands compared with Maori	
	<i>actual</i> ^{*a}	<i>predicted</i> ^{*b}	<i>actual</i>	<i>predicted</i>	<i>actual</i>	<i>predicted</i>
Overall	2.7	1.1	2.0	1.1	1.3	1.1
40-49 years	3.0	0.6	N/A	1.0	N/A	0.6
50-59 years	2.5	1.2	2.0	0.9	1.3	1.3
60-69 years	2.0	1.5	1.3	1.0	1.5	1.6

^{*a} Cancer incidence ratios were calculated on 1998-99 cancer registration statistics. For example: cancer incidence ratio for New Zealand European compared with Maori = New Zealand European age-specific cancer incidence/Maori age-specific cancer incidence; ^{*b} Predicted cancer prevalence ratios were calculated using the % cumulative risks (Table 3).

Figure 1 is a comparison of crude age-specific prostate cancer incidence rates and the prevalence of raised PSA by ethnic group in New Zealand and other selected countries. Although the prevalence of elevated PSA is substantially higher than the prostate cancer incidence rates, ethnic differences in levels of elevated PSA generally reflect the trends in difference in incidence between ethnic groups.

New Zealand European men (at 5.2%) have a higher proportion of prostate cancers in relation to elevated PSA levels than all other groups, followed by African-Americans (2.3%), Pacific Islands (2.2%), Maori (2.1%), and American Europeans (1.9%). Japanese men appear to have lower incidence rates in comparison to elevated PSA levels, than shown for all other ethnic groups (0.2%).

Table 2 gives the percentage of men in the study population with a PSA value above the current reference range (0.0–4.0 ng/mL), compared to the age-specific reference ranges (as used for calculating the prevalence of undetected cancer), by age group.

The prevalence of elevated PSA in each age and ethnic group increased using age-specific ranges, with the exception of Maori men aged 60–69 years, for whom the prevalence decreased (13.2% using 0.0–4.0 ng/mL; compared to 7.5% using age-specific reference ranges).

Only age was significantly related to the chances of having an elevated PSA level as defined by either the current reference range (0.0–4.0 ng/mL) or the age-specific range ($p < 0.001$). The chi-squared test and logistic regression model demonstrated no significant difference between the New Zealand ethnic groups in the prevalence of elevated PSA and no differences in the chances of getting a change in elevated PSA age pattern between the ethnic groups. NZDep96 was not associated with the prevalence of elevated PSA. Potential confounding factors were adjusted for within the multivariate models.

Table 3 gives the estimated prevalence of undetected prostate cancer by age and ethnic group. Data show that 2.2% of Pacific Islands and New Zealand European men aged 40–69 have undetected prostate cancer and 2.1% of Maori men have undetected prostate cancer. While, overall, New Zealand European men have the highest cumulative risk of prostate cancer (2.4%), Maori men have the highest cumulative risk in the 40–49 year age group (1.4%) and Pacific Islands men have the highest cumulative risk in the 50–59 and 60–69 year age groups (2.1 and 4.4% respectively).

Calculations suggest that Pacific Islands men have the highest fraction of prostate cancer (95.6%) remaining undiagnosed of all ethnic groups. With Maori intermediate at 95.5% undiagnosed and New Zealand Europeans the lowest fraction undiagnosed (91.7%).

Table 4 gives the cancer incidence ratios and predicted cancer prevalence ratios between New Zealand European, Maori and Pacific Islands men. Prostate cancer registration data from 1998 to 1999 shows the cancer incidence ratios between New Zealand Europeans compared with Maori is 2.7, New Zealand European compared with Pacific Islands men is 2.0, and Pacific Islands compared with Maori men is 1.3. However, the predicted cancer prevalence ratios are 1.1 across all ethnic comparisons.

Conclusions

We compared ethnic groups in Wellington (New Zealand) and Japan, Austria, United States, Singapore, and the Caribbean region using >4.0 ng/mL as a cut-off for determining levels of elevated PSA. Although there was no significant difference in the prevalence of elevated PSA between the New Zealand ethnic groups, there was a difference between the New Zealand groups and the prevalences shown for ethnic groups in other community-based studies.

The overall New Zealand age-standardised elevated PSA prevalence (3.9%) was closest to the prevalence found for Japanese men (7.8%)²⁶ and lower than the prevalence of any other ethnic group shown (Table 1). For example, Austrian European (8%),²⁹ American European (9.7%),¹⁴ African-American (13%),⁸ Singaporean Asian (13.1%),²⁸ and Afro-Caribbean (31%).²⁷

Available New Zealand prostate cancer crude incidence rates showed that, among different ethnic groups, New Zealand Europeans had the highest rate, followed by Pacific Islands men and lowest were Maori men (191, 94.2, and 70.4 per 100,000 respectively). In general, New Zealand men were most similar to American European men in age-specific prostate cancer crude incidence rates.

The prevalence of elevated PSA reflects ethnic differences in prostate cancer incidence (Figure 1). Results highlight that levels of elevated PSA can parallel prostate cancer risk¹⁷—and thus may be useful in estimating the incidence of undetected cancers in a population. For example, data suggests that African-Americans have both the highest prostate cancer incidence and prevalence of elevated PSA; equally, Japanese men have both the lowest prostate cancer incidence and prevalence of elevated PSA.

Determining elevated PSA using age-specific reference ranges generally increased the elevated PSA prevalence estimates, indicating a possible degree of under-diagnosis in all New Zealand groups through PSA based screening using the standard cut-off (>4.0 ng/mL) (Table 2). The use of an age-specific PSA reference range has been found to produce more accurate results.¹⁹

Predicted cancer risks for New Zealand ethnic groups were age-dependant. As younger men (under 50 years old) who develop this disease are less likely to survive it,² age-specific prostate cancer risk could be important. Maori men were shown to have the highest cumulative risk when younger (40–49 years), whereas Pacific Islands men had higher risks when aged between 50–69 (Table 3).

Results suggest that New Zealand Europeans had the highest rate of prostate cancer detection of any other ethnic group compared and that estimates of undiagnosed cancer were highest for Pacific Islands men. Data also show that at over two-fold the incidence, prostate cancer incidence ratios based on available New Zealand prostate cancer registration data likely overestimate differences in incidence rates between New Zealand European, and Maori and Pacific Islands men. The incidence ratios are more likely to be closer to 1. Under-utilisation of health services by Maori and Pacific Islands men and under-reporting of ethnicity in health data, would lead to fewer prostate cancer diagnoses, and thus, lower incidence statistics shown for these groups.

Statistics show that although more New Zealand European men are diagnosed with prostate cancer, more Pacific Islands and Maori men die from this disease. This

supports the possibility that Pacific Islands and Maori men receive prostate health care later than other ethnic groups, as has been found for African Americans.⁸ Cultural barriers to healthcare access are likely to be an important factor determining prostate healthcare utilisation rates amongst Maori and Pacific Islands people.^{10–13,30}

There are several caveats to consider with our study. Elevations of serum PSA levels beyond that accounted for by prostatic size alone, may reflect levels of non-detectable prostate cancer.²¹ As there was no difference in the prevalence of significant lower urinary tract symptoms between the three Wellington Study's ethnic groups, we believe that levels of elevated PSA in subjects may reflect ethnic-specific levels of non-detectable prostate cancer.

Estimates of undetected cancer prevalence only include cancers that cause an elevated PSA level, whereas the detected cancer prevalence rates include cancers that are still too small to do so. Therefore, there may be some underestimation in undetected prostate cancer incidence using elevated PSA alone.⁹

The potential for self-selection bias in our non-random sample was quantitatively evaluated using a variety of methods, including: the exclusion of groups with possible biases such as urinary symptoms; comparison of urinary symptoms with a randomised New Zealand study;³⁰ standardisation for age, smoking and NZDep96 and sensitivity analyses. All investigations indicated that self-selection bias was unlikely to explain levels of elevated PSA in our Wellington Study.

In conclusion, the prevalence of elevated PSA in New Zealand men was lower than found in other community-based studies and not significantly different between the three New Zealand ethnic groups. However, levels of elevated PSA may be useful for predicting prostate cancer incidence rates in ethnic groups.

Available incidence data show New Zealand European men to have a higher prostate cancer incidence rate than both Maori and Pacific Islands men; however, this study found that prostate cancer incidence ratios between these groups are more likely to be closer to 1. Therefore, prostate cancer incidence appears to be at least as high in Maori and Pacific Islands men as New Zealand European men, and prostate cancer related mortality greater.

It is likely that under-utilisation of health services by Maori and Pacific Islands men is reflected in the lower prostate cancer incidence shown for these groups. Findings may indicate cultural barriers in the health system for Maori and Pacific Islands men; highlighting the need for clinicians to further consider cultural appropriateness in practice and target prostate health promotion for these groups.

Appendix 1: Discussion of methodology—Shibata et al estimated the prevalence of undetected cancers in the participants with elevated PSA, using a formula that determines that the excess prevalence minus the false positive rate (P=0.05) is approximately the proportion of men with undetected cancer. However, this method assumes that the levels of false positives will be consistent across the age groups.⁹ Such consistency across age groups has been shown to be untrue.¹⁴ As a significant parameter in determining the value of cancer detection tests is the PPV,¹⁴ we used the PPV for the purposes of our research.

The PPV is the fraction of patients who have a cancer when a method of detection, such as PSA screening, shows a positive result.¹⁴ Because not all study subjects were referred for further prostate cancer diagnoses and data were not available for all those who attended their GP, we could not calculate PPVs for our Wellington Study Group.

Therefore, to estimate the numbers of undetected cancers in our Wellington Study, New Zealand incidence data were compared with overseas incidence data to establish the most appropriate PPV. We used a PPV based on the age-specific reference range for elevated PSA from a large population of American European volunteers, because their prostate cancer incidence rates were the closest to all three ethnic groups in our study.¹⁴ Further research into actual PPVs for New Zealand populations could make such a method more useful.

Because of the need to assume a PPV for ethnic groups within the Wellington Study there are important limitations to this method of estimating cancer incidence. If Maori men's PPV were 18.2% and New Zealand Europeans remained at 31.8%, the estimated number of undetected cancers overall in Maori men would decrease (from 2.1% to 1.2%), as would the cumulative risk of prostate cancer (from 2.2% to 1.3%). This, in turn would cause an increase in estimated cancer prevalence ratio of New Zealand European men compared to Maori men (from 1.1 to 1.9). As the resulting estimated cancer prevalence (1.9) is still lower than that shown by available incidence statistics (2.7), we feel that the potential for ethnic differences in PPV do not fully explain our results.

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A hen's tooth in the prostate

Jonathan Burge, Niall Corcoran, Anthony Costello

This case report demonstrates an interesting and rare form of prostate cancer. It outlines the presenting symptoms and the subsequent management of such a rare and malignant prostatic cancer. Of note is the prostate-specific antigen (PSA) within normal range at the time of presentation. The cancer was advanced at first presentation to our Service.

Case report

A previously healthy 56-year-old white male presented with a 3-week history of lower abdominal pain. Physical examination revealed a large mass in what was thought to be the anterior rectal region. He had no lower urinary tract symptoms. PSA=1.8 µg/L.

Colonoscopy demonstrated indentation in the middle third of the rectum anteriorly with an intact overlying mucosa. Abdominal and pelvic computed tomography (CT) scan demonstrated a large soft tissue mass in the pelvic cavity with pararectal lymphadenopathy. Transrectal ultrasound guided biopsy revealed malignant spindle cells in all cores with foci of adenocarcinoma, designated Gleason 3+3.

Based on the histology from the transrectal biopsy findings and the radiological diagnosis from CT, a decision was made to perform a radical prostatectomy. Intraoperative findings revealed a large mass arising from the prostate involving bladder base and rectum.

A radical cystoprostatectomy was performed with en-bloc resection of the rectum and sigmoid colon. Excision was thought to be complete at the time of operation. Histology revealed extensive infiltration and replacement of the prostate by a malignant spindle cell neoplasm with a multinodular pattern of growth, extracapsular extension, prominent lymphovascular invasion, and a mitotic rate of 50 per mm² (Figure 1).

There was extracapsular invasion into bladder base and rectum. The malignant spindle cells were relatively uniform and arranged in short fascicles. There was patchy staining for desmin, but stains for CD34, actin and S100 were negative. Distributed throughout the spindle cell malignancy were numerous small foci of prostate, which stained positively for cytokeratins and PSA. This adenosarcomatous component was assigned a Gleason score of 3+3.



Figure 1. Haematoxylin-and-eosin stained slide displaying sarcomatous component of excised tumour



Discussion

Carcinosarcoma of the prostate is a rare tumour. It is characterised histologically by a mix of malignant epithelial and mesenchymal elements, with the sarcomatous component typed by immunohistochemical staining.¹

In the literature, there are 42 reported cases of carcinosarcoma with only a small percentage having a highly undifferentiated sarcomatous component as seen here. Similar to our patient, the tumour is often advanced at presentation—and despite multimodal therapy, the prognosis is poor.² The most significant factor determining prognosis is completeness of surgical excision. Our patient had advanced disease at the time of surgery. He returned less than 6 months later with multiple metastases throughout his abdomen and lungs.

The origin of carcinosarcoma is unknown. Several possibilities have been proposed including development of the carcinomatous and sarcomatous areas from different areas within the prostate, differentiation from a primitive cell line, or transformation of the carcinomatous component into a sarcomatous cell line.^{3,4} Most prostatic

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tumours of the elderly are adenocarcinomas with sarcomas accounting for less than 1% of total prostate tumours. Primary carcinosarcomas of the prostate are rare. They readily metastasise as in the case of our patient who returned within 2 months from the time of his primary surgery with multiple metastases to his peritoneum and lung. Other sites for metastasis are lung, spine, brain, and spleen.⁴

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Beware: compartment syndrome of the hand

Warren Leigh, Vasu Pai

Compartment syndrome of the forearm or leg, is a well-known postoperative complication. However, compartment syndrome of the hand is not common and is not easy to diagnose. Various aetiologies been reported¹⁻³—and when diagnosis is missed, it results in ischaemic contractures and permanent disability.

The occurrence of compartment syndrome in the hand after radial osteotomy (or any other elective osteotomy of the forearm bones requiring fasciotomy) has not been described. This article presents a case of interosseous muscle compartment syndrome as a complication of this procedure and highlights difficulty in diagnosing such a problem.

Case report

A 30-year-old gentleman presented with a malunited fracture of his left distal radius. He underwent corrective osteotomy of the distal radius, bone grafting, and Kirschner wire fixation. Two days after surgery he was complaining of worsening pain in his hand. As he could not fully straighten his fingers, his cast was changed and he was discharged from the hospital on oral analgesics.

The next day he presented to the emergency department of a different hospital with worsening pain in his left hand. On examination, there was gross swelling of the hand. Blisters were present on both the radial and ulnar borders at the level of the wrist. The wire sites were clean and there was no sign of infection at the operation site. He could not actively straighten without worsening of pain. The pain was most severe on trying to make a fist. Finger movements were limited with only 15° to 30° degrees at metacarpophalangeal and interphalangeal joints. Passive flexing his interphalangeal joints with metacarpophalangeal joint in extension caused severe pain. Forearm muscles were soft and non-tender. Sensation was intact in median, ulnar, and radial nerve distributions; and capillary return as well as radial and ulnar arterial pulses were normal.

Compartment syndrome of the hand was diagnosed, and the patient underwent fasciotomy. Through double dorsal incisions along second and fourth metacarpals, all four dorsal interosseous spaces (as well as three palmar spaces) were released. This was followed by a carpal tunnel, and thenar compartment release.

Upon fascial release, the interosseous muscles were found to be swollen, but there was no evidence of necrosis. A delayed closure of wounds was carried out. Postoperatively the patient did well. There was dramatic improvement in his symptoms. At last follow-up, patient regained full motor control and there was no deformity.

Discussion

There are 10 compartments in the hand: the thenar, hypothenar, adductor, 3 palmar, and the 4 dorsal interosseous compartments.⁸ It has been suggested that the muscles

situated on the radial side of the hand are more prone to compartment syndrome than those on the ulnar side (as they are supplied by more end-arteries).¹ Decompression should include fasciotomy of all compartments of the hand.

Compartment syndrome is caused by an increase in pressure within a closed compartment that increases to a level causing vascular perfusion to be compromised. This can be secondary to swelling or external compression.^{3,4} When this ischaemia is unrecognised, it can greatly impair normal hand function.

A variety of causes of hand compartment syndrome have been reported such as fractures, suction injuries, crush injuries, metacarpal fractures, arterial injuries, and injection of intravenous fluids and contrast material.²⁻⁸ Ouellette and Kelly² reviewed 19 patients with compartment syndrome of the hand and had had only 1 related to surgery following an arthrodesis of the wrist.

Figure 1. Test for compartment syndrome of the forearm: extension of finger from flexed position



Our case illustrates several important points. Compartment syndrome in the postoperative patient should always be in the differential diagnosis. Presence of hand sensation, a normal capillary return and ability to straighten fingers (diagnostic of compartment syndrome of the forearm; Figure 1), may all be normal in compartment syndrome of the hand. It is a diagnosis that requires a high index of clinical suspicion. The main symptoms are a tense swollen hand with severe pain that is out of proportion to the clinical situation.

The definitive test⁶ for compartment syndrome of the hand is a positive stretch test for intrinsic muscles of the hand. This is carried out by flexing the interphalangeal joints of the fingers while the metacarpal joints are held in neutral (Figure 2).

Figure 2. Test for compartment syndrome of the hand: flexion of interphalangeal joint with metacarpophalangeal joint in extension



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Prostate cancer screening: is it possible to explain diametrically opposed views?

Ann Richardson

Prostate cancer screening is controversial.¹⁻³ Clearly, the views of those who support and actively offer prostate cancer screening are very different from the views of those who see no justification for screening at present. How can we understand and explain such opposing views?

It is likely that differences of opinion about prostate cancer screening reflect differences in the way people assess the benefits and risks of screening. By examining each of these in turn, it is possible to understand the controversy, but also determine whether prostate screening is ethical.

Benefits

Underlying the discrepant views on screening is the extent to which people assume that screening is beneficial. If men and their doctors assume that screening *must* be beneficial, then irrespective of the outcome of prostate-specific antigen (PSA) testing, both the man and his doctor will be positively reinforced:

A physician is positively reinforced for recommending screening, regardless of the test result, because a negative result makes the patient grateful for reassurance and a positive result makes a patient grateful for early detection. A patient who is impotent and incontinent after a decision for curative treatment may attribute his survival to surgery and be grateful for having his cancer cured. Individual experience provides almost no negative feedback that early detection and aggressive treatment may not work.⁴

But is prostate cancer screening beneficial? Intuitively it seems obvious; surely picking up disease earlier must be good? Unfortunately we know that screening is *not* always beneficial. Randomised controlled trials (RCTs) have revealed that some screening procedures (for instance, screening for lung cancer using chest radiography and sputum cytology in high-risk individuals, and screening for breast cancer with breast self-examination), which were previously assumed to be beneficial, are not.⁵⁻⁷

Using methods such as survival comparisons, observational studies, or ecological studies to assess screening is dangerous. These studies are vulnerable to biases, which can cause any benefit of screening to be overestimated, and at worst, can make screening appear beneficial even when it is not (Table 1). Only an appropriately designed and analysed RCT can avoid these biases, and determine whether screening for prostate cancer really is beneficial.

At present, we do not know if there is any benefit from prostate cancer screening, because there is not yet evidence from appropriately designed and analysed randomised controlled trials.⁸⁻¹¹ Two large RCTs are presently underway.¹²

Table 1. Why do we need randomised controlled trials of prostate cancer screening?

Bias is a defect in study design or analysis that causes results to deviate from the truth. The following biases can affect the assessment of prostate cancer screening:

Lead time bias

Screening can detect prostate cancer early, thereby extending the interval between diagnosis and death, *even if the time of death is unchanged*. Because survival time is measured from time of diagnosis to death, men whose prostate cancer was detected by screening will have longer survival times than men diagnosed clinically, even if screening does not actually extend life.

Length bias

Fast growing prostate tumours, which tend to have the worst prognosis, are less likely to be detected by screening, because they grow so rapidly that the period when the tumour could be detected by screening before signs or symptoms develop, may be very short. Screening will therefore detect a disproportionate number of slow growing tumours with a good prognosis. Comparisons of outcome between men with screen-detected prostate cancer and men with clinically-diagnosed prostate cancer are likely to be invalid because of this.

Selection bias

Men who take up the offer of screening may differ in their underlying risk of dying from prostate cancer, so that their prognosis would have differed from non-participants even in the absence of screening. Thus, comparing the outcome for men who have been screened with men who have not may be inappropriate. Selection bias can be avoided in a randomised-controlled trial by appropriate (intention to treat) analysis.

Overdiagnosis bias

Screening may detect abnormalities that are of questionable malignancy and cancers that would not have been diagnosed in the absence of screening. Although some of these abnormalities and cancers may never have been diagnosed, nor affected a man's life in the absence of screening, they will be counted as 'screen-detected' cancers. Because of the inclusion of these cancers, the outcome for a group of screened men with prostate cancer will appear better than the outcome for unscreened men with prostate cancer.

Risks

People who are unwell are often prepared to take risks in order to get better. Many patients will accept treatments that carry risks; for instance, drugs that have potential side effects, and radiotherapy or surgery despite their possible complications. Clinicians are used to interacting with people who are unwell and who tolerate potential harm in order to get better. Clinicians may transfer this tolerance of harm to the screening situation, hence urologists may be more likely to support prostate cancer screening than public health physicians.

But people who are *well* may not share the same tolerance towards potential harm, especially if the benefit is uncertain. If there is *no* benefit, the net effect of screening will be to harm those who take part. Apart from the obvious harms related to false negative and false positive screening results, overdiagnosis is likely to be a major problem in prostate cancer screening.

Autopsy studies, where prostate biopsies were taken from men who had died of causes other than prostate cancer,^{13,14} have shown that the histological evidence of prostate cancer in such men is much higher than the lifetime incidence and mortality from prostate cancer. In other words, histological evidence of prostate cancer can be found in far more men than would ever be expected to suffer from or die from the disease.

Further evidence of overdiagnosis comes from the Finasteride Trial,¹⁵ which was designed to find out whether the drug Finasteride could prevent prostate cancer. Men aged 55 years and over were randomly allocated to an intervention group (which took Finasteride) and a control group (which took a placebo). All the men had normal digital rectal examinations (DRE) and PSA results at entry to the trial. During the trial, the men underwent annual DRE and PSA tests.

At the end of the trial, all men who had not been diagnosed with prostate cancer during the trial were offered an end-of-study prostate biopsy. Of 4692 men in the control group who had prostate biopsies, nearly a *quarter* (24.4%) had histological evidence of prostate cancer at the end of the 7 years' follow-up. The investigators had expected 6.0% of the men to be diagnosed with prostate cancer during the trial, and the expected lifetime prevalence in these men was 16.7%. The trial investigators themselves stated '*The rate of 24.4% suggests the possibility of overdiagnosis of disease.*'¹⁵

The results from autopsy studies and the Finasteride trial are a warning. If healthy men have PSA tests, some will be diagnosed with prostate cancer that they would otherwise never have known about, and that would never have threatened their lives. This would be bad enough, but many men who are diagnosed with prostate cancer are offered treatment such as radiotherapy or surgery, and these treatments have significant side effects. The potential side effects include impotence, incontinence, diarrhoea, and death.⁹

Some of the men who suffer these side effects would never have known they had prostate cancer in the absence of screening, so they will have been directly harmed as a consequence of being screened.

Conclusion

Although it is possible to understand and perhaps explain opposing views on prostate cancer screening, examining the risks and benefits shows that prostate cancer screening is not justified at present. Whether there is any benefit from prostate cancer screening is unknown. It is inappropriate to support screening in the hope that it will be found to be beneficial, since this would be gambling with men's health.

Prostate cancer screening fails to meet criteria for screening,^{16,17} and carries potentially serious risks. In the absence of conclusive evidence of benefit, it is entirely possible that prostate cancer screening could cause more harm than good. Therefore, at present, it is unethical to offer prostate cancer screening.¹⁸ In the future, screening should only be offered if the randomised controlled trials of prostate screening that are currently underway, demonstrate a benefit.

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More of 'The scope and limitations of balneological treatment'

This extract comes from an article by Dr Arthur Wohlmann that was published in the New Zealand Medical Journal 1905, Volume 4 (14), p104–16.

Gout and Rheumatism.—These, of course, constitute the largest class of all, and, indeed, there are few cases of either complaint that will not derive benefit at some time or other from spa treatment. Patients, however, should be warned not to expect bony distortions to disappear, nor to expect permanent results from one course of baths. Pains may disappear and health be re-established, but for how long depends upon the individual: the attack may be “cured,” but the *tendency* to the disease remains.

In the rheumatoid cases, while the judicious use of baths will ease pain and stiffness, bathing is only one factor of a great number that must simultaneously be employed. To the stimulating effect of the change of environment to a health resort must be added the judicious use of every tonic influence within our power—fresh air, sunshine, liberal diet, drugs, and sometimes massage and electricity. A health resort at a high level, with cheerful surroundings and abundant sunshine, is essentially needed. Thus, so far as climatic considerations are concerned, I would put Hanmer before Rotorua, and Rotorua before Te Aroha. As to prognosis, provided incessant care can be guaranteed, and climatic and other conditions are favourable, most cases improve immensely. A few go progressively downhill in spite of everything that can be done.

In osteo-arthritis, on the other hand, treatment is directed to the local ailment rather than to the building-up of the constitution of the patient. No treatment, of course, can restore the worn-off cartilages or remove the bony protuberances, but the stiffness which has involved surrounding ligamentous and musculo-tendinous structures may be very greatly diminished and pain alleviated, so that often the most striking results are obtained. For such a condition douche massage, such as is obtainable at Rotorua, can be utilised to remove stiffness, while the heat and counter-irritation afforded by hot acid baths, and the local application of sulphurous-vapour baths, will relieve the pain.



The downside of screening

Breast cancer mortality has begun to decline in European countries and the USA, in part due to screening mammography. That is the good news. The bad news is the false positive recall rate. In a recent report from Norway it has been estimated that about 21% of women who begin screening in their early 50s will have a false positive recall during ten biennial screens.

And in the USA almost half (49%) of women aged 40–69 years will have at least one false-positive mammogram after ten screens. False positive recalls involve much physical and psychological suffering in women who do not have breast cancer. Very discouraging.

Lancet 2005;365:7–8

And now the really bad news

Global warming is pushing the world's climate past a point of no return that could be reached within a matter of years, a prestigious international taskforce has warned. The 14-strong taskforce, established a year ago, has been co-chaired by the former British cabinet minister Stephen Byers and the US Republican senator Olympia Snowe.

The danger level has been assessed as an average world temperature of two degrees celsius above the average global temperature in 1750—before the industrial revolution. But the taskforce says the average temperature has already risen by 0.8 degrees since then, the rise is accelerating, and the danger threshold may be reached within 10 years. A pity the US does not agree with the Kyoto protocol.

The Guardian Weekly, 28 January–3 February 2005, p1

Some good from the telly

Valerie Curtis is director of the Hygiene Centre at the London School of Hygiene and Tropical Medicine. In a recent paper she points out that “more than a million lives could be saved each year if people washed their hands at key times, because diarrhoeal and respiratory pathogens—the biggest child killers in the world today—are carried on people’s hands. Despite this, our studies in developing countries suggest that fewer than 10 per cent of mothers do this, and mothers in developed countries are often not much better. Evidently, teaching people about germs is not enough.”

So how do you change these bad habits? Dr Curtis says “we did this through a national campaign that included a television advertisement depicting a caring mother inadvertently contaminating her child's food via her hands after using the toilet. After watching it, 58 percent of the mothers we asked said they had changed their hand-washing habits.”

New Scientist, 18 December 2004, p21

Hospital beds across the ditch

In recent years, there has been emerging concern about declining access to hospital beds with its attendant flow-on of increasing waiting lists for elective surgery and overcrowded emergency departments. Lack of access to acute hospital beds has been variously attributed to reductions in hospital funding and bed availability, population ageing, and bed “blocking” by older people awaiting places in long-term care facilities.

This has a familiar ring—but it is actually a reference to the Australian scene. Furthermore analysis of hospital data from 1993–94 to 2001–02 reveals that the proportion of hospital beds occupied by older patients remained stable at 47%.

These trends are contrary to common perception. Ageing of the Australian population was not associated with an increase in the proportion of hospital beds used by older patients. Could this be true in New Zealand?

Med J Aust 2004;181:478–81

Don't overdo the steroids

Corticosteroids are commonly used as anti-inflammatory and immunosuppressive therapy in diseases such as asthma, inflammatory bowel disease, and inflammatory musculoskeletal disease. Predictable adverse effects include fluid retention, hypertension, diabetes mellitus, and obesity (all of which are independent risk factors for cardiovascular disease).

In a recent large cohort study from Scotland it has been shown that patients who take 7.5 mg or more of prednisone (or its equivalent) do have an increase in cardiovascular disease (relative risk 2.56).

Lower dosage did not increase the risk. The take-home message is obvious.

Ann Intern Med 2004;141:764–70



‘Opioid poisoning deaths in New Zealand (2001–2002)’ and the UK’s recent decision to withdraw the pain killer coproxamol

In the current issue of the *New Zealand Medical Journal*, Reith et al report on opioid poisoning deaths in New Zealand in the years 2001 and 2002.¹ One of the conclusions of that study is that the availability of dextropropoxyphene-containing medicines in New Zealand should be restricted. This conclusion is based upon a combination of a relatively high number of poisoning fatalities due to dextropropoxyphene-containing medicines compared with prescription volumes, in combination with a lack of clinical trial evidence of efficacy compared with paracetamol alone.

Recent developments in the UK, with the gradual withdrawal of coproxamol (a paracetamol-dextropropoxyphene fixed combination medicine) for marketing, highlight these concerns. Coproxamol products have been withdrawn from marketing in the United Kingdom upon the advice of the Committee for Safety in Medicines.² The Committee advised that ‘the efficacy of coproxamol is poorly established and the risk of toxicity in overdose, both accidental and deliberate, is unacceptable’. In addition, the risks of coproxamol have been reported for individuals other than those for whom it is prescribed, because of suicidal ingestions by taking the medicines of family members.³

The hazards of dextropropoxyphene-containing medicines are increased by co-ingestion of alcohol.³ Reith et al also highlight the contribution of alcohol and sedatives to opioid poisoning deaths. Hence, prescribers need to consider the added risk of co-prescribing opioids and sedatives, and the prescribing of opioids to patients at risk of ethanol and other substance abuse. The dispensing of ‘takeaway’ doses of methadone has been highlighted as a hazard by Reith et al and by a previous research report in New Zealand.^{1,4}

The case of dextropropoxyphene demonstrates the role that the New Zealand National Poisons Center, and its collaboration with the University of Otago, can play in medication safety. During the drug development process, medicines are not tested for their effects in overdose (or in combination with other drugs in overdose). Hence, new medicines are marketed with little or no knowledge of toxicity in overdose.

Toxicity in overdose also contributes valuable information about toxicity at normal doses in susceptible individuals (such as those with impaired drug elimination) and in combination with other drugs.^{5,6} This role can complement the activities of CARM, IMMP, and MARC in identifying medication hazards to the New Zealand population. In addition, the routine collection of poisoning fatality data from the Coronial Services Office should continue to be supported.

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Exceptional circumstances and heart transplantation

The newer immunosuppressive drugs, sirolimus and mycophenolate mofetil (MMF), have an increasing role following heart transplantation. Their use may lessen or avoid some of the adverse effects of standard immunosuppression, such as calcineurin inhibitor nephrotoxicity,^{1,2} and will reduce the incidence of acute and chronic rejection.³⁻⁵ In overseas centres, both sirolimus and MMF are becoming firmly established as first-line drugs following the results of large randomised controlled trials in heart transplant recipients.⁶

In New Zealand, applications for community use of these drugs must be made to the Exceptional Circumstances Panel (EC Panel). The EC Panel adheres to an arbitrary definition of 'rare' and 'unusual', namely a prevalence of less than 10 cases nationally. Applications which exceed this number, seemingly irrespective of merit, are refused. It is difficult to determine whether rarity is defined by the condition or its context, by the number of approvals or the number of applications, or whether subsequent deaths allow further accommodation for others.

Lately, the majority of applications for the community use of either sirolimus or MMF have been denied, but the EC Panel has then recommended instead that the drug be provided by the relevant DHB. In this instance, however, the DHB is not obliged to fund the medication and, conversely, is not permitted to provide the drug if the EC Panel refuses the application.

The sole criterion on which the DHB may base its decision is cost-effectiveness.⁷ If the cost of providing alternative treatments (e.g. dialysis) might exceed the cost of the drug, then the application is likely to be successful. The best interests of the patient and an evidence-based approach do not appear to be factors which weigh very heavily in this process, if at all. For heart transplant recipients, the need to prevent or reduce progressive renal impairment, transplant coronary vasculopathy and graft failure, or to allow the safe treatment of chronic gout by replacing azathioprine, may be difficult to justify using a commercial paradigm.

After nearly two decades of heart transplantation in New Zealand, it is not surprising that these serious conditions have become more common, and now exceed the EC Panel's criterion of 'rare'. Newer drugs such as sirolimus and MMF have the proven potential to favourably affect outcomes after heart transplantation, compared to standard immunosuppressive regimes. The EC Panel and Pharmac should advocate a more liberal access to these agents. A committee was established by Pharmac over a year ago to consider this issue, but it appears that no substantial progress has yet been made. The current entangled system requires a more flexible and cooperative approach in its response to reasonable requests.

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Gastric bypass surgery enables recovery of cardiac structure and function

Stubbs' prospective observations after gastric bypass surgery show remarkable sustained weight reduction in severely obese patients, with accompanying amelioration in hypertension, diabetes, asthma, hyperlipidaemia, and obstructive sleep apnoea.¹ Meta-analysis of bariatric surgery confirms similar long-term regression of cardiovascular and medical complications of obesity.²

Longstanding visceral obesity may lead to obesity-cardiomyopathy in a proportion of patients.³ Necropsy examinations of obese patients who died of congestive cardiac failure showed non-ischaemic cardiomyopathy.⁴ An obese Maori male who died shortly after admission from congestive heart failure, was shown at necropsy to have cardiomegaly involving all chambers with patent coronary arteries.⁵

Clinicopathological studies in 43 patients with congestive obesity cardiomyopathy demonstrated elevated right heart pressures, cardiac output, and pulmonary vascular resistance. Endomyocardial biopsy showed mild myocyte hypertrophy.⁶ Obesity results in alterations in cardiac structure and function in the absence of systemic hypertension and underlying organic heart disease. Increased total blood volume induces a high cardiac output state that leads to ventricular dilatation and eccentric hypertrophy of the left (and sometimes the right) ventricle.⁷

Prospective echocardiographic assessment after bariatric surgery in obese patients shows gradual regression in left ventricular mass,^{8,9} associated with improvement in cardiac function.¹⁰

Obesity congestive cardiomyopathy is often neglected, and distressed patients are left with limited physical capabilities and little zest for life on standard therapy. Dieting is ineffective,² however the cycle of events may conceivably be changed by bypass surgery where reversal of obesity-related cardiac dysfunction can occur.

Indeed, selected patients suffering a life-threatening poor quality existence may be revitalised by a proactive approach that gastric bypass surgery may provide.

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Hand-hygiene practices of medical staff: room for improvement

Good hand-hygiene practice remains the most effective means to reduce the transmission of nosocomial pathogens.^{1,2} Methicillin-resistant *Staphylococcus aureus* (MRSA) is an increasing problem in hospitals in New Zealand, and nosocomial transmission of MRSA occurs.

National guidelines for the control of MRSA within healthcare settings in New Zealand place strong emphasis on hand hygiene.³ MRSA is not the only multi-resistant organism in New Zealand; strains of extended spectrum beta-lactamase-producing Enterobacteriaceae are now present within our hospitals and community.⁴

Other multi-resistant pathogens such as vancomycin-resistant *Enterococcus* spp. and multi-resistant *Pseudomonas aeruginosa* and *Acinetobacter* spp. are less of a problem but cause major problems in overseas hospitals and have the potential to become problems in New Zealand.

Hand hygiene includes the use of plain or medicated soap and water, and alcohol hand rubs or gels. There are local as well as international guidelines¹ for hand-hygiene practices. Despite these recommendations, published compliance rates show that hand hygiene is done poorly by healthcare workers.^{1,2} Average adherence with recommended hand-hygiene practices is usually low, and is estimated to be below 50%.² Indeed, a recent study of handwashing in a teaching hospital showed non-compliance was high among physicians; compliance with recommended handwashing practice was only 30%.⁵

Little is known of the hand-hygiene practices of medical staff in the New Zealand setting. Our Infection Control Service (ICS) regularly holds education sessions for nursing staff, is involved in orientation sessions for all new staff, and actively promotes hand hygiene during their annual hand-hygiene week.

To obtain a baseline rate of adherence for future educational opportunities, we performed an observational study to record compliance with hand hygiene by medical staff during ward rounds. . Our observational study was conducted at Auckland City Hospital over a 12-month period, December 2003–November 2004. The period of observational coincided with the move to a newly constructed facility. The ICS had input into the placement of hand basins and dispensers for alcohol-based preparations containing 70% ethyl-alcohol w/w outside each room.

The study took place in the General Medical and General Surgical wards. Observers were recruited and trained to observe and record the hand-hygiene practices of medical staff on daily ward rounds. The trained observers were instructed about the local Hand Hygiene Policy, and this was the standard for assessment of compliance.

For each patient encounter, the observer recorded whether contact had occurred and whether hand hygiene was performed. Contact was defined as examining a patient or contact with the patient's bedding or clothing. Hand hygiene included washing with soap or antiseptic solution and water, or the use of an alcohol hand rub. These

observations were recorded on a data sheet and entered onto a Microsoft Excel spreadsheet.

Consent was obtained from the clinical leaders for each service. Medical staff were informed that a survey of hand-hygiene practices was to take place. The Auckland Ethics Committee approved the study.

Over the 12-month period, 397 episodes of patient contact were observed. The majority of observations, 374 (94%), occurred in the General Medical Service. The overall hand-hygiene compliance was 62%. Compliance differed between the senior medical staff (71%), registrars (57%), and house officers (47%). The median percentage for compliance by senior medical staff was 71% (range, 0–100%).

The senior medical staff were statistically more likely to be compliant with hand hygiene than the junior staff; difference 16% (95% confidence interval: 3.5–28.5) ($p=0.0012$, Fisher's exact test).

The compliance with hand hygiene in the observed group of medical staff was better than anticipated. Reported rates of compliance by healthcare workers in other observational studies show mean baseline rates of 5%–81% (overall average: 40%).¹ Our compliance rate overall was 62%.

Observed risk factors for poor adherence to recommended hand-hygiene practices include physician status, nursing assistant rather than a nurse, male gender, working in an intensive care unit, working during the week (Monday–Friday), wearing gowns/gloves, automated sinks, activities with high risk of nosocomial transmission, and high number of opportunities for hand hygiene per hour of patient care.²

Apart from the level of seniority, we did not look for other risk factors associated with poor adherence in our organisation—as the intent of the study was to obtain a baseline rate for hand-hygiene practice to allow us to assess improvement in adherence following intervention programmes.

We did not ask the observers to comment on any barriers to compliance. Perceived barriers to adherence with hand-hygiene practice recommendations include skin irritation caused by hand-hygiene products, inaccessible hand-hygiene facilities, interference with healthcare worker (HCW)-patient relationship, priority of care, wearing gloves, forgetfulness, lack of knowledge about hand hygiene policy, high workload, and understaffing^{1,2}

Considerable planning went into the placement of hand basins in the new hospital, and the introduction of wall-mounted alcohol hand-gel dispensers has certainly made hand hygiene more accessible. Similarly, the inclusion on some of the trolleys used by the medical staff during ward rounds of alcohol hand-gel dispensers has further improved access. Alcohol-based hand gels or rubs have the advantage over soap and water of being fast acting, having optimal antimicrobial spectrum, being available in a variety of packaging, taking less time to apply than a hand wash, and (with the addition of an emollient) being well tolerated by HCWs.⁶

Some reports have suggested that role models, group behaviour, and the level of managerial support influence the levels of compliance.^{2,7} One recent study showed that HCWs present in the room with a higher-ranking person or peer who did not perform hand hygiene were significantly less likely to wash their hands.⁸ But we were pleased to see that the senior medical staff had a rate of compliance of 71%.

There are several limitations in our study. Our observers were as unobtrusive as possible but observation bias and the Hawthorne effect must be considered. The study took place over 1 year and we hope that any initial change in usual practice was short-lived. We initially attempted to get equal numbers of observations in both the medical and surgical services but had difficulty recruiting observers in the surgical services.

Whether the results seen in the medical services can be generalised to the surgical services is uncertain. Nor did we observe the hand-hygiene compliance of nursing staff—the group of HCWs having the most patient contact. We chose to perform our observations during routine ward rounds, an activity more likely to be associated with good adherence. In the largest study published on compliance with hand-hygiene, nonadherence is highest in intensive care units, during procedures that carried the highest risk of bacterial contamination (before intravenous care, before respiratory care, and care between a dirty and a clean body site), and when the activity index is high.⁹

Hand hygiene remains one of the most important factors in the prevention of nosocomial infection. The cost to New Zealand of nosocomial infections is substantial; estimates place the cost of hospital-acquired infections, in all patients admitted to Auckland District Health Board [ADHB] hospitals in 1999, at close to NZ\$23 million.¹⁰

Interventions aimed at improving hand-hygiene practices are necessary to reduce transmission of nosocomial infections; the use of alcohol hand rubs, educational interventions to improve adherence, and the provision of senior management support are essential. Senior clinical staff (both nursing and medical) also need to take on the role of role models to improve adherence.

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Paul Arthur Searle

Paul Searle died suddenly at his home in Hamilton on 15 November 2004, aged 47.



Paul was born in Auckland in 1957. He was educated at St Kentigern's College, where he excelled academically.

He then attended Otago Medical School and graduated M.B.,Ch.B. in 1981.

Paul spent his House Surgeon and Registrar years in Dunedin, and it was during this time that he studied and passed his part 1 FRACS and developed his interest and passion in Emergency Medicine.

He spent 3 years as Registrar at Dunedin Hospital's Accident and Emergency Department.

Then in 1987, to further his career, he moved to Auckland to take up the position of Head Medical Officer at Greenlane's A&E Department, before finally moving to Hamilton to pursue his goal of establishing his own A&E centre

Davies Corner Accident & Medical Centre was then the next lucky recipient of his skills. In addition to managing the Centre, Paul also furthered his studies, obtaining his Postgraduate Diploma in Community Emergency Medicine.

As a doctor, Paul was highly respected, and will be sadly missed by both his staff and patients due to his down-to-earth no-nonsense approach.

In his spare time, Paul bred champion Angus bulls and was quite the gentleman farmer, however his main sporting love was the sea. As a boy he and his father would spend most weekends on the Auckland Harbour and Hauraki Gulf where many hours were spent fishing, yachting, and enjoying numerous sailing regattas. Indeed, throughout his life he maintained, and shared with friends, an active interest in boating and the thrill of the catch.

Paul is survived by his wife, Debbie, and his 12-year-old daughter, Samara.

We are grateful to Paul's sister, Kayelesley, for writing this obituary.



The Hawke's Bay Medical Research Foundation: funding applications invited

Applications for funding for Research Projects are now being accepted by the Foundation.

A Studentship Grant up to \$NZ2,600 based on a 10-week course is also available.

Applications can obtain an Application Form together with the guidelines of assessment by phoning the Secretary on:

(06) 879 9190

or writing to:

The Secretary, P O Box 596, Napier

or email:

jmbax@xtra.co.nz

(website: www.hawkesbaymedicalresearch.org.nz)

Applications close March 31, 2005

(Priority will be given to applications from appropriate persons employed in Hawke's Bay or having an association with the region.)

**JM Baxter
Secretary**



Computerworld Excellence Awards 2005 (Excellence in the Use of IT in Health): entries invited

The annual Computerworld Excellence Awards are approaching. For the first time, we have a category for Excellence in the Use of IT in Health. This award is for those who have improved the delivery of healthcare in New Zealand through the implementation of IT.

We are now calling for entries from teams and individuals doing great things with IT in New Zealand. The Excellence Awards has established itself as the leading recognition programme within the New Zealand IT industry, with many IT professionals striving to achieve the accolade of excellence.

Now in their 8th year, the awards honour outstanding achievement by users of information technology in New Zealand. In the 2004 year, the event attracted over 300 entries with an equally large turnout at the gala presentation dinner. There are 14 separate categories for 2005, covering an individual award (CIO of the Year) and group awards in areas such as Small to Medium Enterprises, Education, and Government.

An exciting new development for 2005 sees the launch of 3 new categories— Excellence in the Use of IT in for Export, Excellence in the Use of IT in Health, and Excellence in the Use of IT for a Community Project. Each represents the changing face and the social impact of information technology in New Zealand.

For the second year, the Awards will not only recognise, but reward, innovative IT applications and solutions implemented within Not for Profit Organisations. Often these groups work with extremely limited budgets and resources, however, what they achieve with IT can have a huge positive impact on the wider community. Proudly sponsored by Westpac, the category winner will be rewarded with a NZ\$10,000 cash prize donation.

Each category is judged by an independent panel of three. Among the 42 Judges involved in the awards are some of New Zealand's most prominent IT and business leaders.

For full category, eligibility and entry form please go to www.idg.net.nz/cwea – or contact Darrell Denney on (09) 302 5727, or email: events@idg.co.nz

Entries will close on 18 March 2005, so be sure to act quickly to register your entry for the event.





New Zealand Evidence Based Practice Awards

Entries close on the 31st March 2005

The New Zealand Branch of the Australasian Cochrane Centre and the New Zealand Guidelines Group are offering Evidence Based Practice Awards. These awards are for those who have demonstrated the best use of research evidence, contained in a guideline or from the Cochrane Library, to enhance patient care. Entries are sought from all healthcare workers, including primary/community care workers, librarians, students/health professionals in training, government policy makers and consumer organisations.

To enter go to <http://www.cochrane.org.nz/>

The Awards

The value of the Guideline Users and Cochrane Users awards are \$500 each. These awards are open to all applicants New Zealand wide. There will also be a runner up prize for the Guideline award of \$300.

Entry to the awards is free.

Entries may be submitted by individuals or in partnership with others.

The awards will be shared equally among all applicants named on the entry form.

Conditions of Entry

Applicants must submit an abstract of no more than 500 words, suitable for publication, explaining how the evidence, contained in the Cochrane Library or a guideline, was used to enhance the health care of New Zealanders. Evidence used within the last 5 years is applicable.

Abstract Requirements

The abstracts for each award, of no more than 500 words, must address the following five questions (when relevant):

- What clinical situation led you to seek information?
- What information did you use from the Cochrane Library or Guideline?
- What did you do with this information?
- What changes were made to patient care?
- What were the real benefits to the patient(s) over time?

Referees

Applicants are required to nominate two referees who can confirm the effect of the intervention resulted from evidence accessed from the Cochrane Library or Guideline.

Who Can Enter

The New Zealand Cochrane Users Award is open to all New Zealand Residents with the following exceptions:

- Employees of Update Software and John Wiley & Sons and their families.
- Employees, directors and Advisory panel members of the New Zealand Branch of the Australasian Cochrane Centre and their families.
- Employees, directors and Advisory panel members of the New Zealand Guidelines Group and their families.
- The Judges



GRAHAM AITKEN NUFFIELD TRUST

Graham Aitken Nuffield Medical Postgraduate Travelling Scholarship

Applications are invited from well-qualified New Zealand medical graduates in the 25–35 age group for the above Scholarship.

The purpose of the Scholarship is to provide travel funds to enable New Zealand graduates to further their clinical medical training and research interests in the United Kingdom.

The Scholarship will provide up to three return air fares to the UK, together with allowances amounting to \$3000.

Candidates for the Scholarship must submit a training or research programme for approval together with the name of a person in the UK who will provide salary and facilities.

For further information please consult the Deans of the Schools of Medicine, or write to:

Professor A D Campbell, Graham Aitken Nuffield Trust, C/- Chemistry Department,
University of Otago, P O Box 56, Dunedin.

Applications must be submitted to Professor Campbell by 31 March 2005



Brain and Spinal Tumors of Childhood

Walker PA, Perilongo G, Punt JAG, Taylor RE (eds). Published by Hodder Arnold, 2004. ISBN 0340762608. Contains 531 pages. Price \$360.00

This book gives a very comprehensive overview of the rapidly evolving field of the treatment of children with tumours of the brain and spinal cord.

The editors and individual authors have presented an overwhelming case for the treatment of children with these tumours to be undertaken jointly by a multidisciplinary team comprising specially trained people. The members of such a multidisciplinary team have been identified as having 55 different roles and should exhibit what the authors describe as 'joined-up thinking'. The book is not intended to be a 'bible' but rather a comprehensive handbook for all these people.

The editors rightly point out that there is a hiatus in most of the books on tumours of the central nervous system; specifically that tumours in children often receive inadequate attention (particularly embryological, genetic, and molecular aspects), and insufficient detailing of the effects and outcomes of treatment. This hiatus is rapidly closing, however, with expanding research and advances in the genetics and biology of these tumours, with advances in imaging, and now with increasing pooling of information and cooperation between treatment centres on a worldwide basis. Indeed, this book makes a major contribution to closing this hiatus.

There are 65 contributing authors covering a wide range of specialties and the book is arranged in six parts: *Introduction, Epidemiology, Diagnosis and Treatment Planning, Treatment Techniques and Neurotoxicities, Disease-Specific Multidisciplinary Management, and Late Consequences and Supportive Care*. Each part contains between 2 to 13 chapters. It is the inclusion of certain specific topics that broadens the interest and appeal of this book, making it extremely informative by presenting a wide variety of topics in sufficient detail to interest and educate all who use it.

Topics include (in Parts 1 to 4): *Epidemiology, Neuro-embryology, Tumour biology, Pathology and molecular classification, Clinical syndromes, Diagnostic imaging* (with excellent quality illustrations), *Clinical trials, Neurosurgical techniques* (in reasonable detail with the basic principles adequately described and aimed at paediatric medical staff, paediatric oncologists, radiation specialists, nursing staff, etc), and *Radiotherapy techniques* (in reasonable detail with the basic principles adequately described and aimed at neurosurgeons, paediatric medical staff, nursing staff, etc).

Part 5 contains 13 chapters—the first 12 chapters each dealing with a specific tumour type and the last entitled '*Exploiting biology for therapeutic gain*'. Part 6 comprises 6 chapters—*Toxicity and late effects, Physical care, Rehabilitation and complementary therapies, Cognitive development and educational rehabilitation, Quality of survival, Information needs for children and families, and Future challenges* (this section nicely broadens the appeal of this book and contributes to the concept of 'joined up thinking').

Additional features that I found very useful and informative include a three-page list of abbreviations at the start, and a 16-page, well-set-out, and well-illustrated *Timeline*, outlining the development of Paediatric Neuro-oncology—under the headings *Neurosurgery, Imaging, Radiotherapy, and Chemotherapy*.

I believe this book offers a timely and major contribution to the treatment of children with brain and spinal tumours, and I strongly recommend this book as an essential text for all Units involved in the treatment of these children as well as essential reading for the many and varied personnel working in these Units.

Martin R MacFarlane

Neurosurgeon, Department of Neurosurgery
Christchurch Hospital