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This Issue in the Journal

Trends in survival and life expectancy by ethnicity, income and smoking in New Zealand: 1980s to 2000s
Kristie N Carter, Tony Blakely, Matthew Soeberg

Life tables have been successfully developed for subpopulations (by ethnicity, income and smoking) over 20 years in New Zealand. Ethnic and income gaps in life expectancy have widened over the past 20 years. Surprising results were found for smoking by ethnicity with the greatest gaps in life expectancy between Māori and non-Māori for never smokers compared to current smokers.

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We estimated additional gains in life expectancy by 2040 if no one smokes tobacco by 2020. Compared to the 2006 census smoking rates continuing indefinitely, we estimate that if nobody smokes tobacco from 2020 onwards there will be about 5 years of additional life expectancy for Māori (range 2.5 to 7.9 years), about 3 years for non-Māori (range 1.2 to 5.4 years), and therefore about a 2 year closing in ethnic gaps in life expectancy (range 0.3 to 4.6 years).

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Lavinia Tan, Tony Blakely, June Atkinson

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The prevalence of colorectal adenomas in Māori and New Zealand Europeans parallels colorectal cancer rates
Graeme Dickson, Chris W Cunningham, Susan Parry

NZ has a high incidence of colorectal cancer. Māori have a documented incidence that is approximately half that found in NZ Europeans, possibly the result of under-reporting. To find out whether this is true we studied colonoscopy records from the

Middlemore Colonoscopy Audit Database between 2001 and 2005. The comparative rates of adenomas in NZ Europeans and Māori were 16.7% and 8.7% respectively. We have documented the prevalence of colorectal adenomas in Māori to be approximately half that found in NZ Europeans. This finding mirrors the reported difference in colorectal cancer incidence and supports this being a real finding, rather than a consequence of under reporting.

Orbital infection in New Zealand: increased incidence due to socioeconomic deprivation and ethnicity

Nicholas R Johnston, Gordon Sanderson

This study used data collected from all hospital discharges in NZ. It has identified that orbital infections—a condition that is normally admitted to hospital as it is sight and life-threatening—is rare: 1.31 cases per 100,000 per year. Orbital infections are often caused by upper respiratory tract infections with sinusitis. It has shown that Maori (1.9×) and Pacific people (3.6×) are more likely to be admitted with orbital infections than Europeans in NZ. It also shows that as socioeconomic deprivation increases so does the rate of admission with orbital infection. The most deprived group has about 3 times the rate of orbital infection. This relationship occurs within the ethnic groups as well, meaning the most deprived Pacific people have about 6 times the rate of orbital infection compared to Europeans.

Patients "falling through the cracks". The Canterbury Charity Hospital: initial progress report

Philip F Bagshaw, Randall A Allardyce, Susan N Bagshaw, Brian W Stokes, Carl S Shaw, Lorraine J Proffit, M Gary Nicholls, Evan J Begg, Christopher M Frampton

The Canterbury Charity Hospital Trust, established in 2004, completed the purchase of a residential villa in 2005 and converted it into the Canterbury Charity Hospital. This was to provide free selected elective healthcare services to patients in the Canterbury region who were otherwise unable to access treatment in the public health system or afford private healthcare. It performed its first operations in 2007. By the end of December 2009, 115 volunteer health professionals and 79 non-medical volunteers had worked at the Hospital, provided a total of 966 outpatient clinic appointments, of which 609 were initial assessments, and performed 610 surgical procedures. Funding of $NZ4.3 million (end of last financial year) came from fundraising events, donations, grants and interest from investments. There has been no government funding. The overwhelming community response raises the question of whether the current public health system needs attention to be re-focused on unmet need. We contend that unless this occurs it might be necessary to establish charity-type hospitals elsewhere throughout the country.

Life chances going up in smoke

Philippa Howden-Chapman, Bridget Robson, Geoff Fougere

Health starts where we live, learn, work and play. This is the deliberate reframing of the social determinants of health inequalities by the United States Robert Wood Johnson Foundation, designed to better appeal to politicians, policymakers and the public.[1]

For more than a decade, a more explicit emphasis on the social, economic, cultural and environmental determinants of health has driven the work of the World Health Organization.[2] In New Zealand, efforts to reduce health inequalities have been made by ministers, ministries,[3,4] district health boards,[5] regional public health services, primary health organisations, NGOs[6] and public health academics,[7–9] who often work in partnership with these other organisations. Recognition of our obligations under the *Treaty of Waitangi* has helped to focus the activities.

Last year, the media reported that, in what seems to be largely an act of self-censorship, words such as 'the organised efforts of society', 'public health', 'inequalities and advocacy', were among the phrases that should no longer be used in Ministry of Health documents.[10]

When explicit discussion of health inequalities and public health is censored, and inequality becomes a politically prescribed word, we run the risk of collective amnesia. Whatever language we use, the underlying determinants of health, the enduring power structures of society, are slow to change. However, we know from work in New Zealand and the United Kingdom—where there is closer, more regular monitoring of policies designed to reduce health inequalities—health inequalities are easier to let grow than to reduce.[11] If we do not measure and analyse these trends, we can all too easily find that just like weight gain, the scales, when we finally look at them, are telling us what we would rather not know.

Unlike GDP, income inequality is not measured routinely in New Zealand. In the mid-1990s, it was the UK Roundtree Foundation that alerted us to the dubious honour of having the largest percentage point change in the Gini coefficient of inequality in the preceding decade of the OECD's industrialised countries.

In the mid-2000s, New Zealand was in the top quarter of the OECD for inequality of incomes, as measured by the Gini coefficient—only below Mexico, Turkey, Portugal, United States and Poland and on par with the United Kingdom.[12] It appears that income inequality in New Zealand reduced slightly in the middle of last decade, but associated harmful behaviours, have been slower to decrease.

One of the consequences of an unequal distribution of resources in New Zealand in terms of income, wealth, education, employment and home ownership opportunities has been the smoking epidemic which hit the Māori community (tobacco was unknown before European colonisation) after it had already peaked in European men.

These inequalities may also prevent more rapid falls in smoking prevalence than might otherwise be the case.

Māori once again appear to be the 'shock absorbers of the economy' with unemployment rates, like smoking rates, over double those for European.[13] The additional burden of tobacco exacerbates the costs of widespread unemployment and detrimentally affects *whānau* (extended family) well-being. The Māori Affairs Select Committee has laudably adopted the *Tupeka kore* vision of a tobacco-free New Zealand by 2020. Radical action and strong political will is now required to bring this about.

In the current NZMJ, Blakely and colleagues, using a thought experiment, pose a counterfactual to the current position, what *would* happen to health inequalities, if nobody smoked cigarettes after 2020? Compared to the prevalence of smoking in 2006, ethnic inequalities in life expectancy would fall by 0.3 to 4.6 years (on average 1.8 years, but consistently greater for females). The authors maintain that making New Zealand tobacco-free is one of the most important steps to achieving health equality between Māori and non-Māori by 2040—200 years after the signing of the *Treaty*.

The eminent philosopher John Rawls used just such a thought-experiment as the basis of his theory of justice. He posed a 'veil of ignorance' as a device, which decisionmakers, for example, could use when considering policy options. If I did not know my position in society, if I had no idea as to whether I was rich or poor, Māori or European, what policies would I favour? Blakely and colleagues' careful social epidemiology over the last decades suggest that rich or poor, Māori or non-Māori, legislating to phase tobacco out as a legal policy in New Zealand would be an important step in reducing health inequalities in New Zealand.

In a truly Smokefree New Zealand, we would expect further acceleration of the long-run trend of closing ethnic gaps in mortality that we enjoyed after World War 2 and have recently glimpsed again. The latest New Zealand Values Survey clearly indicates this vision is strongly shared by most New Zealanders.[14]

If we, as health professionals, can find and use words that resonate with the rest of society, the vision of a tobacco-free New Zealand—where life chances are not determined by ethnicity—is indeed possible.

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References:

1. Robert Wood Johnson Foundation. A New Way to Talk About the Social Determinants of Health. Vulnerable Populations Portfolio. Princeton, N.J.: Robert Wood Johnson Foundation, 2010.

2. Commission on Social Determinants of Health. Closing the Gap in a Generation: health equity through action on the social determinants of health. Final Report of the Commission on Social Determinants of Health. Geneva: World Health Organization, 2008.

3. Ministry of Health. Reducing Inequalities in Health. Wellington: Ministry of Health, 2002.

4. Ministry of Social Development. The 2008 Social Report: indicators of social wellbeing in New Zealand. Wellington: Ministry of Social Development, 2008.

5. Signal L, Martin J, Reid P, Carroll C, Howden-Chapman P, Keefe Ormsby V, et al. Tackling health inequalities: moving theory to action. International Journal of Equity in Health 2007;6(12):doi:10.1186/1475-9276-6-12.

6. Signal L, Rochford T, Martin J, Dew K, Grant M, Howden-Chapman P. Strengthening the capacity of mainstream organisations to tackle inequalities in health for Māori: a heart health case study. Health Promotion Journal of Australia 2004;15(3):221-225.

7. Howden-Chapman P, Tobias M, editors. Social Inequalities in Health: New Zealand 1999. Wellington: Ministry of Health, 2000.

8. Robson B, Harris R, editors. Hauora: Māori Standards of Health IV: A study of the years 2000-2005. Wellington: Te Rōpū Rangahau Hauora a Eru Pōmare, 2007.

9. Blakely T, Martin T, Atkinson J, Yeh L, Huang K. Tracking Disparity: Trends in ethnic and socioeconomic inequalities in mortality, 1981-2004. Wellington: Ministry of Health, 2007.

10. Ministry issues list of words and phrases. http://www.stuff.co.nz/national/health/1999869/Ministry-issues-list-of-words-and-phrases 2009.

11. Howden-Chapman P. Evidence-based politics: How successful are government reviews as policy instruments to reduce health inequalities? Social Science and Medicine (in press).

12. Hills J, Brewer M, Jenkins S, Lister R, Lupton R, Machin S, et al. An Anatomy of Economic Inequality in the UK. London: Centre for Analysis of Social Exclusion, 2010.

13. Department of Labour. Employment and Unemployment – June 2010 quarter, http://www.dol.govt.nz/publications/lmr/lmr-hlfs-summary.asp

14. Carroll P, Casswell S, Huakau J, Howden-Chapman P, Perry P. The Widening Gap: perceptions of poverty and income inequalities and implications of population health Social Policy Journal of New Zealand (in press).

Is bowel cancer screening important for Māori?

Diana Sarfati, Sarah Hill, Tony Blakely, Bridget Robson

In this edition of the *Journal*, Dickson et al report lower prevalence of colorectal adenomas in Māori compared with NZ European patients undergoing colonoscopy at Middlemore Hospital in Auckland, New Zealand (NZ).[1]

Compared with European patients, Māori were approximately half as likely to be diagnosed with colorectal adenomas (commonly regarded as a risk factor or precursor to colorectal cancer). While this study is based on a symptomatic population and may therefore be subject to selection bias, the findings are consistent with the lower incidence of colorectal cancer observed in the Māori population overall.[2–4]

Following the recent announcement of a pilot bowel cancer screening programme (to be introduced in late 2011), the study by Dickson et al is a salient reminder of the need to consider cancer control measures in terms of their impact on both total cancer burden and inequalities in cancer.

Given their lower incidence of colorectal cancer, it may be tempting to conclude that bowel cancer is less of a problem for Māori than for non-Māori in New Zealand. A closer look at the evidence shows this is not the case: bowel cancer screening is just as important for Māori as for non-Māori New Zealanders.

Putting aside the background of consistently large health inequalities between Māori and non-Māori, the increasing contribution of cancer to these inequalities[5] and the fact that the national screening programmes for breast and cervical cancer have yet to achieve equitable coverage for Māori women, there are other important reasons for considering that a bowel cancer screening programme must focus on delivering screening in a way that is acceptable to Māori.

First, while Māori currently have lower incidence of colorectal cancer compared with NZ Europeans, their rates are increasing more rapidly. The recent *CancerTrends* study indicates that rates of colorectal cancer are converging for Māori and non-Māori population groups.[2] Dickson et al note the potential for under-reporting to contribute to reports of lower cancer incidence in Māori[1]—a problem which previously led to substantial underestimates of mortality among Māori New Zealanders.[6] By linking census and Cancer Registry data and using the former to categorise self-identified ethnicity, *CancerTrends* overcomes problems of incomplete ethnicity recording at the time of diagnosis to generate robust time-trend information on cancer incidence by ethnicity from 1981 to 2004.

Results from the *CancerTrends* study confirm around 40% lower incidence of colorectal cancer among Māori over the time studied, with pooled standardised rate ratios (SRR) of 0.61 (95% CI 0.56–0.66) for Māori compared with NZ Europeans.[2] The ethnic gap in colorectal cancer incidence appears to have narrowed during the study period, however, with the Māori / European SRR among men increasing from

0.48 in 1981 to 0.73 in 2001 (p for trend=0.04). A similar but less marked trend is also seen among women.[2]

Second, as Dickson et al note, Māori and NZ Europeans have similar mortality from colorectal cancer despite Māori having lower incidence.[3 4 7] In other words, colorectal cancer imposes a similar mortality burden on both Māori and European New Zealanders.

As with incidence trends, the pattern of high colorectal cancer mortality in Māori is a relatively recent phenomenon. In the early 1980s, colorectal cancer mortality rates were lower for Māori compared with NZ Europeans by 39% and 62% in men and women respectively. Since then, colorectal cancer mortality rates have generally increased among Māori while remaining stable or declining among NZ Europeans.[2]

Third, as Dickson et al note there is considerable evidence that survival among Māori with colorectal cancer is lower than that for non-Māori.[3,4,8,9] Part of this survival disparity may relate to colorectal cancer being diagnosed at a later stage among Māori,[3 9] but other factors—including health care—are also implicated. A detailed study by Hill et al (2010) concluded that the most important factors contributing to poorer survival in Māori were patient comorbidity and markers of health care access.[8]

Based on a cohort of Māori and non-Māori patients diagnosed with colon cancer between 1996 and 2003, the study found Māori patients were around 30% more likely to die from their cancer compared with non-Māori with patient comorbidity and markers of health care access each accounting for around a third of this survival disparity.

It can be tempting to attribute differences in cancer incidence and survival to inherited characteristics, but there is currently little evidence that they play an important role for ethnic differences in colorectal cancer.[10] The rapid increase over the last 25 years in colorectal cancer incidence among Māori is supportive of a change in exposure to environmental factors.[7] Environmental factors—including health services—are also likely to have a far greater influence on survival, and are also amenable to change.

The study by Hill et al found no evidence of more aggressive tumours in Māori compared with non-Māori patients, but did find evidence of poorer access and quality of care, contributing to poorer survival.[8 11] Evidence from the USA shows a similar picture in relation to poorer colorectal cancer outcomes in African American compared with White patients.[12] These findings highlight the need to consider the differential effectiveness of health service provision for patients from different ethnic groups. While health services have the potential to reduce the burden of cancer, they also have the potential to increase cancer inequalities.

Planning is now well underway for a pilot bowel cancer screening programme. There are several reasons why it will be important to pay attention to the effectiveness of this programme for Māori as well as European New Zealanders. First, while Māori currently have lower rates of colorectal adenoma and cancer these are increasing rapidly and are likely to converge with European rates in the near future. Second, early detection of colorectal cancer offers particular benefit to Māori patients, who are currently more likely to have their cancer diagnosed at a later stage.[3] And third, introduction of a screening programme has the potential to either reduce or increase

ethnic disparities in colorectal cancer survival depending on the accompanying investment in management of colon cancer.[13]

Māori patients currently receive less access to high-quality colorectal cancer care compared with non-Māori patients.[11] The introduction of a screening programme is likely to place greater pressure on diagnostic and treatment services (such as colonoscopy); without accompanying investment in these services, existing vulnerabilities in service provision are likely to be exacerbated with the risk that the access gap for Māori becomes even more pronounced.

On the other hand, if screening is accompanied by renewed investment in cancer services, better auditing of cancer care pathways and careful attention to cancer outcomes, there is potential for it to strengthen the pathway of colorectal cancer care. Such strengthening will offer particular benefit to those populations currently underserved—not only Māori, but all individuals and population groups who do yet receive benchmark treatment and care.

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References:

1. Dickson G, Cunningham C, Parry S. The prevalence of colorectal adenomas in Māori and New Zealand Europeans parallels colorectal cancer rates. NZ Med J 2010;123(1320). http://www.nzma.org.nz/journal/123-1320/4260

2. Blakely T, Shaw C, Atkinson J, et al. CancerTrends: Trends in Incidence by Ethnic and Socioeconomic Group, New Zealand 1981-2004. Wellington: University of Otago and Ministry of Health (in press).

3. Robson B, Purdie G, Cormack D. Unequal impact: Maori and non-Maori cancer statistics 1996-2001. Wellington: Ministry of Health, 2006.

4. Robson B, Purdie G, Cormack D. Unequal Impact: Maori and Non-Maori Cancer Statistics by Deprivation and Rurality 2002-2006. Wellington: Ministry of Health, In press.

5. Blakely T, Tobias M, Atkinson J, et al. Tracking Disparity: Trends in ethnic and socioeconomic inequalities in mortality, 1981-2004. Wellington: Ministry of Health, 2007.

6. Blakely T, Robson B, Atkinson J, et al. Unlocking the numerator-denominator bias. I: Adjustments ratios by ethnicity for 1991-94 mortality data. The New Zealand Census-Mortality Study. NZ Med J 2002;115(1147):39-43.

7. Shaw C, Blakely T, Sarfati D, et al. Trends in colorectal cancer mortality by ethnicity and socio-economic position in New Zealand, 1981-99: one country, many stories. Aust NZ J Public Health 2006;30(1):64-70.

8. Hill S, Sarfati D, Blakely T, et al. Survival disparities in Indigenous and non-Indigenous New Zealanders with colon cancer: the role of patient comorbidity, treatment and health service factors. J Epidemiol Comm Health 2010;64:117-23.

9. Jeffreys M, Stevanovic S, Tobias M, et al. Ethnic differences in cancer survival in New Zealand: linkage study. American Journal of Public Health 2005;95(5):834-7.

10. Pearce N, Foliaki S, Sporle A, Cunningham C. Genetics, race, ethnicity, and health. BMJ 2004;328(7447):1070-2.

11. Hill S, Sarfati D, Blakely T, et al. Ethnic disparities in treatment of Māori and non-Māori New Zealanders with colon cancer. Cancer (in press).

12. Marcella S, Miller JE. Racial differences in colorectal cancer mortality. The importance of stage and socioeconomic status. J Clin Epidemiol 2001;54(4):359-66.

13. Sarfati D, Shaw C, Simmonds S. Inequalities in cancer screening programmes. Int J Epidemiol 2010;39:766-8.

Trends in survival and life expectancy by ethnicity, income and smoking in New Zealand: 1980s to 2000s

Kristie N Carter, Tony Blakely, Matthew Soeberg

Abstract

Background Survival and life expectancy are commonly used metrics to describe population health. There are two objectives to this paper: (1) to provide an explanation of methods and data used to develop New Zealand life-tables by ethnic, income and smoking groups; and (2) to compare cumulative survival and life expectancy trends in these subpopulations.

Method We generated sex-specific life-tables for seven subpopulations: ethnicity (Māori and non-Māori); income tertiles; smoking (never and current); and two-way combinations (ethnicity by income; ethnicity by smoking; and smoking by income). This was repeated for five census-mortality cohorts (1981–84, 1986–89, 1991–94, 1996–99, and 2001–04).

The method used to create the life-tables brings together three pieces of information: (1) the official Statistics New Zealand (SNZ) life-tables by year and sex; (2) the proportionate distribution of the total population by subpopulation (e.g. smoking prevalence); and (3) estimates of the differences in subpopulation mortality rates (from the New Zealand Census-Mortality Study [NZCMS]).

Results Survival and life expectancy improved in all subpopulations across the five census cohorts. However, improvements were greater in non-Māori compared to Māori and high income compared to low income subpopulations. This led to widening of the gap in life expectancy between 1981 and 2001 between Māori and non-Māori (males), which increased from 5.4 years in 1981 to 9.0 in 2001 and between low income and high income which increased from 4.4 in 1981 to 6.5 in 2001 for males.

The gap in life expectancy between current and never smokers in 1996 was 7.6 in males and 6.7 in females. However, the size of this gap varied by ethnicity: 7.3 and 6.2 for non-Māori males and females, and 4.3 and 3.9 for Māori male and females. Correspondingly, the gap in life expectancy between Māori and non-Māori is greater among never smokers (9.7 and 8.4 for males and females) than among current smokers (4.3 and 6.6 for males and females).

Conclusion Life-tables have been successfully developed for subpopulations in New Zealand, and provide an alternative understanding of health and life in New Zealand over the past 20 years. Ethnic and income gaps in life expectancy have widened, and perhaps surprising results were found for smoking by ethnicity. These life-tables provide an important basis for subpopulation modelling and projections, and are freely available to researchers.

Background

Life expectancy is a commonly used metric to describe population health.[1,2] Life expectancy is calculated from life-tables that give mortality rates by single year of age, up to 100 or more years of age, usually for a 'synthetic' population of 100,000 people to which current mortality rates are applied, i.e. what is called period life expectancy. Whilst an artificial construct that does not actually represent any birth cohort's expectancy of life, they are very useful summary health measures for policymaking, monitoring and communication to the public.

Life-tables are useful for modelling the impact of interventions and calculating other epidemiological measures. For example, relative cancer survival is determined by subtracting expected survival (derived from life-tables) from the observed survival of cancer patients, and to be accurate requires having life-tables for each subpopulation within which one wishes to calculate cancer survival (e.g. by income group, or smoking group).[3-5]

It is this methodological requirement for subpopulation life-tables in order to calculate accurate cancer survival estimates that precipitated the work and outputs presented in this paper. However, subpopulation life-tables and life expectancy are also useful outputs in their own right for describing differences in health status between population groups, and reflecting on societal and other casual mechanisms that have got us to where we are.

In this paper we present life-tables and life expectancies from 1981 to 2001 for various groupings of ethnicity, income and smoking status. An accompanying paper in this issue, uses the ethnic by smoking life-tables to estimate life expectancy in New Zealand in 2040.[6]

Calculating subpopulation life-tables requires reliable data on mortality rates for each subpopulation, which is often not the case when one is relying on routine mortality data that is not linked to census (denominator) data. The existence of linked census-mortality data in New Zealand, especially in light of smoking data being collected on 1981 and 1996 (and more latterly 2006) census data, provides a strong basis for the development of subpopulation life-tables.

Ethnic-specific life-tables are available from Statistics New Zealand (SNZ), but are prone to error prior to 1995-97 due to historic undercounting of Māori deaths (and over counting of non-Māori deaths).[2, 7-9] The Ministry of Health have created life-table estimates by ethnicity and New Zealand Deprivation Index.[10, 11] The existence of linked census-mortality data provides a rich data source for direct calculation of age-specific variations in mortality between ethnic, income and smoking groups that is not usually available to demographers generating life-tables.

Thus, the two objectives to this paper are:

- Explanation of the methods and data used to develop New Zealand life-tables by ethnic, income and smoking groups.

- Comparison of cumulative survival and life expectancy trends in these subpopulations.

Methods

Overview—We generated life-tables for seven subpopulations, each for males and females separately, namely: two ethnic groups (Māori and non-Māori); three income groups (tertiles of household income: low, medium, high); and two smoking groups (never and current). Then we generated life-tables for two-way combinations of these subgroups (for males and females): six ethnic by income groups; four ethnic by smoking groups; and six smoking by income groups. That is, another 16 life-tables for each sex. Finally, this was repeated for each of the five census-mortality cohorts (1981–84, 1986–89, 1991–94, 1996-99, and 2001-04), a total of 158 life-tables (110 for various ethnic and income combinations but only 48 for life-tables involving smoking as it was only elicited at the 1981 and 1996 censuses).

Before giving more detail, it is useful to first understand that our method brings together three pieces of information:

- The official SNZ life-tables by year and sex (i.e. all ethnic, income and smoking groups combined).

- The proportionate distribution of the total New Zealand population by subpopulation (e.g. smoking prevalence) (sourced from census data).

- Estimates of the differences in subpopulation mortality rates (sourced from the New Zealand Census-Mortality Study [NZCMS]).

These three inputs were combined to produce mortality rates by single year of age for each subpopulation, and thence complete life-tables for each subpopulation. Put another way, we used NZCMS information on subpopulation differences in mortality applied to official life-tables.

A brief overview of life-table terminology is provided in Appendix 1 at the end of this article.

Data

The SNZ m_x (mortality rate in each single year age group on official life-table)

Complete period life-tables, by sex and year of age, are available from SNZ for the three years surrounding each census, 1980-1982, 1985-1987, 1990-1992, 1995-1997, and 2000-2002.[2, 12] In the SNZ life-tables the construction of each complete life-table involved two stages. First, central death rates (m_x) were calculated for each age (x), except the first year of life, and were then smoothed to eliminate any apparent irregularities. Second, the smoothed rates were used to calculate a set of age-specific probabilities of death (q_x), which were then used to derive other life-table functions.[2, 12]

The proportion of the population in each social group of interest within each single year age group

We used census data to determine the proportion of the population within each one year age group in each census year (i.e. 1981, 1986, 1991, 1996, 2001) in each subpopulation of interest (including combinations of, say, ethnicity by income). If the census count by single year of age in the subpopulation was less than 5 (i.e. as often occurred at older ages), age was aggregated up to 5 year age bands, and the subsequently calculated proportion was assumed to apply uniformly to all subsumed single-year ages.

The estimated mortality rate (ratios) between social groups from NZCMS data

Much previous work using NZCMS data has documented social inequalities in mortality [8], and we used this prior information to specify analyses for generating life-tables. Briefly, we pooled all NZCMS data (i.e. 1981-84, 1986-89, 1991-94, 1996-99 and 2001-04), and ran pre-specified Poisson regression models by sex based on prior information to specify interactions (SAS code is available at www.uow.otago.ac.nz/nzcms-info.html).

We conducted the modelling of NZCMS data on observations with complete data for each analysis (nearly all observations for smoking, 20% missing for household income). To 'smooth' estimates across multiple small categories (e.g. single year age group; calendar year, small social groups), we used continuous variable specifications of age (centred at 60 years of age and linear splines with knots at ages 15, 24, 45 and 60) and calendar year (linear and quadratic terms). NZCMS data includes deaths up to age 77 (except for 2001-04 which includes all ages).

To allow for variations in mortality between subpopulations of interest, main effect and interaction coefficients were specified. For example, an interaction term was specified for each subpopulation with the spline function of age (as mortality rate ratios by ethnicity, smoking and income vary by age, often in a non-linear way[8]).

Due to missing deaths above age 77, the rate ratio (RR) between the groups of interest (e.g. Māori compared to non-Māori) was specified to decrease linearly to 1.0 at age 100 from that predicted at age 80. For example, if the estimated RR was 1.40 at age 80, then we set it at 1.38 at age 81, 1.36 at age 82, and so on. For two-way life-tables (e.g. ethnic by smoking groups) rate ratios for cross-classified subpopulations compared to the overall referent group (e.g. non-Māori never smokers) reduced linearly to the null.

Calendar year (census), and calendar year squared, were included as continuous variables in the regression models to allow for secular trends in mortality over time, and interactions with social group variables. Tertile groups of household income were calculated, separately for each five-year age group, for all census years pooled after CPI adjustment (see CancerTrends technical report for details[13]). Ethnicity was coded as Māori and non-Māori. We did not include Pacific ethnicity in the calculation of life-tables due to small numbers and the imprecision around the mortality estimates over time.

Previous research has shown no interaction of ethnicity and income for mortality on the relative scale on average across time[8]; therefore no interaction between income and ethnicity was included in the regression models.

Figure 1. Plot of rate ratio of mortality for Māori low income versus non-Māori high income for males, derived from regression modelling of NZCMS data
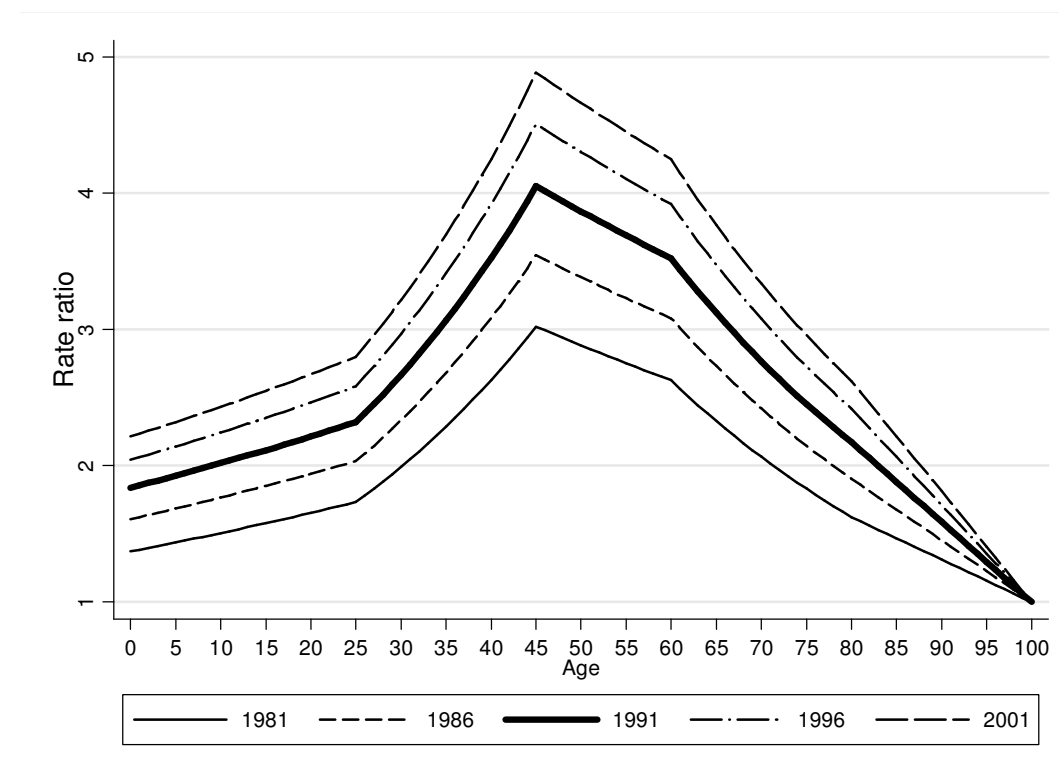
Figure 1 shows the estimated rate ratios across ages for the five census years for the ethnicity by income model (rate ratio of Māori low-income v non-Māori high-income). Figure 1 highlights the effect of the age knots and age spline in the rate ratios. The 1991 census is highlighted as the model was centred on this year. The figure also highlights the large inequalities for simultaneous ethnic by income stratification, with a predicted rate ratio of all-cause mortality between low-income Māori and high-income non-Māori of nearly five at 45 years of age in the 2001 Census.

For the smoking models the 1981 and 1996 Census data (pooled) were included, for people aged 0-74 on census night. Those with missing smoking data were excluded. Note that only those aged 15 and up were

included in the models of smokers. For smoking life-tables, mortality rates up to age 14 were assumed to be those of the sex by ethnic group (i.e. not stratified by smoking status).

Generation of subgroup specific life-tables—Having estimated the rate ratios by age, year and subpopulation group of interest using the NZCMS regression estimates, the m_x in the reference group was calculated using simple algebra. The three pieces of information, SNZ mortality rate ($m_{x,SNZ}$), proportion of the population (P_M), and mortality rate ratio (RR) described above (further index by M and nM for Māori and non-Māori, respectively) were brought together to estimate m_x.

For example, m_x non-Māori (reference):

$$m_{x,nM} = \frac{m_{x,SNZ}}{\left(P_{nM} * RR_{nM}(1) + P_M * RR_M\right)}$$

Therefore, estimating m_x in Māori is:

$$m_{x,M} = RR_m * m_{x,nM}$$

Finally, with estimated mx's for each subpopulation group and single year of age (x = 0 to 100), the whole life-table could be generated. The subsequent qx (probability of dying in the year) and px (probability of surviving another year) were back calculated using the above formulae. The cumulative survival (to age x) and life expectancy (from age zero) were then calculated as follows:

Cumulative survival = $\displaystyle\prod_0^x p_x$

Life Expectancy at birth = $e_0 = \dfrac{\sum_{i=0}^{100} L_i}{l_x}$, where l_x at age 0 is estimated to be 100,000.

Validation—The estimated ethnic-specific life-tables were compared to the official SNZ Māori and non-Māori life-tables for more recent years where numerator denominator bias is not problematic, and found to closely agree.

Comparison of ethnic-specific life expectancy to SNZ official life expectancy was also made. Our estimated life expectancy at age 0 for non-Māori was very similar to the SNZ estimates of life expectancy. However, the SNZ ethnic-specific life-tables do not take into account the undercounting of mortality in Māori and therefore SNZ Māori life expectancy in 1981 to 1996 censuses was overestimated.

Comparing life expectancy at birth for the 2001 census cohorts, the results for females were similar for Māori and non-Māori but for Māori males our calculated life expectancy was underestimated by about 0.8 years. This may be an artefact of the assumptions made in the modelling of mortality, i.e. the quadratic for calendar year may not have 'been enough' to fit the notable fall in Maori mortality between the 1996-99 and 2001-04 cohorts. We also compared our estimated life expectancy to adjusted ethnic-specific national life-tables,[14] and the overall results were similar.

Results

Figure 2a compares cumulative survival across year of age in the 1981 and 2001 censuses by ethnicity, for males and females. The further to the right the curve, the greater the life expectancy, which in turn is given by the area under the curve. Of note, the survival curve for non-Māori in 1981 is to the right of the survival curve for Māori in 2001, consistent with Māori life expectancy in 2001 not having caught up with where non-Māori life expectancy was in 1981. All survival curves in females are shifted further right towards older ages compared to males. Survival improved the most in non-Māori males over these two decades.

Figure 2a. Cumulative survival probability by ethnicity and sex, 1981 and 2001
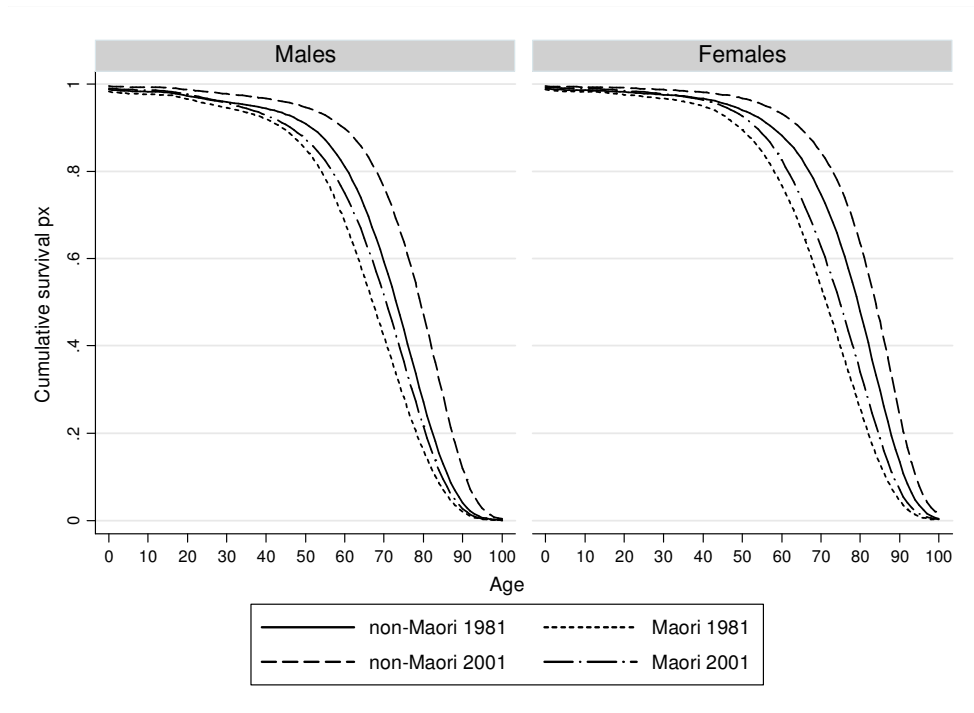
Figure 2b. Cumulative survival probability by income and sex, 1981 and 2001
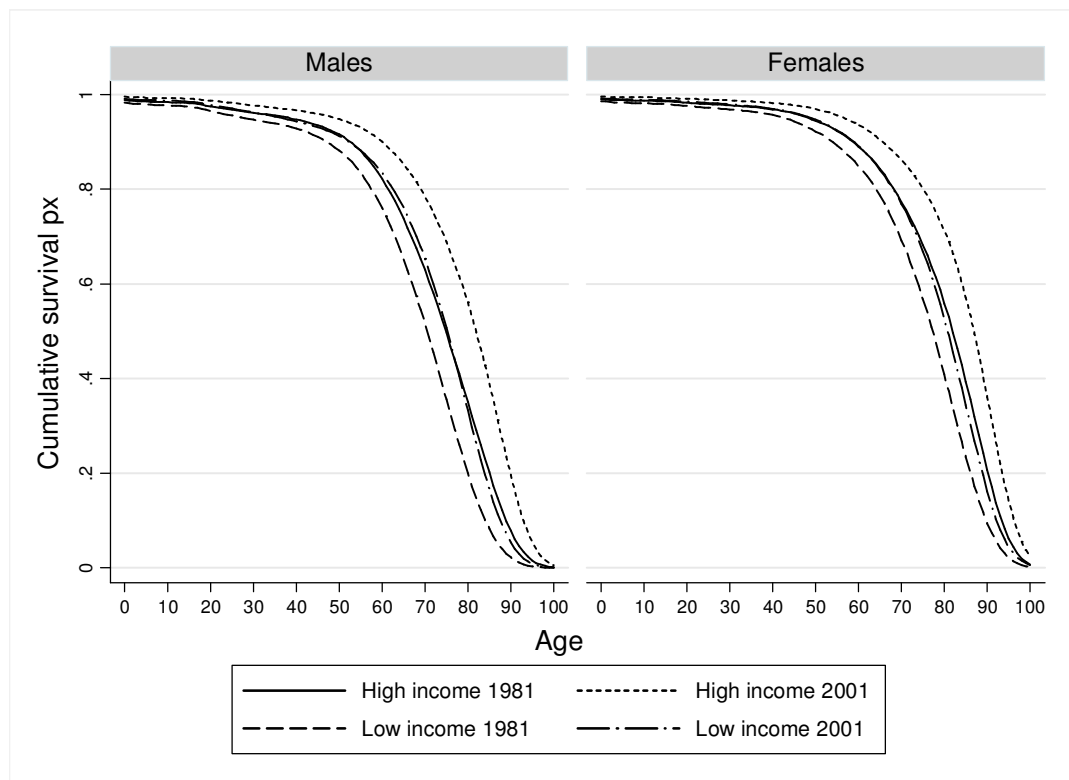
Figure 2b compares cumulative survival across year of age in the 1981 and 2001 censuses by low and high income tertiles (medium income not presented here), for males and females. There is a trend of better survival with increasing level of income in 1981 and 2001 and for males and females. Although survival at younger ages has improved in 2001, there seems to be a steeper decline in survival at older ages (> 75 years) in males in 2001. The survival of low income females in 2001 is less than survival of high income females in 1981, so it seems that female improvements in survival over time and within income groups were not as great as those for males.

Figure 3 presents cumulative survival for the ethnicity and income combinations in the 2001 census, for males and females. There are strong disparities in the survival curves between Māori and non-Māori for both males and females. The disparities between Māori and non-Māori within income groups appear to be greater in females compared to males. For males at older ages (85+ years) the survival of non-Māori low income is similar to that of the Māori high income group. When looking at survival across the income groups within ethnicity, the distance between low and high income survival curves is greater in Māori compared to non-Māori.

Figure 3. Cumulative survival probability for ethnicity by income and by sex, 2001.
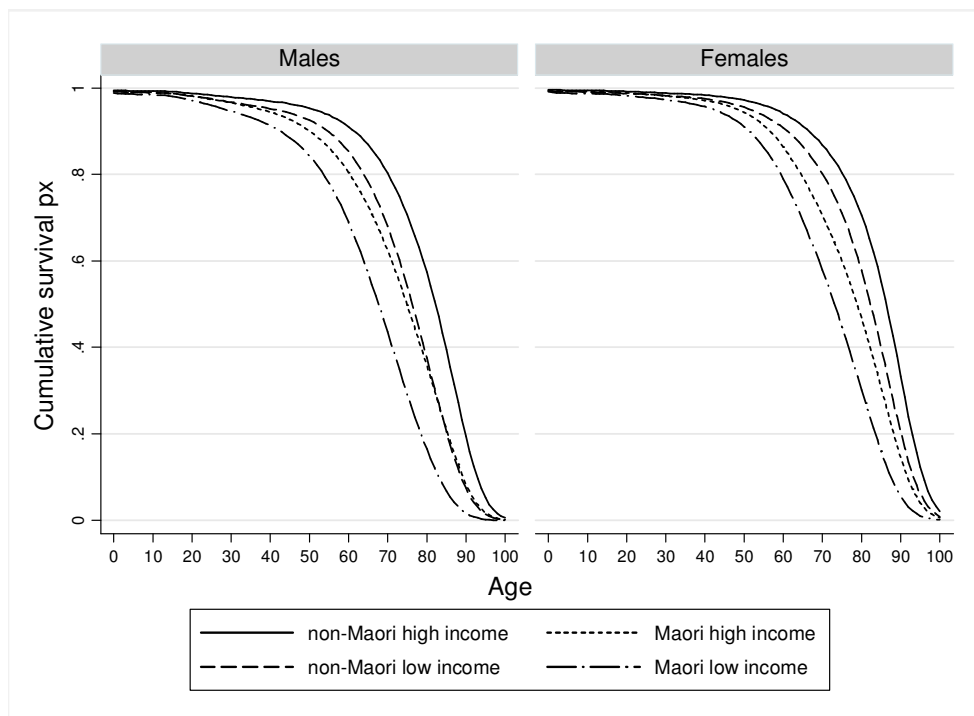
Table 1. Life expectancy at birth (age 0) by subpopulations for males and females by cohort

Variables		Male					Female				
		1981	**1986**	**1991**	**1996**	**2001**	**1981**	**1986**	**1991**	**1996**	**2001**
Ethnicity	Māori	65.4	64.5	65.1	66.4	68.2	69.7	69.4	70.4	71.4	73.0
	non-Māori	70.8	71.7	73.6	75.4	77.2	76.9	77.7	79.4	80.5	81.8
Income	Low	68.1	68.2	69.6	71.0	72.7	74.9	75.2	76.6	77.4	78.8
	Medium	70.5	71.0	72.6	74.2	76.1	76.7	77.2	78.7	79.6	81.0
	High	72.5	73.5	75.4	77.2	79.2	78.2	78.9	80.7	81.8	83.5
Smoking	Current	68.1			69.7		73.8			74.9	
	Never	73.2			77.3		77.8			81.6	
Income*Ethnicity											
Low	Māori	63.3	62.2	62.8	64.0	65.6	68.2	67.6	68.6	69.6	71.3
	non-Māori	68.8	69.1	70.7	72.3	74.0	75.6	76.0	77.5	78.5	79.8
Medium	Māori	65.9	65.2	66.1	67.5	69.5	70.3	70.0	71.1	72.1	73.9
	non-Māori	70.8	71.5	73.2	74.9	76.8	77.1	77.7	79.3	80.2	81.6
High	Māori	67.8	67.4	68.6	70.3	72.5	71.7	71.5	72.9	74.4	76.6
	non-Māori	72.8	73.8	75.8	77.7	79.7	78.6	79.3	81.0	82.3	83.9
Smoking*Ethnicity											
Current	Māori	63.9			63.8		68.8			69.5	
	non-Māori	68.6			70.9		74.3			76.0	
Never	Māori	65.6			68.1		69.7			73.4	
	non-Māori	73.8			78.3		78.2			82.2	
Smoking*Income											
Current	Low	65.8			66.5		72.2			72.6	
	Medium	68.1			69.7		73.8			74.9	
	High	70.3			72.7		75.4			77.3	
Never	Low	71.0			74.2		76.5			79.7	
	Medium	73.5			77.3		78.1			81.6	
	High	75.3			79.8		79.3			83.5	

Table 1 presents life expectancy at birth (age 0) for all main effect and interaction subpopulations of interest. There are improvements in life expectancy at age 0 for all subpopulations across the cohorts, from 1981 to 2001. When looking at trends over time the largest improvements in life expectancy were seen in non-Māori males and more generally for non-Māori compared to Māori (absolute increase 1981-2001 6.5 years male non-Māori, 2.8 years male Māori; 5.0 years female non-Māori, 3.3 years female Māori) and for the high income population compared to the low income population (absolute increase 6.7 years male high income, 4.6 years male low income; 5.3 years female high income, 3.9 years female low income). This led to the greatest improvements in survival in non-Māori high income males and females over time. The smallest improvement in life expectancy by ethnicity and income was in the Māori low income population (particularly males).

The estimated gap in life expectancy between Māori and non-Māori widened over the 20 years, with the absolute difference between ethnic groups in males increasing from 5.4 years in 1981 to 9.0 years in 2001.

The widening of the gap occurred mainly between 1981 and 1996 and stabilising in the following years. In females the gap was relatively stable over time. There were also differential increases in life expectancy across income groups leading to widening of the gap in life expectancy between then low and high income groups (absolute difference between high and low income was 4.4 years for males in 1981, and 6.5 in 2001; and 3.3 years for females in 1981, compared to 4.7 years in 2001). When looking at the differences in life expectancy by ethnicity and income the size of the gap between Māori and non-Māori is similar within each income group, but increasing over time.

The difference in life expectancy between non-Māori and Māori are greatest in the low income group for both males and females. The ethnic gap within income groups increases over time in males but appears to peak in 1991 for females. Disparities in life expectancy between the low and high income groups in Māori are at least 1 year greater than in non-Māori.

Figure 4 shows a plot of trends in life expectancy at age 0, for the ethnicity and income cross-classified subpopulations, across the five cohorts, for males and females. This shows the widening of the gap in life expectancy among the income groups for Māori and non-Māori over time. The estimated gap between low and high income groups, within ethnic groups was greatest in the 2001 census.

The gap in life expectancy in 1996 (the most recent smoking cohort) between current smokers and never smokers (ex-smoker data not presented here) is 7.6 years in males and 6.7 years in females. This is less than the gap in life expectancy between Māori and non-Māori. However, the difference in life expectancy in 1996 between current and never smokers is greater within non-Māori (7.4 years males, 6.2 years females) compared to within Māori (4.3 years males, 3.9 years females). Also the gap in life expectancy between Māori and non-Māori is greater within never smokers (10.2 years males, 8.8 years females) than within current smokers (7.2 years males, 6.5 years females). There have been minimal improvements in life expectancy between 1981 and 1996 in current smokers (less than 2 years).

Figure 4. Life expectancy at birth for Māori and non-Māori by high, medium and low income groups and by sex, 1981-2001.
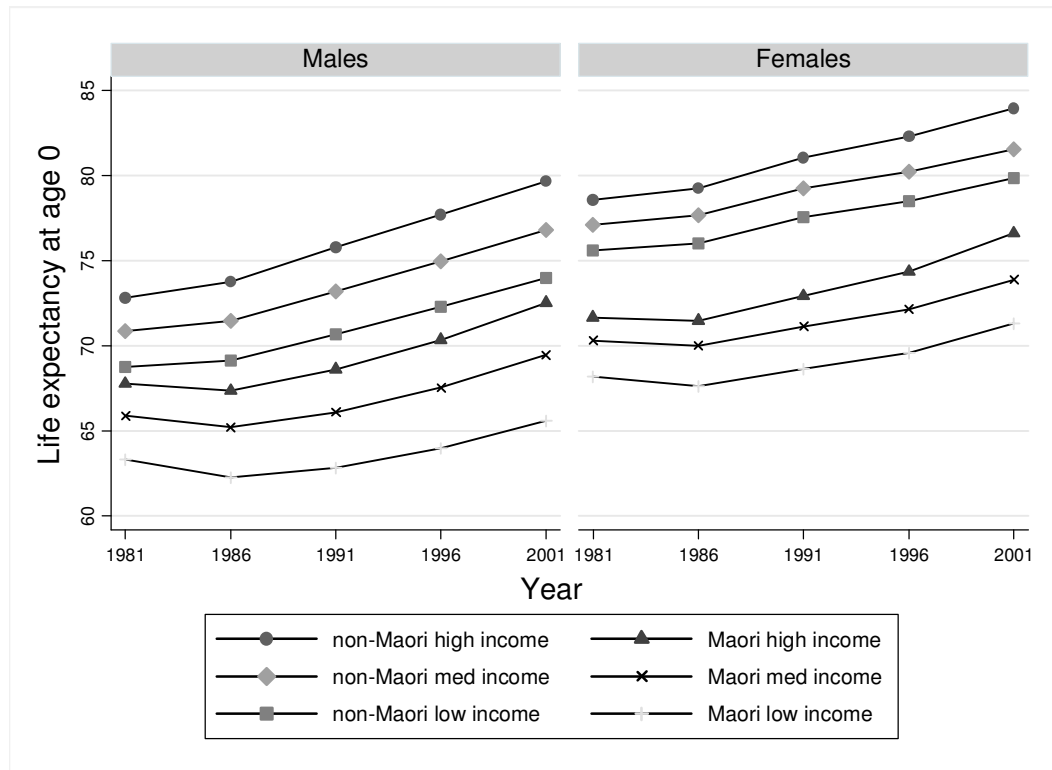
Discussion

The results from these life-tables are an important contribution for the understanding of "life" in New Zealand over the past 20 years, as well as to estimation of the future life expectancy of New Zealanders across important subpopulation groups. This study is unique in that it utilises mortality data linked to the New Zealand census, to enable examinations of life expectancy between important subpopulation groups by ethnicity, income and smoking status. This study adds to unique data created by the Ministry of Health and Statistics New Zealand breaking down life expectancy by ethnicity and area deprivation and rurality.[10, 11] We have identified widening of gaps in life expectancy between Māori and non-Māori, particularly in low income populations, and up to the turn of the century.

The development of these subpopulation life-tables provides an alternative set of data for policymakers and the public to monitor summary health measures. The production of subpopulation group life-tables also makes a significant contribution to the methods for estimating population-based cancer survival rates by subpopulation group over time.

Of particular note, and the motivating reason for us doing this work, cancer relative survival calculations that do not draw expected mortality from subpopulation life-

tables will give spurious estimates—especially in a society such as New Zealand where there are large ethnic inequalities in mortality. Further, the existence of smoking on New Zealand census data allows the estimation of smoking life-tables, and provides a significant methodological step forward for calculation of smoking-related cancer survival.

These life-tables, however, have their limitations. Caution is required in their use for year by year monitoring of health status. Indeed, some of the strengths of our data and methods (e.g. smoothing across the multiple censuses) can manifest as weaknesses for ethnic-specific life expectancy in a particular year when trends over time in mortality rates may not have been 'smooth' (be that smooth in linear and quadratic terms as included in our underlying regression analyses on NZCMS data). Also NZCMS data does not include deaths beyond the age of 77 prior to 2001, causing us to assume that mortality rate ratios tended to 1.0 above the age of 80. Thus, we recommend using 'official' SNZ ethnicity life-tables and life expectancy since 1996 if one specifically wants to monitor and report on recent ethnic life expectancy trends.

We do, however, provide life expectancy estimates combing ethnicity, income and smoking status that were hitherto unavailable in New Zealand. These show, perhaps, surprising results with greater gaps in life expectancy between current smokers compared to never smokers in non-Māori compared to Māori. The reason for the lesser life expectancy impact of smoking within Māori, compared to non-Māori, is twofold.[15-17]

First, the rate ratio of mortality comparing current to never smokers is less among Māori, due to the much higher background mortality of Māori never smokers compared to non-Māori never smokers (due to other causes of ethnic disparities in mortality). Second, and related, the survival curve is generally shifted to the left for Māori, to an age profile that is beneath that for a maximal impact of smoking on life expectancy. But, this will not necessarily be the case in the future; an accompanying paper estimates Māori and non-Māori life expectancy out to 2040, and suggests that the smoking impact on life expectancy will be more comparable between ethnic groups by 2040.

Finally, we have developed methods for life expectancy estimation that, to our knowledge, are novel—albeit driven and permitted by the particular strengths of New Zealand data. We encourage others, nationally and internationally, to critique our methods and results. The life-tables can be found at www.uow.otago.ac.nz/nzcms-info.html We encourage colleagues to use these life-tables for modelling and projections.

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References:

1. Siegel JS, Swanson DA, eds. The Methods and Materials of Demography. 2nd ed. California: Elsevier Academic Press; 2004.

2. Statistics New Zealand. New Zealand Life-tables: 2005-07. Wellington: Statistics New Zealand; 2009.

3. Dickman PW, Adami HO. Interpreting trends in cancer patient survival. . Journal of Internal Medicine. 2006;260:103-117.

4. Dickman PW, Coviello E, Hills M. Estimating and modelling relative survival. The Stata Journal. 2009;in press.

5. Sarfati D, Blakely T, Pearce N. Measuring cancer survival in populations: relative survival versus cancer-specific survival. Int J Epidemiol. 2010; 39(3):766-768.

6. Blakely T, Carter K, Wilson N, et al. If nobody smoked tobacco in New Zealand from 2020 onwards, what effect would this have on ethnic inequalities in life expectancy? N Z Med J. 2010;123(1320). http://www.nzma.org.nz/journal/123-1320/4264

7. Ajwani S, Blakely T, Robson B, et al. Unlocking the numerator-denominator bias III: adjustment ratios by ethnicity for 1981-1999 mortality data. The New Zealand Census-Mortality Study. NZ Med J. 2004;116(6).

8. Blakely T, Tobias M, Atkinson J, et al. Tracking disparity: trends in ethnic and socioeconomic inequalities in mortality, 1981-2004. Wellington: Ministry of Health; 2007.

9. Blakely TA, Robson B, Atkinson J,et al. Unlocking the numerator-denominator bias. I: Adjustments ratios by ethnicity for 1991-94 mortality data. The New Zealand Census-Mortality Study. NZ Med J. 2002;115(1147):39-43.

10. Ministry of Health. Life Expectancy and Small Area Deprivation in New Zealand. Wellington: Ministry of Health; 2001.

11. Tobias M, Yeh L-C. Life Expectancy in New Zealand 2005-07. Technical Note. Wellington: Ministry of Health; 2009.

12. Statistics New Zealand. New Zealand Life-tables: 2000-02. Wellington: Statistics New Zealand; 2004.

13. Atkinson J, Shaw C, Blakely T, et al. Linkage of Census and Cancer Registrations, 1981-2004: CancerTrends Technical Report No. 1. Wellington: Department of Public Health, University of Otago, Wellington; 2010.

14. Blakely TA, Tobias M, Robson B, et al. Widening ethnic mortality disparities in New Zealand, 1981-99. Soc Sci Med. 2005;61(10):2233-2251.

15. Blakely T, Fawcett J, Hunt D, Wilson N. What is the contribution of smoking and socioeconomic position to ethnic inequalities in mortality in New Zealand. Lancet. 2006;368(9529):44-52.

16. Hunt D, Blakely T, Woodward A, Wilson N. The smoking-mortality association varies over time and by ethnicity in New Zealand. Int. J. Epidemiol. 2005;34(5):1020-1028.

17. Wilson N, Blakely T, Tobias M. What potential has tobacco control for reducing health inequalities? The New Zealand situation. International Journal for Equity in Health. 2006;5:14-11.

Appendix 1: Life-table terminology

x Exact age (e.g. exact age 5 corresponds to 5 years and 0 days).

L_x Average number of persons alive in the age interval x to $x + 1$.

l_x Number of persons alive at exact age x.

d_x Number of deaths in the age interval x to $x + 1$.

q_x Probability that a person at exact age x dies within a year.

p_x Probability that a person at exact age x lives another year.

m_x Central death rate for population in the age group x to $x + 1$.

e_x Expected number of years of life remaining at age x.

The mortality rate (m_x) is calculated as the number of deaths in each age interval divided by the person time lived within the interval:

$$m_x = d_x/L_x$$

These rates can be converted into probabilities using the linear model.[1] The probability of dying within the next year at age x is:

$$q_x = m_x/(1+0.5m_x) = 2m_x/(2+m_x)$$

This formula is based on the assumption that deaths between ages x and $x+1$ occur, on average, at age $x+0.5$. Deaths at age x in a given year are uniformly distributed by age and time interval.[1] The probability of surviving another year is:

$$p_x = 1-q_x$$

If nobody smoked tobacco in New Zealand from 2020 onwards, what effect would this have on ethnic inequalities in life expectancy?

Tony Blakely, Kristie Carter, Nick Wilson, Richard Edwards, Alistair Woodward, George Thomson, Diana Sarfati

Abstract

Background Smoking contributes to the 7 to 8 year gap between Māori and non-Māori life expectancy (2006 Census). To inform current discussions by policy-makers on tobacco control, we estimate life-expectancy in 2040 for Māori and non-Māori, never-smokers and current-smokers. If nobody smoked tobacco from 2020 onwards, then life expectancy in 2040 will be approximated by projected never-smoker life expectancy.

Method Life-tables by sex/ethnicity/smoking status for 1996–99 were estimated by merging official Statistics New Zealand life-tables, census data and linked census-mortality rate estimates. We specified six modelling scenarios, formed by combining two options for future per annum declines in mortality rates among never-smokers (1.5%/2.5% and 2.0%/3.5% for non-Māori/Māori; i.e. assuming a return to long-run trends of closing ethnic gaps as in pre-1980s decades), and three options for future per annum reductions in the mortality rate difference comparing current to never-smokers (0%, 1% and 2%).

Results In 1996–1999, current smokers had an estimated 3.9 to 7.4 years less of life expectancy relative to never-smokers. This smoking difference in life expectancy was less among Māori than among non-Māori.

If the 2006 census smoking prevalence remains unchanged into the future, we estimate the difference in 2040 between Māori and non-Māori life expectancy will range from 1.8 to 6.1 years across the six scenarios and two sexes (average 3.8). If nobody smokes tobacco from 2020 onwards, we estimate additional gains in life expectancy for Māori ranging from 2.5 to 7.9 years (average 4.7) and for non-Māori ranging from 1.2 to 5.4 years (average 2.9). Going smokefree as a nation by 2020, compared to no change from the 2006 Census population smoking prevalence, will close ethnic inequalities in life expectancy by 0.3 to 4.6 years (average 1.8 years; consistently greater for females).

Discussion If smoking persists at current rates it will become an even greater constraint on life expectancy improvements for New Zealanders in the future. Continued increases in life expectancy, and closing of the Māori:non-Māori gaps in life expectancy, would be greatly assisted by the end of tobacco smoking in Aotearoa-New Zealand by 2020.

The public health case for concern and action on tobacco use in Aotearoa-New Zealand is overwhelming; 4500 to 5000 deaths per year in New Zealand can be attributed to tobacco use.[1]

The long-run trends in life expectancy show continual improvement in non-Māori life expectancy and a substantial increase in Māori life expectancy since the turn of the 19th Century.[2-4] Over the last century, average annual reductions in mortality have been approximately 3.5% per annum for Māori and 2.0% per annum for non-Māori (Woodward and Blakely, work in progress). Such reductions do not occur by chance, but reflect concerted policy and public health efforts, in addition to general improvements in health services and standard of living. Furthermore, closing of ethnic inequalities in life expectancy are far from guaranteed, as evidenced by a widening of Māori:non-Māori life expectancy gaps in the 1980s and 1990s associated with structural changes in New Zealand society.[3,5–7]

The gap in life expectancy between Māori and non-Māori c.2006 remains large at 7 to 8 years, but has narrowed from the 9 to 10 year gap c.1996.[3,4,7] Such a pattern suggests the possibility of a return to long-run trends of closing ethnic gaps in mortality in New Zealand.

One of the greatest obstacles to extending the long-run improvements in life expectancy into the future is tobacco use. We have previously quantified the impact of smoking on ethnic and socioeconomic inequalities in New Zealand during the 1990s.[8–11] A Tupeka Kore Vision has been developed which seeks the end of tobacco use in New Zealand by 2020.[12]

This paper asks the question: if New Zealand did end tobacco smoking by 2020, what would the effect be on life expectancy by 2040, and in particular the gap between Māori and non-Māori?

To attempt to answer this question, we present estimates of life expectancy for Māori and non-Māori, and current- and never-smokers, in 1996–99 and then project these life expectancy estimates out to 2040. Assuming a substantive upgrade in tobacco control such that smoking prevalence is negligible by 2020, and allowing a 20 year wash-out period for the majority of tobacco's effect on excess mortality, we assume that life expectancy in 2040 will be that projected for never-smokers.

Methods

In an accompanying paper in this issue of the *Journal,*[7] we present life-tables and life expectancies for multiple combinations of time, ethnic group, income tertiles and smoking status, using mortality rates from linked census-mortality data.[7] Here we use life-tables and life expectancy by sex/smoking/ethnicity, estimates of future mortality decline among never-smokers, and estimates of future rate differences for current- versus never-smokers (and ex- compared to never-smokers in one analysis), to estimate life expectancy in 2040.

Life-tables and life expectancy

Briefly, the method used to create the life-tables brings together three pieces of information:

- The official Statistics New Zealand life-tables by year and sex;
- The distribution of the total New Zealand population by the variables of interest (e.g. the proportion who smoke and ethnicity); and
- Estimates of the differences in subpopulation mortality rates (from the New Zealand Census-Mortality Study [NZCMS]).

These three inputs were combined to produce mortality rates by single year of age for each subpopulation, and complete life-tables including central death rates (m_x) and probabilities of death (q_x), calculated for each age $(x: 0-100)$, which were then used to derive other life-table functions such as life expectancy for each subpopulation. The life-tables used in this paper were generated for males

and females by ethnicity (Māori and non-Māori), and smoking status (never-, current- and ex-smoker), for the 1996-99 census-mortality cohort. For smoking life-tables, mortality rates up to age 14 were assumed to be those of the sex by ethnic group (i.e. not stratified by smoking status). (The 2006 Census, which also includes a smoking variable, is not yet linked to mortality data.)

Projections

We focus first on projections for never- and current- smokers. The method of estimation to 2040 involved three steps:

- Estimating mortality rates by single year of age for never-smokers (by sex and ethnic group) in 2040;
- Estimating mortality rate differences (by single year of age) between current- and never-smokers in 2040, and adding this to the never-smoker mortality rates to get the smoker mortality rates; and
- Deriving life-tables and life expectancy in 2040 using these estimated mortality rates (m_x) to calculate mortality risk (q_x) and remaining life-table parameters.

These steps are outlined in more detail below.

Step 1—We set an initial estimate of the annual percentage reduction in never-smoker mortality rates m_x of 2.5% for Māori and 1.5% for non-Māori. These figures are consistent with Statistics New Zealand projections for low, medium and high mortality scenarios of 2.1%, 1.6% and 1.0% percent per year reductions in mortality rates for males (ethnic groups combined), and 2.4%, 1.8% and 1.3% percent per year reductions for females (http://www.stats.govt.nz/methods_and_services/TableBuilder/population-projections-tables.aspx).

We modified these estimates to allow for ethnic variation in mortality rate reductions (the long-run trends suggest mortality rates are falling faster for Māori), and assumed that mortality reductions in the future will be the same for males and females. Extrapolating to 2040, we multiplied the non-Māori m_x in 1996, for every single year of age, by $0.985^{44} = 0.514$, where 0.985 is one minus the annual percentage reduction of 1.5% and 44 is 2040 minus 1996. The Māori m_x was multiplied by $0.975^{44} = 0.328$.

We also projected more optimistic annual reductions in mortality rates of 3.5% per annum for Māori and 2.0% per annum for non-Māori using the long-run trends of improving life expectancy over the last 100 years.

Step 2—We have previously found that the mortality rate difference (not the rate ratio) comparing smokers to never-smokers, is consistent across time and ethnic group.[11] Therefore, we set one option for the smoking:non-smoking mortality rate differences in 2040 as being the same as that observed in 1996-99. However, constant rate differences over time mean increasing rate ratios if the mortality rate among never-smokers is reducing.

For example, the rate ratio comparing current- to never-smokers *within* Māori is roughly 1.5 in 1996-99. Under the assumption of 2.5% per annum reduction in Māori never-smoker mortality rates to 2040, a constant rate difference would see the rate ratio increase to 1+ (1.5-1)/0.328 = 2.5. We also explored the effects of alternative assumptions of 1% and 2% per annum reductions in the rate difference, applied similarly within sex by ethnic groups. Under the 2% per annum reduction in the rate difference (and the 2.5% reduction in Māori never-smoker mortality rates), the rate ratio of 1.5 in 1996 would be about 1.6 in 2040.

Step 3—With mortality rates by single year of age for all sex/ethnicity/smoking status groups, life-tables were easily calculated for 2040. Because of the relatively simple nature of our projections and the differential mortality rate declines by ethnic group, it was possible for estimated Māori never-smoker mortality rates to be less than non-Māori never-smoker mortality rates in 2040 for some single years of age, and likewise for estimates of Māori current-smoker mortality rates to be less than non-Māori current- smoker rates in 2040. It is possible that Māori mortality will fall below that of non-Māori at some time in the future, if the long-run trend since 1900 continues. However, for the purposes of this analysis, we assumed no more than convergence and therefore, where necessary, forced the Māori rate to equal the projected non-Māori rate in 2040.

Ex-smokers—The ex-smoker compared to never-smoker mortality rate differences and rate ratios obtained from the NZCMS should be treated with caution. This is because we do not have data on the

time since quitting, which makes ex-smoker mortality experience in 1996-99 difficult to interpret and potentially unreliable as the basis for future projections. Nevertheless, to provide a point of comparison, we also calculated life expectancy in 2040 for sex by ethnic groups assuming the 2006 census distribution of smoking behaviour (never-, current-, and ex-smoker). A parallel method to that described above for current-smoker mortality rate projections was used for ex-smokers.

Sensitivity analyses

In addition to varying the percentage annual decline in never-smoker mortality rates, and percentage decline in the smoking:non-smokiing mortality rate difference, we tested two other assumptions. First, life expectancy in the future will be influenced by mortality over the age of 100, as the proportion of centenarians in the population increases.

Thus, we extended the life-tables out to age 120 by simply assuming that the mortality rate increased by 6% per year of age for every year of age over 100, where 6% is approximately the change in mortality by year of age from 90 to 100. (The methods and life-tables in the accompanying paper in this *Journal*[7] apply to age 100, the top end of current 'official' Statistics New Zealand life-tables.) Altering this 6% percent increase down to 4% and up to 10% had only a negligible influence of the results presented in this paper, and is therefore not discussed further here.

Second, we had to estimate mortality rate ratios for smokers compared to never-smokers beyond the age of 80 in the accompanying paper,[7] because the 1996-99 census-mortality cohort only included deaths up to age 77. Our assumption was that the predicted mortality rate ratio for current-smokers compared to never-smokers at age 80 reduced linearly to 1.0 by age 100. For the estimates to 2040 in this paper, we investigated setting a minimum rate ratio (and hence rate difference) at all ages – essentially ages above 80 years. Setting such a minimum at 1.2, or even 1.5, had negligible impact on the estimations in this paper, so is not presented further.

A copy of the Microsoft Excel spreadsheet used to generate all estimates in this current paper is provided at the NZCMS website (www.uow.otago.ac.nz/nzcms-info.html), and allows interested users to alter any of the input assumptions specified above.

Results

Life epectancy 1996–9

Figure 1 shows estimates of life expectancy in 1996–99, the most recent cohort for which we have smoking data. The estimates are reproduced in Table 1, with the addition of gaps in years of life expectancy between Māori and non-Māori within smoking status groups, and conversely gaps in life expectancy between smoking status groups within Māori and non-Māori populations.

Table 1. Life expectancy estimates for 1996-99 (observed)

1996–99 (i.e. Figure 1)		Life expectancy by smoking status			Smoking gap	
		Never	Current	Total	Never: Current	Never: Total
Males	Māori	68.1	63.8	66.4	4.3	1.7
	Non-Māori	78.3	70.9	75.4	7.4	2.9
	Ethnic gap	10.2	7.2	9.0		
Females	Māori	73.4	69.5	71.4	3.9	2.0
	Non-Māori	82.2	76.0	80.5	6.2	1.7
	Ethnic gap	8.8	6.5	9.1		

Figure 1. Life expectancy in years for 1996-99, by sex, ethnicity and smoking status
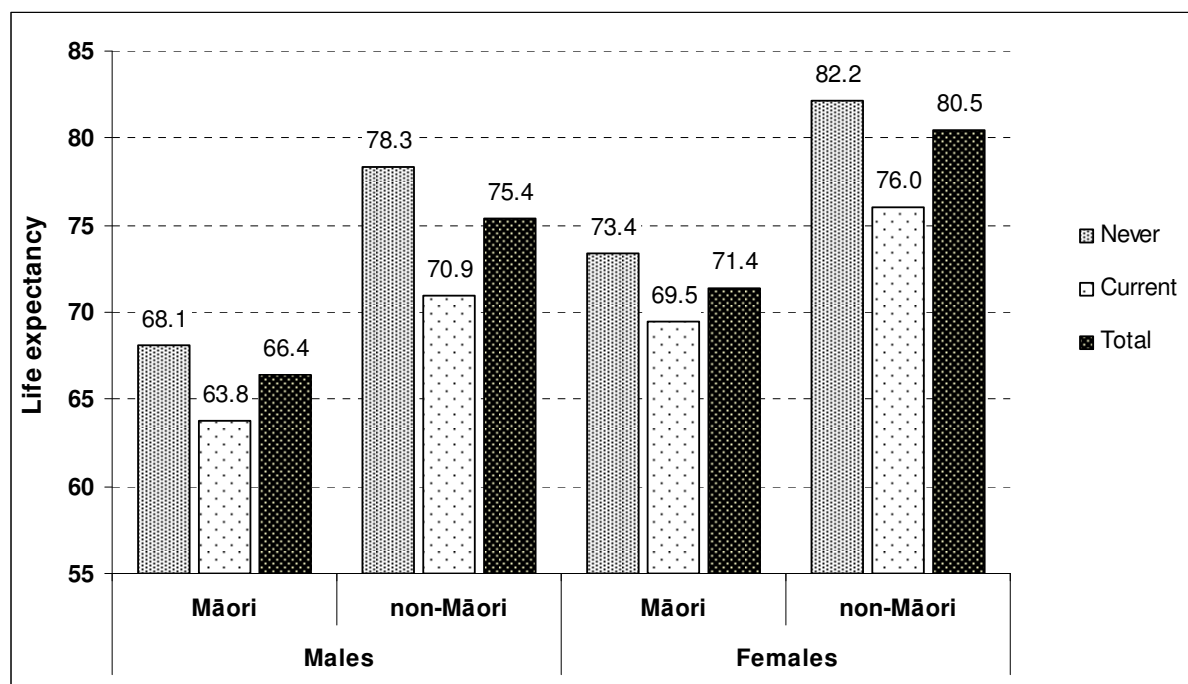
Two patterns are evident. First, current-smokers have lower life expectancy among males and females for Māori and non-Māori strata. However, the gap between current- and never-smokers is less among Māori than among non-Māori (4.3 and 3.9 years among Māori males and females respectively, compared to 7.4 and 6.2 years among non-Māori).

Second, Māori have lower life expectancy in all smoking strata. However, the gap between Māori and non-Māori is greater among never-smokers than among current-smokers (10.2 and 8.8 years for male and female never-smokers respectively, compared to 7.2 and 6.5 years for current-smokers).

Projections to 2040

Table 2 shows our projected 2040 life expectancy estimates for never- and current-smokers for the six scenarios. Assuming New Zealand is smokefree by 2020, and allowing for a wash-out period of 20 years for past smoking-related mortality risk, then we might assume that 2040 life expectancies are approximated by the never-smokers.

Table 2. Projected life expectancy estimates for 2040 for six scenarios of varying reductions in never smoker mortality rates, and reductions in current-never smoker mortality rate differences

Scenario		Smoking status			Smoking gap		Extent of ethnic
		Never	**Current**	**Total (2006 †)**	**Never: Current**	**Never: Total**	**gap closure‡**
		(i)	*(ii)*	*(iii)*	*(iv)*	*(v)*	*(vi)*
A: 2.5%/1.5% p.a. ↓ Māori/non-Māori never smoking mortality; 0% p.a. ↓in smoking rate difference							
Males	Māori	82.3	71.6	77.1	10.7	5.2	
	non-Māori	85.4	74.1	80.8	11.3	4.6	
	Ethnic gap	3.1	2.5	3.8			0.7
Females	Māori	85.9	76.4	79.6	9.5	6.3	
	non-Māori	88.6	79.1	85.8	9.5	2.9	
	Ethnic gap	2.7	2.7	6.1			3.5
B: 2.5%/1.5% p.a. ↓ Māori/non-Māori never smoking mortality; 1% p.a. ↓in smoking rate difference							
Males	Māori	82.3	74.4	78.7	7.9	3.6	
	non-Māori	85.4	77.0	82.3	8.4	3.2	
	Ethnic gap	3.1	2.6	3.6			0.5
Females	Māori	85.9	78.9	81.5	7.0	4.4	
	non-Māori	88.6	81.7	86.7	6.9	1.9	
	Ethnic gap	2.7	2.8	5.2			2.5
C: 2.5%/1.5% p.a. ↓ Māori/non-Māori never smoking mortality; 2% p.a. ↓in smoking rate difference							
Males	Māori	82.3	76.6	79.8	5.7	2.5	
	non-Māori	85.4	79.4	83.3	6.0	2.1	
	Ethnic gap	3.1	2.8	3.4			0.3
Females	Māori	85.9	81.0	83.0	5.0	3.0	
	non-Māori	88.6	83.7	87.4	4.9	1.2	
	Ethnic gap	2.7	2.8	4.4			1.7
D: 3.5%/2.5% p.a. ↓ Māori/non-Māori never smoking mortality; 0% p.a. ↓in smoking rate difference							
Males	Māori	86.6	73.3	79.9	13.2	6.7	
	Non-Māori	87.7	74.9	82.3	12.8	5.4	
	Ethnic gap	1.2	1.6	2.5			1.3
Females	Māori	89.8	78.0	81.9	11.8	7.9	
	Non-Māori	90.8	80.0	87.4	10.8	3.3	
	Ethnic gap	1.0	2.0	5.5			4.6
E: 3.5%/2.5% p.a. ↓ Māori/non-Māori never smoking mortality; 1% p.a. ↓in smoking rate difference							
Males	Māori	86.6	76.6	81.9	10.0	4.7	
	Non-Māori	87.7	78.1	84.0	9.7	3.8	
	Ethnic gap	1.2	1.5	2.1			0.9
Females	Māori	89.8	81.0	84.3	8.8	5.5	

	Non-Māori	90.8	82.8	88.5	8.0	2.2	
	Ethnic gap	1.0	1.8	4.3			3.3

F: 3.5%/2.5% p.a. ↓ Māori/non-Māori never smoking mortality; 2% p.a. ↓in smoking rate difference

Males	Māori	86.6	79.3	83.4	7.2	3.2	
	Non-Māori	87.7	80.7	85.2	7.0	2.5	
	Ethnic gap	1.2	1.4	1.8			0.6
Females	Māori	89.8	83.5	86.0	6.3	3.8	
	non-Māori	90.8	85.1	89.3	5.6	1.4	
	Ethnic gap	1.0	1.6	3.3			2.3

† The total estimates are assuming the 2006 census population distribution of smoking continued to exist to 2040.

‡ The 'extent of ethnic gap closure' is the [never smoker ethnic gap] minus [total (as per 2006 census smoking distribution) ethnic gap]. That is, the reduction in the ethnic life expectancy gap resulting from ending tobacco smoking by 2020 c.f. alternative of 2006 census population smoking prevalences continuing.

For all scenarios, and all strata of ethnicity by smoking, there are substantial improvements in life expectancy compared to 1996-99. Regardless of the scenario, ethnic gaps within strata of smoking are also reduced. Gaps in life expectancy between current- and never-smokers (column iv, Table 2) range from 5 to 6 years in Scenario C (2.5/1.5% annual reduction in Māori/non-Māori never-smoker mortality; 2% per annum reduction in mortality rate difference between current- and never-smokers) up to 11 to 13 years in Scenario D (3.5/2.5% annual reduction in Māori/non-Māori never-smoker mortality; 0% per annum reduction in mortality rate difference between current- and never-smokers). Regardless of the scenario, the impact of smoking on life expectancy is more similar for Māori and non-Māori than that observed in 1996-99. '

If the 2006 census smoking prevalence remains unchanged into the future (i.e. 'total' in Table 2), we estimate the difference in 2040 between Māori and non-Māori life expectancy to range from 1.8 to 6.1 years across the six scenarios and two sexes (average 3.8 years; 'ethnic gap' estimates in column (iii)).

By comparing the life expectancy of 'never-smokers' and 'total' across scenarios A to F, we have estimates of the additional gains in projected life expectancy in 2040 if nobody smoked tobacco from 2020 compared to the 2006 smoking distribution continuing indefinitely (i.e. column (v) in Table 2). Accordingly, we estimate additional gains in life expectancy for Māori ranging from 2.5 to 7.9 years (average 4.7) and for non-Māori ranging from 1.2 to 5.4 years (average 2.9). That is, going smokefree as a nation will (we estimate) result in larger improvements in Māori life expectancy, compared to non-Māori, and therefore result in a closing in ethnic inequalities in life expectancy ranging from 0.3 to 4.6 years (average 1.8 years).

The estimated closing of ethnic gaps in life expectancy was consistently greater for females, reflecting the particularly high 2006 smoking prevalence among Māori females.

Discussion

If the 2006 census smoking prevalence remains unchanged into the future, and background non-smoking related mortality continues to decrease more so for Māori

than non-Māori, we estimate that the difference in 2040 between Māori and non-Māori life expectancy will be about three and a half years (averaged across sex). However, if nobody smokes tobacco from 2020 onwards, we estimate *about* 5 years of additional gain in life expectancy for Māori (range across six scenarios and sexes 2.5 to 7.9 years) and *about* 3 years for non-Māori (range 1.2 to 5.4 years), therefore contributing *about* 2 years (range 0.3 to 4.6 years) of closing in ethnic inequalities in life expectancy.

We emphasise that our exact estimates are quantitatively uncertain. But we do conclude that if nobody smokes tobacco by 2020 in New Zealand there will be substantive improvements in overall population life expectancy. And it will be an important, if not necessary, step towards the ending of Māori:non-Māori inequalities in mortality by 2040—200 years after the signing of the Treaty of Waitangi.

A number of subsidiary findings also arise out of our projections. Perhaps surprisingly at first glance, we find that the gap between current- and never-smoker life expectancy in 1996-99 was *less* among Māori (4.3 and 3.9 for males and females respectively) than among non-Māori (7.4 and 6.2). The reason for this is that the absolute additional mortality burden from smoking (i.e. the rate difference in mortality between current- smokers and never-smokers) is about the same across ethnic groups, sexes and time.[8]

The corollary of this is that the relative risk comparing the mortality of smokers and never-smokers is *less* among Māori. This is due to the much higher background mortality among Māori never-smokers compared to non-Māori never-smokers, which in turn is due to all the other non-tobacco determinants of mortality that vary between Māori and non-Māori.[9,11] However, if as we assumed in this paper, Māori mortality rates fall faster than non-Māori mortality rates in the next few decades (i.e. if mortality rate reductions return to their long-run trends of the last century, as opposed to a reversed pattern in the 1980s and 1990s[3,4,13] associated with structural changes in the economy), then the relative impact of smoking on mortality among Māori will increase faster than among non-Māori. That is, the Māori life-table will move into a state where smoking has a larger impact on life expectancy gains than it does now.

Thus, by 2040 we estimate that the gap in life expectancy between current-smokers (hypothetical if the country is free of tobacco smoking by then) and never-smokers will be similar for both Māori and non-Māori (Table 2).

There are a number of limitations in our analyses. First and foremost, we necessarily make a number of assumptions to project life expectancy in 2040. Therefore, our projections must be interpreted as indicative only. For example, if it is assumed that mortality cannot continue to decline at the rates it has in the last 100 years, and that our 2.0%/3.5% per annum reductions in non-Māori/Māori never smoker mortality are too optimistic (partly because some of the rapid fall in mortality rates may be due to smoking cessation itself), then scenarios A, B and C would be assumed to be more accurate.

As another example, we think that a 2% per annum decline in the smoking:non-smoking mortality rate difference into the future is unlikely, and that 0% or 1% is more likely. Accordingly, we have presented six scenarios in this paper for readers to peruse, and we have provided a copy of our Excel spreadsheet at the NZCMS website

to allow testing of alternative assumptions (www.uow.otago.ac.nz/nzcms-info.html). We have also undertaken sensitivity analyses varying assumptions of the decaying smoking:non-smoking mortaltiy rate ratio above age 80 and future mortality rate decline among never smokers above age 100 – neither greatly affected the results presented here.

Finally, and importantly, the general conclusion remains unaltered across the Scenarios we specified: smoking will have a bigger impact on life expectancy in the future; and if Māori life expectancy converges with non-Māori life expectancy then the impact of smoking will also become more similar between the two ethnic groups.

Second there are limitations in the underlying NZCMS data, including likely misclassification biases of smoking status and incomplete linkage of mortality records. Regarding the former, this has probably led to some underestimation of difference in mortality by smoking status, therefore underestimating differences in life expectancy between never- and current-smokers. Regarding the latter, we used linkage weights that have been shown elsewhere to correct reasonably well for linkage bias.[14]

Third, we have not explicitly allowed for passive smoking. This currently affects more Māori than non-Māori. As a result, we have probably further underestimated the full impact of smoking on ethnic gaps in mortality in 2040 if current smoking prevalence continues, and will have underestimated the overall gains in life expectancy and the impact on reducing ethnic gaps in life expectancy of ending smoking by 2020 .

Fourth, our estimates of the association of smoking with mortality will be prone to residual confounding. That is, smoking is associated with other risk factors that positively confound the smoking-mortality association. Indeed, we have shown this previously using NZCMS data for socioeconomic position as the potential confounder.[15] There is likely to be some off-setting between such residual confounding and the misclassification of smoking and non-inclusion of passive smoking, however it is difficult to know the net effect.

Policy implications

What does this mean for policy making around tobacco control and reducing health inequalities? Simply, if tobacco is consumed in the future as it is currently, this will act as an increasingly important "handbrake" on overall improvements in life expectancy, and will also impede the closing of ethnic gaps in life expectancy. Ending tobacco smoking by 2020 will not only release greater life expectancy gains for the total New Zealand population, but also accelerate the closure of Māori:non-Māori gaps in life expectancy.

Is ending tobacco smoking in New Zealand by 2020 feasible? We argue "yes", given the availability of a range of existing and plausible interventions. In addition to intensifying established tobacco control interventions (e.g. tax rises as just announced in New Zealand, plain packaging as just announced in Australia, removing residual marketing such as point-of-sale displays, and enhanced cessation services), new 'endgame' solutions are available. For example, a reducing quota mechanism, or sinking lid [16], of 10 percentage points per annum reduction in tobacco imports from

2010 to 2020 would ensure that no tobacco is available for direct retail to the public by 2020.

Alternatively, large recurrent tax rises of about 20% per annum (accompanied by boosted and strong cessation services) may be able to drive prevalence down to less than 2% by 2020. Such end-game solutions have been voiced by both non-governmental organisations in New Zealand[12] and political leaders[17], and are receiving serious scrutiny by the current Māori Affairs Select Committee Inquiry (due to report in the next few weeks). Such solutions may even be favoured by New Zealand smokers since a majority of them regret smoking [18], and support stronger regulation of tobacco.[19 20]

Conclusions

As mortality rates decrease in the future, the relative impact of smoking on mortality will increase. If nobody smoked tobacco in New Zealand from 2020 life expectancy will be substantially improved for all, and our average estimate is that ethnic inequalities in life expectancy will be about two years less (compared to a scenario of 2006 smoking prevalence continuing into the future). Making New Zealand smokefree by 2020 is achievable – but strong political will is needed, along with improved recognition of the desire of smokers to be free of their addiction.

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References:

1. Ministry of Health. Tobacco Trends 2008: A brief update of tobacco use in New Zealand. Wellington: Ministry of Health, 2009.
2. Pool I. Te iwi Maori: A New Zealand population past, present, and projected. Auckland: Auckland University Press, 1991.

3. Blakely T, Tobias M, Robson B, et al. Widening ethnic mortality disparities in New Zealand 1981-99. Soc Sci Med 2005;61(10):2233-2251.

4. Tobias M, Blakely T, Matheson D, et al. Changing trends in indigenous inequalities in mortality: lessons from New Zealand. Int. J. Epidemiol. 2009;38(6):1711-1722.

5. Statistics New Zealand. New Zealand Life Tables: 2005–07. Wellington: Statistics New Zealand, 2009.

6. Blakely T. "Social injustice is killing people on a grand scale". N Z Med J 2008;121(1281):7-11. http://www.nzmj.com/journal/121-1281/3235/content.pdf

7. Carter K, Blakely T, Soeberg M. Trends in survival and life expectancy by ethnicity, income and smoking: 1980s to 2000s. N Z Med J. 2010;123(1320). http://www.nzma.org.nz/journal/123-1320/4263

8. Blakely T, Fawcett J, Hunt D, Wilson N. What is the contribution of smoking and socioeconomic position to ethnic inequalities in mortality in New Zealand? Lancet 2006;368(9529):44-52.

9. Wilson N, Blakely T, Tobias M. What potential has tobacco control for reducing health inequalities? The New Zealand situation. Int J Equity Health 2006;5(1):14.

10. Blakely T, Wilson N. The contribution of smoking to inequalities in mortality by education varies over time and by sex: two national cohort studies, 1981-84 and 1996-99. Int. J. Epidemiol. 2005;34(5):1054-1062.

11. Hunt D, Blakely T, Woodward A, Wilson N. The smoking-mortality association varies over time and by ethnicity in New Zealand. Int. J. Epidemiol. 2005;34:1020-1028.

12. The Smokefree Coalition. Tupeka kore/Tobacco Free Aotearoa/New Zealand by 2020 (sfc.org.nz/pdfs/TheVision2020.pdf): The Smokefree Coalition.

13. Blakely T, Tobias M, Atkinson J, et al. Tracking Disparity: Trends in ethnic and socioeconomic inequalities in mortality, 1981-2004. Wellington: Ministry of Health, 2007.

14. Fawcett J, Blakely T, Atkinson J. Weighting the 81, 86, 91 & 96 census-mortality cohorts to adjust for linkage bias. NZCMS Technical Report No. 5. ISBN 0-473-09112-7 (Also at http://www.wnmeds.ac.nz/nzcms-info.html). Wellington: Department of Public Health, Wellington School of Medicine and Health Sciences, University of Otago, 2002:92.

15. Blakely T, Hunt D, Woodward A. Confounding by socioeconomic position remains after adjusting for neighbourhood deprivation: an example using smoking and mortality. *J* Epidemiol Community Health 2004;58(12):1030-1031.

16. Laugesen M. Snuffing out cigarette sales and the smoking deaths epidemic. N Z Med J 2007;120:U2587. http://www.nzmj.com/journal/120-1256/2587/content.pdf

17. Gifford H, Bradbrook S. Recent actions by Māori politicians and health advocates for a tobacco-free Aotearoa/New Zealand: A brief review. Wellington: Whakauae Research Services; Te Reo Mārama; Health Promotion and Public Health Policy Research Unit (HePPRU), 2009.

18. Wilson N, Edwards R, Weerasekera D. High levels of smoker regret by ethnicity and socioeconomic status: national survey data. . N Z Med J 2009;122(1292):99-100.

19. Edwards R, Wilson N, Thomson G, et al. Majority support by Māori and non-Māori smokers for many aspects of increased tobacco control regulation: national survey data. N Z Med J 2009;122(1307):115-118.

20. Thomson G, Wilson N, Edwards R. Kiwi support for the end of tobacco sales: New Zealand governments lag behind public support for advanced tobacco control policies. . N Z Med J 2010;123(1308):106-111.

Ethnic counts on mortality and census data 2001–06: New Zealand Census-Mortality Study update

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Abstract

Aim To provide an update for the assessment of discrepancies in ethnicity counts in the 2001 census and mortality data for the 2004–2006 period.

Methods 2001 census anonymously and probabilistically linked to 5 years of subsequent mortality data (135,849 eligible mortality records), allowing a comparison of ethnicity recording for the years 2001–2004 and 2004–2006.

Results Using a total definition of ethnicity, census and mortality counts agree reasonably well in 2004–06 and resemble comparisons in 2001–04, except at younger ages where counts for Pacific and Asian ethnicities are up to a third less for mortality data. Due to multiple ethnicities being more commonly recorded on census data, sole ethnicity counts are generally greater on mortality than census data, particularly for Māori ethnicity.

Conclusion Similar to 2001–2004, there is little bias in ethnic group counts between census and mortality data when using total ethnicity. Calculations of mortality rates by ethnicity using unlinked census and mortality data and a total definition of ethnicity should be unbiased. These results support ongoing use of the census definition of ethnicity on all health datasets.

The New Zealand Census-Mortality Study (NZCMS) has previously shown that Māori and Pacific deaths were significantly undercounted on mortality data relative to census data prior to 1995, and relatedly, non-Māori non-Pacific deaths overcounted on mortality data. In 1995, the ethnicity question on the death registration form was altered from the biological definition of race (blood more than half) to a more self-defined question resembling that on the 1996 census.

Thus, although historic mortality trends prior to 1995 required recalculation due to the considerable bias in ethnicity group counts, more recent NZCMS data has shown increasing agreement between census and 2001–04 mortality ethnic counts,[1] at least for the concept of "total ethnicity". This paper provides an update for the 2001 census linked to 2004–06 deaths, assessing any existing discrepancies between mortality and census data for ethnicity counts.

The main objectives of this paper are to examine any existing discrepancies in ethnicity counts for mortality (2004–06 and, by way of comparison, 2001–04) and 2001 census data, and how these differ depending on the ethnicity definition used. This paper provides an update for a series of publications focused on the numerator-denominator bias and changes in this bias over time.

Methods

The methodology used in this paper, using on linked census and mortality data, has been described previously in technical reports and papers.[2–7] For the update 6 March 2004 to 5 March 2006 period, 79.8% of eligible mortality records were anonymously and probabilistically linked to census records. A subset of these records with highly probable links (HPL,76.1% of 2004–06 eligible mortality records), where ethnicity had no effect on linkage probability, was used in analyses to assess any discrepancy in ethnicity counts on the mortality and census data.

The number of deaths in the HPL dataset was weighted up so that it was representative of all 2004–06 eligible mortality records.[7] Weights were calculated based on variables that were predictors of HPL in logistic regression analyses: Age at census, sex, prioritised ethnicity, rurality, residential mobility of area unit, NZ deprivation index, Regional Health Authority, and cause of death. Cells within a stratum that met the numerical criterion of >3 linked records, were separated and assigned an independent weight, whereas the remaining cells were collapsed. The order of collapsing of strata variables to ensure sufficient cell sizes was based on the strength of their relationship with HPL.

Counts of ethnic groupings for census and mortality data were compared using weighted cross-classifications of the HPL dataset. Tabular count output is random rounded to a near multiple of three as per Statistics New Zealand protocol, but the census-mortality ratios were calculated on unrounded data.

Ethnicity definitions

Three ethnicity definitions were used to assess any discrepancies in ethnic counts between the census and mortality data:

- *Total ethnicity* was assigned as Māori if any ethnic group identified on the census or mortality record was Māori. The same was done for Pacific, Asian and "non-Māori non-Pacific non-Asian" (nonMPA) ethnicities. Individuals could be assigned multiple ethnicities and consequently the sum of counts across ethnic groups will be greater than actual number of decedents.

- *Prioritised ethnicity* was assigned as Māori if any of the three possible self-identified ethnicity responses was Māori. For non-Māori, an individual was assigned as Pacific if one of the self-identified ethnic groups was Pacific. For non-Māori non-Pacific, an individual was assigned as Asian if one of the self-identified ethnic groups was Asian. The remaining individuals were assigned as nonMPA (equivalent to sole nonMPA).

- *Sole ethnicity* was assigned as Māori if Māori was the only ethnic group self identified. Similarly, an individual was assigned as Pacific or Asian if it was the only self-identified ethnic group. All others were assigned as "Remainder". This group also included some extra decedents that, for example, self-identified multiple ethnic groups.

Results

Table 1 shows the weighted ethnicity counts for census and death registration form data, as well as the census to mortality ratios for the 2001–2004 and 2004–2006 cohorts for all three ethnicity definitions. There is generally close agreement between the census and mortality data for both cohorts in both the 2001–04 and 2004–06 data. There does appear to be some overcounting of the Māori ethnicities in mortality data relative to census data when using the sole ethnicity definition. This is due to fewer mortality records being assigned multiple ethnicity groups relative to the census data, resulting in greater sole Māori counts on the mortality data.

Table 1. Census and death registration form (mortality) ethnicity totals and ratios in 2001–2004 (n = 82,404 deaths), 2004–2006 (n =53,445 deaths) for total, prioritised and sole ethnicity definition

Ethnicity	2001–2004†			2004–2006		
	Census	Mortality	Census to Mortality Ratio	Census	Mortality	Census to Mortality Ratio
Total						
Māori	7,419	7,539	0.98	5,136	5,205	0.99
Pacific	2,448	2,493	0.98	1,449	1,482	0.98
Asian	1,236	1,215	1.02	804	816	0.99
NonMPA‡	73,089	72,051	1.01	47,262	46,530	1.02
Prioritised						
Māori	7,419	7,539	0.98	5,136	5,205	0.99
Pacific	2,373	2,439	0.97	1,386	1,449	0.96
Asian	1,170	1,155	1.01	777	771	1.01
NonMPA‡	71,442	71,274	1.00	46,143	46,020	1.00
Sole						
Māori	5,931	6,891	0.86	4,095	4,764	0.86
Pacific	2,196	2,274	0.97	1,284	1,338	0.96
Asian	1,098	1,086	1.01	744	735	1.01
Remainder#	73,179	72,153	1.01	47,322	46,602	1.02

Counts have been weighted and random rounded to a multiple of three as per Statistics New Zealand protocol. Note that the sum of observations for Prioritised and Sole ethnicities is equal to the total number of records, but the sum of observations for Total ethnicity is larger than this.

† The 2001–04 counts and ratios are sourced from Tables 10–13 of Fawcett et al. (2008)

‡ The 'total nMnPnA' group was defined those people with one or more self-(undertaker-) defined ethnic groups, of which one was nMnPnA. The 'prioritized nMnPnA' is best thought of those remaining after all census respondents or decedents with any one of Māori, Pacific or Asian ethnicity have been 'prioritised out'. This is equivalent to the 'sole nMnPnA' group.

The 'Remainder' group in sole ethnicity includes any people who reported nMnPnA ethnic group (i.e. the 'total nMnPnA' group) *plus* some extra decedents or census respondents who were recorded as, say, both Māori and Pacific and therefore not eligible to be either 'sole Māori' or 'sole Pacific'.

The counts for total ethnicity in 2001–04 and 2004–06, stratified by sex, age, regional health authority and NZ Deprivation Index are shown in Table 2. The counts for census and mortality data are largely congruent, and similar between the two cohorts The notable exception is the undercounting of total ethnicity in the mortality data for younger age groups (<25 years) for all ethnic groups, but especially for Pacific and Asian ethnicities. This is because multiple ethnicities are much more commonly identified on the census data than mortality data. Also note that the 2004–06 cohort counts are fewer and consequently less stable than the counts for the 2001–04 cohort.

Table 2. Census and death registration form Total ethnicity counts and ratios by sex, age, RHA and NZ Deprivation in 2001–04 (n=82,404), 2004–06 (n= 53,445)

Variables	Ethnicity		2001–2004			2004–2006	
		Census	Death Registration Form	Census to Mortality Ratio	Census	Death Registration Form	Census to Mortality Ratio
Sex							
Male	Māori	4,095	4,140	0.99	2,793	2,841	0.98
	Pacific	1,365	1,371	1.00	798	810	0.99
	Asian	681	642	1.06	459	459	1.00
	NonMPA	36,042	35,499	1.02	22,932	22,560	1.02
Female	Māori	3,324	3,396	0.98	2,343	2,364	0.99
	Pacific	1,086	1,122	0.97	651	669	0.97
	Asian	552	573	0.97	348	357	0.97
	NonMPA	37,047	36,552	1.01	24,333	23,970	1.02
Age							
0–14 yrs	Māori	195	189	1.03	69	63	1.10
	Pacific	63	60	1.02	21	15	1.40
	Asian	18	21	0.91	18	12	1.50
	NonMPA	354	309	1.14	126	105	1.20
15–24 yrs	Māori	480	495	0.97	459	483	0.95
	Pacific	174	156	1.10	132	111	1.19
	Asian	87	75	1.16	39	27	1.44
	NonMPA	1,161	1,062	1.10	1,017	912	1.12
25–44 yrs	Māori	786	792	0.99	765	780	0.98
	Pacific	237	234	1.01	186	180	1.03
	Asian	114	105	1.09	87	96	0.91
	NonMPA	2,064	1,947	1.06	1,890	1,779	1.06
45–64 yrs	Māori	2,589	2,637	0.98	1,443	1,458	0.99
	Pacific	732	726	1.01	363	375	0.97
	Asian	282	261	1.09	171	168	1.02
	NonMPA	9,348	9,048	1.03	5,157	4,989	1.03
65–74 yrs	Māori	1,863	1,896	0.98	1,251	1,257	1.00
	Pacific	558	579	0.97	339	345	0.98
	Asian	282	285	0.98	192	201	0.96
	NonMPA	12,852	12,648	1.02	7,512	7,383	1.02
75–84 yrs	Māori	1,098	1,089	1.01	840	852	0.99
	Pacific	480	522	0.92	294	321	0.92
	Asian	297	297	1.00	189	198	0.95
	NonMPA	23,937	23,775	1.01	15,765	15,636	1.01
85+ yrs	Māori	411	441	0.93	315	306	1.03
	Pacific	204	213	0.96	120	138	0.87
	Asian	156	174	0.90	111	114	0.97

Variables	Ethnicity	Census	2001–2004 Death Registration Form	Census to Mortality Ratio	Census	2004–2006 Death Registration Form	Census to Mortality Ratio
	NonMPA	23,370	23,262	1.00	15,804	15,726	1.00
RHA							
Northern	Māori	2,253	2,298	0.98	1,566	1,596	0.98
	Pacific	1,716	1,758	0.98	1,023	1,059	0.97
	Asian	684	687	0.99	459	471	0.97
	NonMPA	19,902	19,578	1.02	12,681	12,486	1.02
Midland	Māori	2,835	2,913	0.97	1,965	1,989	0.99
	Pacific	174	177	0.99	102	84	1.21
	Asian	129	111	1.17	63	69	0.91
	NonMPA	14,850	14,454	1.03	9,831	9,591	1.03
Central	Māori	1,713	1,743	0.98	1,200	1,227	0.98
	Pacific	459	444	1.03	246	255	0.96
	Asian	276	279	0.99	198	189	1.05
	NonMPA	19,080	18,891	1.01	12,390	12,186	1.02
Southern	Māori	618	585	1.05	405	393	1.03
	Pacific	99	114	0.88	78	84	0.93
	Asian	144	135	1.06	90	90	1.00
	NonMPA	19,257	19,125	1.01	12,357	12,267	1.01
NZ Deprivation							
1–4	Māori	312	303	1.03	696	672	1.04
	Pacific	84	93	0.89	147	174	0.84
	Asian	273	267	1.02	303	318	0.95
	NonMPA	11,781	11,709	1.01	16,977	16,824	1.01
5–6	Māori	597	603	0.99	687	678	1.01
	Pacific	159	174	0.93	183	189	0.97
	Asian	237	234	1.01	180	171	1.05
	NonMPA	14,565	11,460	1.01	11,118	11,022	1.01
7–8	Māori	1,062	1,011	1.05	1,140	1,164	0.98
	Pacific	273	294	0.93	276	282	0.98
	Asian	231	228	1.00	171	177	0.97
	NonMPA	16,794	16,659	1.01	10,992	10,839	1.01
9–10	Māori	1,659	1,704	0.97	2,616	2,691	0.97
	Pacific	486	477	1.02	840	834	1.01
	Asian	282	282	1.00	150	153	0.98
	NonMPA	17,451	17,163	1.02	8,175	7,845	1.04

All counts are weighted, and then random rounded to a multiple of three as per Statistics New Zealand protocol. Minimum cell size is 6.

Full cross-classifications of census and mortality 2004–2006 data for prioritised and sole ethnicity definitions are shown in Table 3 and 4. These show the specific mismatches in ethnicity counts between the census and mortality files. The majority

of misclassifications were between Māori and nonMPA groups. Using a sole definition of ethnicity, the number of Māori deaths according to mortality data exceeds census data (i.e. census-mortality ratio <1.0), due to multiple self-identified ethnicities being more common on census data, this causes sole Māori counts to be lower on census than mortality data.

Table 3. Census by mortality counts for prioritised ethnicity by sex, 2004–06

Sex	Census Prioritised Ethnicity	Māori Deaths	Pacific Deaths	Asian Deaths	NonMPA Deaths	Total Census Deaths	Census to Mortality Ratio
Males	Māori	2,556	9	6	222	2,793	0.98
	Pacific	9	723	6	27	756	0.96
	Asian	.	18	405	18	444	1.02
	Non MPA	276	39	30	22,023	22,368	1.00
	Total	2,841	789	435	22,296		
Females	Māori	2,133	6	.	204	2,343	0.99
	Pacific	12	594	6	21	630	0.95
	Asian	.	15	312	9	336	1.01
	Non MPA	219	45	18	23,487	23,772	1.00
	Total	2,364	660	333	23,724		

All counts are weighted, and then random rounded to a multiple of three as per Statistics New Zealand protocol. Minimum cell size is 6.

Table 4. Census by mortality counts for sole ethnicity by sex, 2004–06

Sex	Census Sole Ethnicity	Māori Deaths	Pacific Deaths	Asian Deaths	Remainder Deaths	Total Census Deaths	Census to Mortality Ratio
Males	Māori	2,394	6	6	390	2,268	0.87
	Pacific	9	693	6	60	714	0.98
	Asian	.	6	396	42	417	0.98
	Non MPA	204	27	24	22,110	22,965	1.02
	Total	2,604	732	426	22,602		
Females	Māori	1,992	6	.	345	1,827	0.85
	Pacific	9	570	6	48	570	0.94
	Asian	.	6	294	39	324	1.04
	Remainder	159	33	12	23,568	24,357	1.01
	Total	2,160	609	312	24,003		

All counts are weighted, and then random rounded to a multiple of three as per Statistics New Zealand protocol. Minimum cell size is 6.

Discussion

Using a total definition of ethnicity, census and mortality counts agree reasonably well in 2004–06 and were similar to 2001–04, except at younger ages where mortality data counts for Pacific and Asian are up to a third (1–1.5) less for mortality data – although numbers are sparse and hence estimates unstable. Due to multiple ethnicities being more commonly recorded on census data, prioritised and sole comparisons differ; Māori sole counts are greater on mortality than census data.

The current analyses necessarily used only a subgroup of the total mortality records (the highly probable links, HPL) to estimate numerator denominator bias. By weighting up these HPL links to be representative of all mortality records, we are assuming that within strata of that weighting (i.e. sex by age by prioritised ethnicity (mortality data), rurality, residential mobility of area unit, NZ deprivation index, region, and cause of death) that the (dis)agreement of mortality and census data ethnicity is the same among the HPL dataset as the non-HPL dataset. Unfortunately, we cannot prove this assumption. However, we are reasonably confident that within all cross-classifications of these strata that we essentially adjust for any selection bias that may arise in using the subsidiary HPL data-set.

The use of a total definition of ethnicity appears to be the most accurate and consequently if this definition is used for the calculation of ethnic mortality rates, there should be little to no numerator-denominator bias. As with our last update, these results again provide support to the sector for ongoing attempts to ensure health data uses an ethnicity question as close in wording and layout to the census question as possible.

Competing interests: None known.

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References:

1. Blakely T, Atkinson J, Fawcett J. Ethnic counts on mortality and census data (mostly) agree for 2001-2004: New Zealand Census-Mortality Study update. NZ Med J 2008; 121(1281):58-62.

2. Ajwani S, Blakely T, Robson B, et al. Unlocking the numerator-denominator bias for the 1980s and 1990s. NZCMS Technical Report No. 4. ISBN 0-473-09111-9. Wellington: Department of Public Health, Wellington School of Medicine and Health Sciences, University of Otago, 2002.

3. Ajwani S, Blakely T, Robson B, et al. Unlocking the numerator-denominator bias III: adjustment ratios by ethnicity for 1981-1999 mortality data. The New Zealand Census-Mortality Study. NZ Med J 2004; 116 (1175):6.

4. Blakely T, Atkinson J. Unlocking the Numerator-Denominator Bias, 1991-94 Deaths. NZCMS Technical Report No. 2. Wellington: Department of Public Health, Wellington School of Medicine, University of Otago, 2001.

5. Hill S, Atkinson J, Blakely T. Anonymous record linkage of census and mortality records: 1981, 1986, 1991, 1996 census cohorts. NZCMS Technical Report No. 3. ISBN 0-473-09110. Wellington: Department of Public Health, Wellington School of Medicine and Health Sciences, University of Otago, 2002.

6. Fawcett J, Atkinson J, Blakely T. Record linkage of census and mortality 2001-04 records. New Zealand Census-Mortality Study Technical Report No. 6. Wellington: Health Inequalities Research Programme, University of Otago, Wellington, 2008.

7. Tan L, Blakely T, Atkinson J. Linkage bias and numerator-denominator bias in census and mortality 2001-06 records. NZCMS Technical Report No 7. Wellington: Health Inequalities Research Programme, University of Otago, Wellington, 2010.

The prevalence of colorectal adenomas in Māori and New Zealand Europeans parallels colorectal cancer rates

Graeme Dickson, Chris W Cunningham, Susan Parry

Abstract

Background New Zealand (NZ) has a high incidence of colorectal cancer (CRC). Māori have a documented incidence that is approximately half that found in NZ Europeans, possibly the result of under-reporting.

Aim To determine and compare the prevalence of colorectal adenomas in Māori and NZ European patients.

Methods Colonoscopy records from the Middlemore Colonoscopy Audit Database between 1 July 2001 and 31 December 2005 were reviewed. Studies performed for indications associated with an increased risk of colorectal polyps were excluded from the analysis. Patient demographics, including self-identified ethnicity, and number and location of colonic polyps were recorded. All polyp histology was reviewed.

Results Data was analysed from 2842 colonoscopies—2523 were NZ Europeans (mean age 67 yrs) and 319 were Māori patients (mean age 60.6 yrs). To adjust for age, a comparison of data between 40 and 59 years was undertaken. In 643 (81.2%) NZ Europeans, polyps were identified in 213 (33.1%). In the 149 (18.8%) Māori patients, polyps were identified in 35 (23.5%) p=0.029. The comparative rates of adenomas in NZ Europeans and Māori were 16.7% and 8.7% respectively (p=0.019; 8% difference, CI=2.3-13.9%).

Conclusion The prevalence of colorectal adenomas in Māori is approximately half that found in NZ Europeans. This mirrors the reported difference in CRC incidence between these groups and lends support to this being a real finding and not a bias in the manner in which the data has been collected.

New Zealand has a high incidence of colorectal cancer with an age standardised incidence (non-Māori, 2005) of 51.9 per 100,000.[1,3] However, the rate amongst the Māori population appears to be approximately half that of the non-Māori population.[3] Despite this difference the CRC mortality rate for Māori is no better than non-Māori.[2,3,17] This is partially explained by a greater proportion of Māori presenting at a more advanced stage.[2] Correspondingly there is poorer survival from diagnosis for Māori. The documented lower CRC incidence rate for Māori remains unexplained. One potential explanation is incomplete recording of ethnicity at the time of diagnosis.

To minimise the influence of any confounding factors we decided to study the prevalence of adenomatous polyps which are the precursors of colorectal cancer.[4] The differences in prevalence between ethnicities should reflect the differences in cancer incidence.[5]

Methods

We performed a retrospective review of all colonoscopies recorded on the Middlemore Colonoscopy Audit Database between 1/7/01 and 31/12/05. Demographics, patient self-identified ethnicity, polyp details and indication for colonoscopy were recorded. Indications which were associated with a low prevalence of polyps (e.g. inflammatory bowel disease) or a high prevalence (e.g. previous polyps, family history of cancer or abnormal radiology) were excluded. The endoscopy database, Endoscribe, was used to determine polyp location, number and size. Polyp histology was checked from case notes and presence of high risk features (high grade dysplasia, tubulovillous, size>1cm or >3 adenomas) documented.

Statistical analysis: Continuous data was expressed as the mean ± standard deviation. A p value<0.05 was considered statistically significant. Statistical analysis was carried out using Graphpad.[15] Proportions were analysed using Fisher's exact test and continuous variables with an unpaired t-test.

Results

2842 (2842) colonoscopies were performed for accepted indications (2523 European, 319 Māori). The average age of the European group (64.7±21.1) was significantly older than the Māori group (55.8±14.2; p=0.0001). Accordingly to ensure parity we selected and analysed only the 40-59 years age bracket as the majority of CRC occurs after the age of 50 years, with precursor adenomas developing 5- 10 years previously. This age bracket also had the highest number of patients identified as Māori.

Average ages were not significantly different (Māori 50.2±6.3, European 50.4±6.3; p=0.72) and the sex ratio was also comparable (Māori 44% male, Europeans 40.6%; p=0.46). At colonoscopy, polyps were found in 213 Europeans (33.1%) but only 35 Māori (23.5%; p=0.029). At histology, the adenomatous polyp rate in Europeans was 16.7% compared to 8.7% in Māori (p=0.019; 8% difference, CI=2.3-13.9%).

Table 1 Polyps and colorectal cancer in the 40-59 year old age band

Ethnicity	Number of patients	Number of cancers	Number with polyp at colonoscopy	Number with adenoma at histology
European	643	20	213	107 (16.7%)
Māori	149	1	35	13 (8.7%)

Amongst patients with adenomas, the percentage showing high risk features was not significantly different (European 39.3%, Māori 38.5%; p=1.0). There was also no difference in the average number of adenomatous polyps per person (European 1.7±1.3, Māori 1.9±1.4; p=0.59) or location in the colon (right colon in 41% European and 52% Māori; p=0.39).

Table 2. Adenomatous polyp characteristics, location and number in the 40–59yr age band

Ethnicity	Number of patients with at least one adenoma	Percentage of these patients with high-risk adenomas	Percentage of total adenomas located in the right colon	Average number of adenomas
European	107	42/107(39.3%)	70/169(41%)	1.7±1.3
Māori	13	5/13(38.5%)	13/25(52%)	1.9±1.4

Discussion

Differences in the incidence of CRC, between ethnicities, are likely to result from a combination of genetic and environmental factors.

Epidemiology suggests that colorectal cancer is highly dependent on the environment. This dependence is illustrated by the high rates of CRC in the offspring of Japanese immigrants to USA compared to the low rates of CRC for Japanese in Japan.[1] It is unlikely that genetic predisposition to CRC would change so quickly.

Within the United States of America, African Americans have an increased incidence of CRC compared to the White population.[6] The reasons for the higher incidence are not known but environmental factors such as diet, exercise and smoking have been implicated.[7,8] Some biological differences have also been noted. There is evidence of a more proximal distribution of cancers and adenomatous polyps in African Americans.[9] Also, the rates of microsatellite instability (MSI) in sporadic cancers are much higher in African Americans (45%) than would be expected from the literature (12-17%).[10] MSI-high often implies a better prognosis in CRC patients but its effect on the outcome of African Americans is not clear. Overall, ethnic differences have led to the recommendation that CRC screening starts 5 years earlier in African Americans (45yrs old).

However, recognised environmental risk factors do not appear to explain the differences in incidence of CRC between Māori and NZ Europeans.[11] Factors associated with higher risk of CRC are paradoxically more prevalent in Māori who are more likely to be overweight and have higher intakes of fat, energy and alcohol than NZ Europeans.[12,13,16] Although vegetable intake is similar in Māori and NZ Europeans, it has been suggested that the different types of vegetables consumed may be protective.[14] Māori commonly eat watercress, puha (sow thistle), melon, kumara and silverbeet in greater quantities than NZ European. However evidence for the presence of anticarcinogens in these particular foods is lacking.

An explanation for the reported difference in incidence of CRC in Māori and the NZ European remains illusive. Our study contributes usefully by showing that the prevalence of colorectal adenomas, the precursor lesions in CRC, in Māori is approximately half that found in NZ Europeans. This mirrors the reported difference in CRC incidence between these groups.[3] In addition, amongst patient with adenomas there was no significant difference in the number of adenomas or percentage of adenomas displaying high risk features between Māori and European . Accordingly the poorer outcome for Māori with this disease may reflect a lack of timely access to quality diagnosis and treatment, rather than an adverse biologic predilection per se.

Further research comparing the biology and histology of colorectal cancer between these groups would be required to strengthen this conclusion. However, if timely access to quality medical services is a concern the importance of health initiatives to improve outcomes for CRC in Māori would be reinforced.

We recognise that the sample population in this study were a symptomatic group and may not represent the general population. However, there are limited opportunities to study the general (asymptomatic) population due to the low autopsy rate and the

absence of a CRC screening program in New Zealand. Additionally, the number of adenomas in the Māori group was small and could be associated with a type 2 error.

Conclusion

The prevalence of colorectal adenomas in Māori is approximately half that found in NZ Europeans. This finding mirrors the reported difference in colorectal cancer incidence and supports this being a real finding. There were no significant differences between Māori and NZ Europeans in the proportion of high risk adenomas, the total number of polyps found and the location in right or left colon. This is potentially a positive health finding for Māori as improvements in diagnosis and management of colorectal cancer could see mortality rates halve.

Competing interests: None known.

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References:

1. Boyle P, Langman JS. ABC of colorectal cancer: Epidemiology. BMJ. 2000 Sep 30;321(7264):805-8.
2. Robson B, Purdie G, Cormack D. Unequal Impact: Māori and Non-Māori Cancer Statistics 1996–2001. Wellington: Ministry of Health; 2005.
3. Ministry of Health. 2009. Cancer: New Registrations and Deaths 2005: Revised edition. Wellington: Ministry of Health: www.moh.govt.nz
4. Jass JR. Do all colorectal carcinomas arise in pre-existing adenomas? World J Surg. 1989;13(1):45-51.
5. Clark JC, Collan Y, Eide TJ, et al. Prevalence of polyps in an autopsy series from areas with varying incidence of large bowel cancer. Int J Cancer. 1985 Aug 15;36(2):179-186.
6. Agrawal S, Bhupinderjit A, Bhutani MS, et al. Colorectal Cancer in African Americans. Am J Gastroenterol. 2005 Mar;100(3):515-23; discussion 514
7. Baquet CR, Commiskey P. Colorectal cancer epidemiology in minorities: a review. J Assoc Acad Minor Phys. 1999;10(3):51-8.
8. Hargreaves MK, Baquet C, Gamshadzahi A. Diet, nutritional status, and cancer risk in American blacks. Nutr Cancer. 1989;12(1):1-28.
9. Nelson RL, Dollear T, Freels S, Persky V. The relation of age, race, and gender to the subsite location of colorectal carcinoma. Cancer. 1997 Jul 15;80(2):193-7.
10. Ashktorab H, Smoot DT, Carethers JM, et al. High incidence of microsatellite instability in colorectal cancer from African Americans. Clin CA Res. 2003 Mar;9:1112-7
11. Smith AH, Pearce NE, Joseph JG. Major colorectal cancer aetiological hypotheses do not explain mortality trends among Māori and non-Māori New Zealanders. Int J Epidemiol. 1985 Mar;14(1):79-85.
12. Birkbeck JA. New Zealanders and their diet : A report to the National Heart Foundation on the National Diet Survey 1977. Dunedin: Otago University Press; 1977.

13. Dryson E, Metcalf P, Baker J, Scragg R. The relationship between body mass index and Socioeconomic status in New Zealand: Ethnic and Occupational factors. N Z Med J. 1992;105:233-235

14. Ferguson LR, Yee RL, Scragg R, Metcalf PA, Harris PJ. Differences in intake of specific food plants by Polynesians may explain their lower incidence of colorectal cancer compared with Europeans In New Zealand. Nutr Cancer. 1995;23(1):33-42

15. GraphPad Software Inc 2002-2005. www.graphpad.com

16. Casswell S. Drinking by New Zealanders. Wellington: Alcoholic Liquor Advisory Council, 1980:1-12

17. Shaw C, Blakely T, Sarfati D, Fawcett J, Peace J. Trends in colorectal cancer mortality by ethnicity and socio-economic position in New Zealand, 1981-99: one country, many stories. Aust N Z J Public Health 2006;30(1):64-70.

Orbital infection in New Zealand: increased incidence due to socioeconomic deprivation and ethnicity

Nicholas R Johnston, Gordon Sanderson

Abstract

Aim This study aimed to identify the relationship between the incidence of orbital infection, ethnicity and socioeconomic deprivation in New Zealand.

Method Cases admitted to all public hospitals in New Zealand with the ICD-10 diagnosis of acute inflammation of the orbit for a 9-year period were retrieved from the National Minimum Data Set. Incidence rates of acute infection of the orbit were correlated with socioeconomic deprivation (measured by New Zealand Deprivation Index) and ethnicity.

Results There were 530 cases admitted with acute orbital inflammation over a 9-year period from 1 July 2000 to 30 June 2009. This study identified a significant association between orbital infection incidence and socioeconomic deprivation and ethnicity.

Cases in the moderate deprivation group had 1.5 times the rate of the least deprived group and the most deprived group had 2.9 times the rate of orbital infection of the least deprived group. Māori had 1.9 times the rate of the European group, and Pacific people had 3.6 times the rate of European group.

Conclusion Greater socioeconomic deprivation, and ethnicity was associated with an increased incidence of orbital infection in New Zealand. The reasons why these associations exist are currently not clear.

Orbital infections are a group of potentially vision and life threatening infections. Orbital infections can range from inflammatory oedema to orbital abscess with cavernous sinus thrombosis.[1] They are more common in younger children, and are often associated with sinusitis.[2] All cases presenting with proptosis, red eye, reduced or painful eye movements and decreased vision are assumed to be an orbital infection until proven otherwise.[3]

Māori and Pacific people have been shown to have higher rates of many diseases, often with poorer prognosis.[4–7] Deprivation is associated with late presentation of ocular and other diseases.[8–11] In New Zealand both socioeconomic deprivation and being Māori or Pacific people have been associated with poorer health outcomes.[5,12,13]

No published study to our knowledge has looked at the effect of ethnicity or socioeconomic deprivation on the rate of orbital infection. We report the first study looking at socioeconomic deprivation and ethnicity role in orbital infection in New Zealand.

Methods

The New Zealand National Minimum Dataset (NMDS) is a national collection of public and private hospital discharge information, including clinical information, for inpatients and day cases.[14] It uses ICD-10 codes to record primary and secondary admission diagnoses. There is no ICD -10 code for orbital infection so ICD-10 code H050: acute orbital inflammation was used, which includes abscess, cellulitis, osteomyelitis, periostitis, and tendonitis. [15]

All cases with the diagnosis of acute inflammation of the orbit (ICD-10 H050) for a 9-year period from 1 July 2000 to 30 June 2009 were retrieved from the NMDS.

Socioeconomic deprivation was measured by the New Zealand Deprivation index 2001 (NZDep01).[16] This tool uses a meshblock (a geographically defined) measure of deprivation extracted from New Zealand census data. It is based on income, employment, communication, transport, support, qualifications, home ownership and living space.

The NZDep01 divides New Zealand into geographically defined deciles giving a score from 1 to 10, with 10 being the most deprived.[16] For the purposes of this study we grouped NZDep01 into 3 groups 1-3, 4-7 and 8-10, giving low, medium and high levels of deprivation.

In the NMDS ethnicity is self-identified at the time of hospital admission. Up to three ethnic group codes are reported and the Statistics New Zealand prioritisation algorithm is used to generate a prioritised ethnicity that was employed in this study. The cases were grouped into European, Māori, Pacific peoples and other.

Secondary diagnoses, procedures, admitting specialties, and length of hospital stays were also collected. Adjusted incidences to the New Zealand European population where calculated using New Zealand Census data, using Stata/IC 11.0 for Mac.

Results

Over a 9-year period, 530 cases were identified, an incidence of 1.31 per 100,000/year of which 54.5% were male. The median age of admission was 13 years with range from 0 to 91 years. The median length of admission was 3 days with a range of 0-33 days.

The prioritised ethnicity of the cases were 47.9% European, 23.2% Māori, 21.1% Pacific people, and 7.7% were another ethnic group. Cases were admitted under several specialties: ophthalmology (36.2%), paediatrics (32.6%), otolaryngology (9.7%), medicine (11.4%), or other surgical specialties (10.1%).

Fifty-four percent of cases came from the three most deprived socioeconomic groups, with 22.2% of cases being from the most socioeconomically deprived group. There was an increasing proportion of cases admitted with orbital infection as socioeconomic deprivation increased (See Figure 1).

Sixty percent of cases had had a procedure performed. The most common surgical procedures were nasal or sinus surgery (21.7%) and orbital surgery (18.1%). Cases' secondary diagnoses consistent with orbital infection were sinusitis (37.4%), bacterial infection (29.2%), other ocular inflammation/infection (17%), and cellulitis of the face (8.7%).

Figure 1. Cases of orbital infection by NZDep01
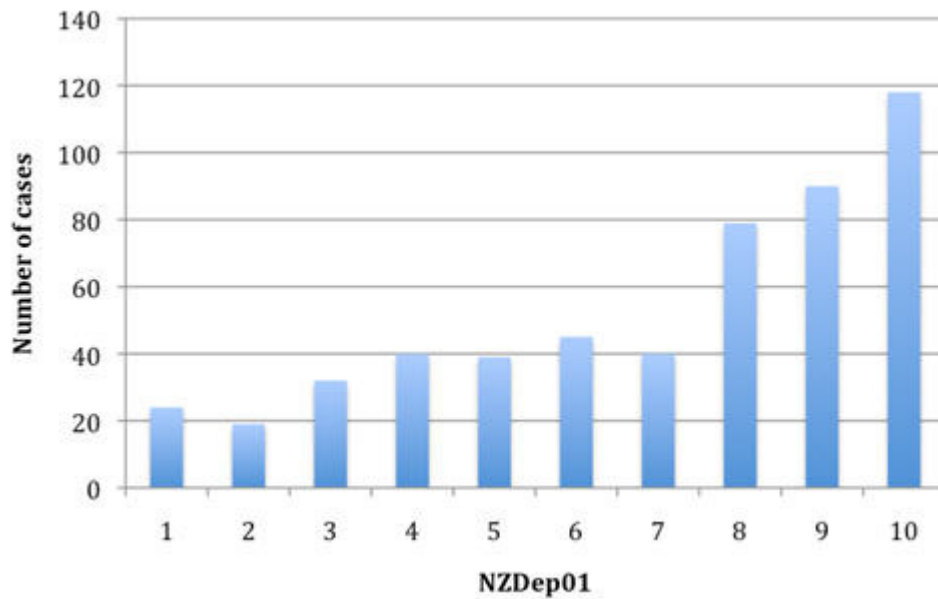
Age group infection rates adjusted for deprivation were calculated for each ethnicity (see Figure 2). Pacific people had, for most age groups, a higher rate than both Māori and European people, except for in the over 70 years of age groups where Māori had the highest rates (2 cases in a population of 888).

Figure 2. Adjusted orbital infection rates in 5-year age groups by ethnicity
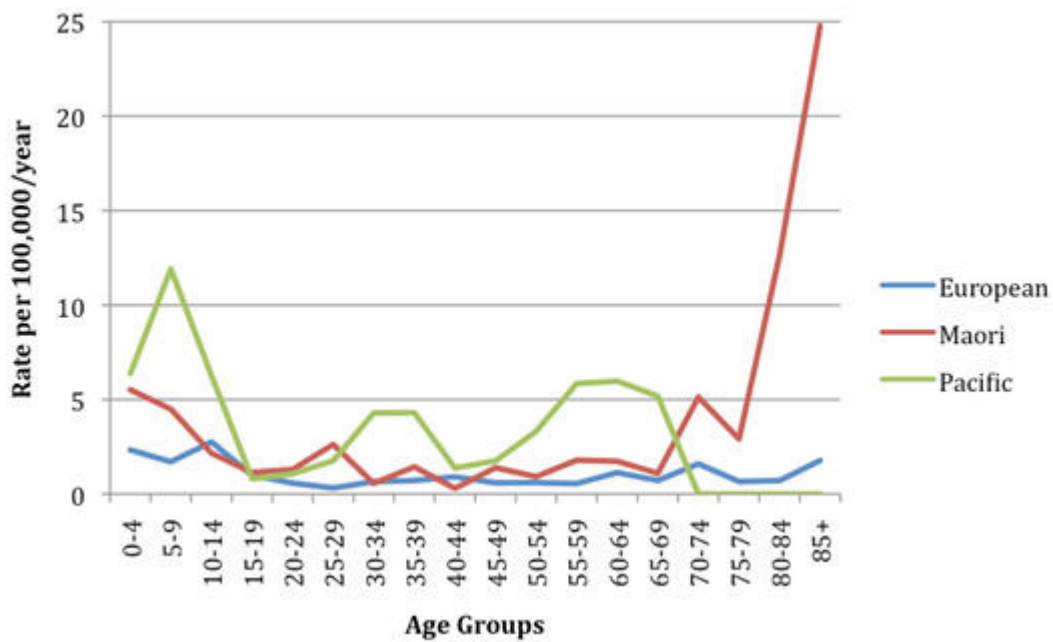
The low socioeconomic deprivation group had an incidence rate (adjusted for age and ethnicity) of 6.3/100,000, the medium socioeconomic deprivation group had 9.2/100,000 and 18.4/100,000 in the high socioeconomic deprivation group. A statistically significant association was found between NZDep01 score, and age and ethnicity-standardised incidence ratios of orbital infection (see Table 1).

Table 1. Orbital infection rates and standardised incidence ratios (SIR) adjusted for age and ethnicity by socioeconomic deprivation

Deprivation group	Cases observed	Adjusted rate/100,000	SIR	SIR confidence interval
Low deprivation	75	6.3	1	0.79–1.25
Medium deprivation	164	9.2	1.5	1.25–1.71*
High deprivation	287	18.4	2.9	2.60–3.29*

*Statistically significant difference (p <0.05).

The European group had an incidence (adjusted for deprivation and age) of 9.7/100,000, the Māori group had 18.3/100,000 and Pacific 31.12/100,000. A statistically significant association was found between ethnicity and age-standardised incidence ratios of orbital infection (see Table 2).

Table 2. Orbital infection rates and standardised incidence ratios (SIR) adjusted for age and deprivation ethnicity

Ethnicity	Cases observed	Adjusted rate/100,000	SIR	SIR confidence interval
European	254	9.7	1	0.88–1.13
Māori	121	18.3	1.9	1.56–2.24*
Other	40	4.2	0.4	0.31–0.59*
Pacific	111	34.7	3.7	2.93–4.30*

*Statistically significant difference (p <0.05).

Compared to Europeans in the low deprivation group there was a higher incidence of orbital infection as deprivation increased in the European group (see Table 3). There was also an increased rate of orbital infection as deprivation increased in the Māori group. In the Pacific people group there was no statistically significant difference between the two least deprived groups and the least deprived European group, but the most deprived group had a statistically significant increased rate of orbital infection.

Table 3. Orbital infection rates and standardised incidence ratios (SIR) by ethnicity and socioeconomic deprivation

Ethnicity	Deprivation	Cases observed	Adjusted rate/100,000	SIR	SIR confidence interval
European	Low deprivation	64	7.7	1	0.77–1.28
	Medium deprivation	113	10.2	1.3	1.08–1.58*
	High deprivation	77	11.3	1.5	1.15–1.82*
Māori	Low deprivation	2	2.8	0.37	0.04–1.33
	Medium deprivation	25	12.9	1.7	1.08–2.47*
	High deprivation	94	27.9	3.6	2.91–4.42*
Other	Low deprivation	5	1.9	0.25	0.08–0.58*
	Medium deprivation	17	4.6	0.60	0.35–0.96*
	High deprivation	18	6.3	0.82	0.49–1.30
Pacific	Low deprivation	4	20.4	2.6	0.72–6.78
	Medium deprivation	9	13.7	1.8	0.81–3.38
	High deprivation	98	48.3	6.3	5.09–7.63*

*Statistically significant difference (p <0.05).

Discussion

This is the first reported population-based study of orbital infection by ethnicity and deprivation index in the world. Both deprivation and ethnicity were independently associated with differences in orbital infection rates. The strength of this study lay in its population-base, and that almost all cases with orbital infection would be admitted to hospital, however the data was anonymised so cases transferred between hospitals may have appeared more than once.

One of the weaknesses of the study was that NZDep is not a perfect measure of each case's socioeconomic deprivation, "Not all 'deprived people' live in deprived small areas".[17] NZDep is not a measure of individual socioeconomic deprivation but of the neighbourhood's deprivation, but this is the best measure available to us at a population level.[17]

We were unable to measure only orbital infection, but used the closest ICD10 code H050, which includes a number of other related diagnoses.[15] There was the possibility for incorrect coding, but clinical coders receive training and audit to ensure accuracy.[14]

It was likely that we did have a significant number of orbital inflections; approximately 40% had a sinusitis and 8% had a facial cellulitis diagnosed, and about 20% had sinus and/or orbital surgery. The low rate of surgery was not surprising as purely medical management in younger cases with intravenous antibiotics, covering upper respiratory tract pathogens is often effective.[18, 19]

In the European and Māori groups there were increased rates of orbital infection as socioeconomic deprivation increased. This was not shown in the Pacific group. This was likely to be due to the large confidence intervals caused by the small number of Pacific people in the two least deprived groups in the New Zealand population.

The very high rate of orbital infection in Māori over 70 years of age may due to the small size of the census population in this group, as this would give a very high incidence rate with a large confidence interval, when a small number of cases of orbital infection were identified.

Links between socioeconomic deprivation and ethnicity, and other diseases have been observed in New Zealand.[5–7,12,20] Higher levels of socioeconomic deprivation are associated with higher rates of total mortality, admission to hospital, second hand smoke exposure, diabetes, rheumatic fever, ischaemic heart disease, obesity, cervical cancer, breast cancer, hypertension, chronic obstructive pulmonary disease, dental disease, psychological distress and complications of pregnancy.[5–7,12,20]

Māori and Pacific people have higher rates of hospital admission, skin infections, mortality, secondhand smoke exposure, obesity, hypertension, ischaemic heart disease, diabetes, chronic obstructive pulmonary disease, dental disease, and psychological distress.[4, 6, 7, 13, 21]

More than half the Māori population and Pacific peoples live in NZdep01 deciles 8-10,[7] but socioeconomic deprivation does not completely explain the health differences between European and Māori or Pacific people.[7]

Higher orbital infection rates may be due to delayed presentation to primary health care, lack of education, transport or finances. Māori and socioeconomically deprived people have been shown to be less likely to be able to see a primary healthcare provider within 24 hours and not have seen a GP due to cost.[6]

Māori and Pacific people are more likely to report that they feel they are not treated with respect or dignity or listened to carefully, by their healthcare provider.[6]

There have been education campaigns addressed at healthcare providers not to give antibiotics for sinusitis as it is often viral.[22] If the socioeconomically deprived, Māori or Pacific people were prescribed antibiotics they may not take it as people in the most deprived neighbourhoods, and Māori and Pacific people are significantly more likely to have an uncollected prescription due to cost.[6]

There may be some other environmental factors, such as overcrowding at home that ethnicity and socioeconomic deprivation are markers of, rather than the cause of increased orbital infection rates. Māori and Pacific people may have different sinus or orbital anatomy or physiology that puts them at a higher risk of orbital infection.

The association between the orbital infection and ethnicity were different from the association with deprivation, which indicated that ethnicity is not just a marker of socioeconomic deprivation. There may be modifiable factors for Māori, Pacific people and those with socioeconomic deprivation that could potentially decrease their risk of orbital infection.

Identification of these factors may lead to a reduction in the incidence of orbital infection in New Zealand. This suggests a need for targeted health interventions to Māori and Pacific people as well as the socioeconomically disadvantaged.

In summary, this study reports a significant association between New Zealand orbital infection incidence and ethnicity and socioeconomic deprivation (as measured by the NZDep01 deprivation index). The reasons why these associations exist are currently not clear.

Competing interests: None known.

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References:

1. Chandler JR, Langenbrunner DJ, Stevens ER. The pathogenesis of orbital complications in acute sinusitis. Laryngoscope. Sep 1970;80(9):1414-1428.

2. Pond F, Berkowitz RG. Superolateral subperiosteal orbital abscess complicating sinusitis in a child. Int J Pediatr Otorhinolaryngol. May 25 1999;48(3):255-258.

3. Ehlers JP, Shah CP, eds. The Wills Eye Manual: Office and Emergency Room Diagnosis and treatment of Eye Disease. Fifth ed. Baltimore: Lippincott, Williams & Wilkins; 2008.

4. Joshy G, Porter T, Lievre CL, Lane J, Williams M, Lawrenson R. Research report: Prevalence of diabetes in New Zealand general practice: the influence of ethnicity and social deprivation. J Epidemiol Community Health. 2009;63:386-390

5. McKenzie F, Jeffreys M, Mannetje AT, Pearce N. Prognostic factors in women with breast cancer: inequalities by ethnicity and socioeconomic position in New Zealand. Cancer Causes Control. 2008;19:403-411.

6. Ministry of Health. A Portrait of Health. Key Results of the 2006/07 New Zealand Health Survey. Wellington: Ministry of Health; 2008.

7. Tobias M, Howden-Chapman P, eds. Social Inequalities in Health, New Zealand 1999. Wellington: Ministry of Health; 1999.

8. Fraser S, Bunce C, Wormald R, Brunner E. Deprivation and late presentation of glaucoma: case-control study. BMJ. March 17, 2001 2001;322(7287):639-643.

9. Klein R, Klein BE, Jensen SC, et al. The relation of socioeconomic factors to age-related cataract, maculopathy, and impaired vision. The Beaver Dam Eye Study. Ophthalmology. Dec 1994;101(12):1969-1979.

10. Saidkasimova S, Mitry D, Singh J, et al. Retinal detachment in Scotland is associated with affluence. Br J Ophthalmol. Dec 2009;93(12):1591-1594.

11. Wang J, Mitchell P, Smith W. Use of eye care services by older Australians: the Blue Mountains Eye Study. Aust N Z J Ophthalmol. 1999;27(5):294-300.

12. McFadden K, McConnell D, Salmond C, et al. Socioeconomic deprivation and the incidence of cervical cancer in New Zealand: 1988–1998. N Z Med J. 26 Nov 2004;117(1206). http://www.nzmj.com/journal/117-1206/1172/content.pdf

13. Tobias M, Yeh L-C. Do all ethnic groups in New Zealand exhibit socio-economic mortality gradients? Aust N Z J Public Health. 2006;30(4):343-349.

14. Ministry of Health. Data and Statistics National Minimum Dataset (Hospital Events) (NMDS). 2010; http://www.moh.govt.nz/moh.nsf/indexmh/dataandstatistics-collections-nmds

15. World Helath Organization. International Statistical Classification of Diseases and Related Health Problems 10th Revision Version. 2007; http://apps.who.int/classifications/apps/icd/icd10online/. Accessed 27 May, 2010.

16. Salmond C, Crampton P. NZDep2001 Index of Deprivation User's Manual, 2002. http://www.moh.govt.nz/moh.nsf/Files/NZDepfiles/$file/phi-users-manual.pdf

17. Blakely T, Pearce N. Socio-economic position is more than just NZDep. N Z Med J. 2002;115:109-111.

18. Oxford LE, McClay J. Medical and surgical management of subperiosteal orbital abscess secondary to acute sinusitis in children. Int J Pediatr Otorhinolaryngol. Nov 2006;70(11):1853-1861.

19. Tanna N, Preciado DA, Clary MS, Choi SS. Surgical treatment of subperiosteal orbital abscess. Arch Otolaryngol Head Neck Surg. Jul 2008;134(7):764-767.

20. Jeffreys M, Sarfati D, Stevanovic V, et al. Socioeconomic Inequalities in Cancer Survival in New Zealand: The Role of Extent of Disease at Diagnosis. Cancer Epidemiol Biomarkers Prev. 2009;18(3):915-921.

21. Finger F, Rossaak M, Umstaetter R, et al. Skin infections of the limbs of Polynesian children. N Z Med J. Apr 23 2004;117(1192):U847. http://www.nzmj.com/journal/117-1192/847/content.pdf

22. BPACnz. Antibiotic choices for common infection. 2009; http://www.bpac.org.nz/magazine/2009/june/antibiotics.asp#ear

Patients "falling through the cracks". The Canterbury Charity Hospital: initial progress report

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Abstract

Aim To present the early experience of establishing a community-funded and volunteer-staffed hospital in Christchurch, New Zealand. This was to provide free selected elective healthcare services to patients in the Canterbury region who were otherwise unable to access treatment in the public health system or afford private healthcare.

Methods Data were reviewed relating to the establishment, financing, staffing and running of the Canterbury Charity Hospital. Details were provided of patients referred by their general practitioners who were seen and treated during the first two and a half years of function.

Results Canterbury Charity Hospital Trust, established in 2004, completed the purchase of a residential villa in 2005 and converted it into the Canterbury Charity Hospital, which performed its first operations in 2007. By the end of December 2009, 115 volunteer health professionals and 79 non-medical volunteers had worked at the Hospital, provided a total of 966 outpatient clinic appointments, of which 609 were initial assessments, and performed 610 surgical procedures. Funding of $NZ4.3 million (end of last financial year) came from fundraising events, donations, grants and interest from investments. There has been no government funding.

Conclusions There is a substantial unmet need for elective healthcare in Canterbury, and this has, in part, been addressed by the recently established Canterbury Charity Hospital. The overwhelming community response we have experienced in Canterbury raises the question of whether the current public health system needs attention to be re-focused on unmet need. We contend that unless this occurs it might be necessary to establish charity-type hospitals elsewhere throughout the country.

The health reforms of the early 1990s introduced a "revolutionary policy of commercialisation" to the public health system of New Zealand.[1] These reforms, which divided the medical profession, had wide-ranging effects on public health services. Perhaps the most visible change was in the provision of elective hospital services.

Four years into the health reforms, "hospital waiting lists for many procedures had become longer, by as much as 50%".[2] Thereafter, patients with non-urgent conditions for whom treatment could not now be provided in the public hospital system within an arbitrary timeframe, were either refused outpatient assessment or dropped from elective waiting lists. The unmet need also became unseen.

A group of individuals in Canterbury, mostly but not exclusively health professionals, became concerned at the apparent inability of some patients to gain access to services previously available within the public health system. This growth of unmet societal need evolved despite an increase in the use of private health care[3] and notwithstanding the fact that some health practitioners were seeing patients in their private rooms free of charge. Failing to find a solution to these problems through official channels, the Canterbury group established the Canterbury Charity Hospital Trust (CCHT).

The aim of CCHT was to provide free specialist medical services, including day surgery, for patients from the Canterbury District Health Board (CDHB) region who were unable to access care through the public hospital system and who could not afford private care. The venture was based on charitable funding, a largely volunteer workforce and a utilitarian concept of treating as many patients as possible within available resources.

It was always accepted by the trustees of CCHT that the venture would not address all the unmet and apparently increasing need in Canterbury. It was, however, intended that the Canterbury Charity Hospital would not compete with either public or private healthcare providers but see only those patients who "fell through the cracks".

We present here the experience of CCHT in setting up the Canterbury Charity Hospital, and report on the staffing and patients involved in the first two and a half years of function. Some implications for healthcare in New Zealand are discussed.

Methods

CCHT initially considered a number of options for patients in the CDHB region who had limited access to elective health services. These included the use of operating theatre and outpatient clinic facilities in Christchurch public and private hospitals during "down-time". This option was found to be impractical for a number of reasons, but mainly due to the difficulty of matching volunteer staff time with service resource availability. It became obvious that a standalone facility was needed. Accordingly, a large, old, residential villa in the Christchurch suburb of Harewood was purchased by CCHT and converted into the Canterbury Charity Hospital.

All funding for the venture has come from the local community. A local newspaper has facilitated the collection of individual donations from the public. CCHT will not consider accepting any government funding in order to retain its independence and avoid the administrative/bureaucratic burden that such funding would inevitably incur.

The design of this facility was provided *pro bono* by a local architectural firm. Renovations were undertaken by a major contracting company and a number of subcontractors at much reduced costs. The architects and the contractor subsequently won regional and national awards for their work on the Canterbury Charity Hospital based on the opinions of their peers.

CCHT is run by four trustees and operates under a Charitable Trust deed. It is supported by a number of committees including a Clinical Board, which oversees the quality and safety of all clinical services. This Board has representation from both primary and secondary care medical practitioners, nurses and lay people. There is also a committee that organises major fundraising events. Legal and accountancy services are provided *pro bono* by local firms.

Canterbury Charity Hospital is staffed by a large number of non-clinical volunteers some of whom provide a regular, recurring commitment, whilst others provide their expertise less regularly according to personal circumstances. They assist with, for example, administration, cleaning, gardening and transport. The current volunteer clinical staff list consists of physicians, surgeons, anaesthetists, nurses and operating theatre technicians, all *pro bono* (see Results). There is also a skeleton staff of two full-time paid equivalents to cover clinical, management and technical functions and a part-time consultant for marketing, public relations and fundraising.

All patients must be referred by their general practitioners (GPs), who are the gatekeepers to the service. Four criteria must be met for acceptance of a patient for treatment:

- The patient must be unable to access a service they require through the public hospital system;
- He/she must be unable to pay for private care, have no medical insurance, and not be entitled to Accident Compensation Corporation funded treatment;
- A signed confirmation of eligibility to treatment at Canterbury Charity Hospital is required from the GP and the patient; and
- The GP must also confirm that the patient's medical condition is affecting their quality of life and/or ability to work.

Canterbury Charity Hospital provides a free day surgical service and medical outpatient consultations for some clinical conditions. The list of clinical services has changed with time depending on the types of referral, and volunteer staff and resource availability. GPs and the public can obtain an updated list of available services on the CCHT website (www.charityhospital.org.nz).

Since there are no overnight stay facilities in the Canterbury Charity Hospital, limited respite care after day surgery has recently been made available *pro bono* through other providers. Where special investigations are required, GPs are requested to organise these but when this has proved impossible they are organised by Canterbury Charity Hospital staff. A limited number of radiological and necessary special investigations are provided *pro bono* by other organisations.

After all medical and surgical procedures, patients have access to a free telephone number for advice about immediate problems. All patients are followed up by telephone call and most are offered follow-up clinic appointments.

Results

The concept of a free, volunteer-staffed, day surgical service for those in the CDHB region who could not otherwise obtain access to care was trialled first by performing operations in the mobile surgical bus, which provides a national surgical service.[4] The first operating list in the new Canterbury Charity Hospital was performed on the 31 August 2007, 2 months after extensive renovations were completed.

The fully equipped Canterbury Charity Hospital was officially opened by the Anglican Bishop of Christchurch, the President of the Royal Australasian College of Surgeons and the Deputy Head of the New Zealand Nurses Organisation in October 2007 (Figures 1 and 2).

Figure 1. The Canterbury Charity Hospital as viewed from Harewood Road

Figure 2. The Canterbury Charity Hospital Operating Theatre

A regular day surgical service was not implemented until the following year. The timeline of major events is shown in Table 1.

Table 1. Timeline of major events for Canterbury Charity Hospital (CCH)

July 2004	CCH Trust established
April 2005	First day surgery procedures performed on mobile surgical bus
May 2005	First major fundraising event (Christchurch Town Hall concert)
August 2005	Purchase of old villa at 349 Harewood Road, Christchurch
August 2006	Contract signed with main contractor to renovate old villa
June 2007	Renovations of old villa into CCH complete
August 2007	First operations at CCH—General Surgery service started
October 2007	Official Opening of CCH
August 2008	Gynaecology and Cardiology services started
Sept 2008	First cataract operations performed
Nov 2008	Podiatry service started
Dec 2008	Dermatology service started
Feb 2009	Plastic and Hand Surgery services started
March 2009	Neurology service started
June 2009	Vascular Surgery service started
July 2009	Orthopaedic Surgery service started

All data are summarised as at 31 December 2009.Table 2 shows the increase in the number of patients seen and treated annually at the Canterbury Charity Hospital since

its opening in 2007, and prior to that in the mobile surgical bus or in specialists' private rooms. There were a total of 609 initial outpatient appointments and 357 follow-up appointments, and 610 surgical procedures were performed.

Table 2. Numbers of outpatient appointments and surgical procedures at Canterbury Charity Hospital

Calendar year	2005-2006	2007	2008	2009
Initial appointments	11	24	199	375
Follow-up appointments	0	11	125	221
Total number of treatments	11	11	220	368

Of the 609 initial appointments, data were collated for 597. The total number of patients seen at these 597 initial outpatient appointments was 575 (56% male; 44% female) with a mean age of 51.6 years, (SD=19.1, range 2 to 92 years). Of this group, 466 patients (56% male; 44% female; 73.3% <65 years) received treatment with 25 further patients awaiting treatment. Some (65, 13.9%) received two or more treatments.

The group who received treatment were significantly younger than those who did not (50.6 years compared to 55.9 years, Independent t-test p=0.010) but the percentage of males did not differ between the groups; 52% for those not treated compared to 56% for those who were (Chi-square test, p=0.395). For the rest, either no treatments were required or they were deemed unsuitable because: the treatments they needed were outside the scope of what Canterbury Charity Hospital could provide; they had extensive medical comorbidities; or, their temperaments or social circumstances were inappropriate for day surgery.

Where co-morbidities were a relative contraindication to treatment, patients were usually seen at an anaesthetic and/or appropriate medical assessment clinic before a final decision was made about management.

In an analysis of the last 300 patients seen at Canterbury Charity Hospital, 20% stated that they were currently employed, 41% indicated that they were unemployed, and 39% did not specify their employment status.

Table 3 provides a breakdown of medical and surgical procedures by specialty provided at Canterbury Charity Hospital. The most frequently performed procedures were for the treatment of groin and umbilical hernias, haemorrhoids, pilonidal sinuses, cataracts, and tubal ligations for sterilization. There were three surgical complications: a local infection following repair of an umbilical hernia, which responded to antibiotic treatment; a haematoma in an inguinal hernia repair which required evacuation; and, insertion of a lens of incorrect refraction, requiring replacement surgery. Overnight respite care after surgery was required because of social circumstances for three patients.

Table 3. Breakdown by specialty of medical and surgical procedures performed at Canterbury Charity Hospital

Calendar Year	2005–2006	2007	2008	2009
Medical	0	0	8	22
Dermatology	0	0	3	4
General Surgery	7	11	198	221
Ophthalmology	4	0	4	42
Gynaecology	0	0	5	36
Orthopaedic/Hand	0	0	0	22
Podiatric	0	0	0	13
Plastic	0	0	2	8

The clinical and support staff complement involved in providing service through Canterbury Charity Hospital is shown in Table 4. Whereas the vast majority of staff are resident in Canterbury, valuable service has also been provided by medical personnel from Invercargill, Nelson, and Hawke's Bay. Many staff beyond those indicated in Table 4, both medical and non-medical persons, have offered their time and may be called upon as services provided by the Canterbury Charity Hospital continue to expand.

Table 4. Staff Composition at Canterbury Charity Hospital

Staff type		Numbers ever worked since 2005	Numbers working in 2009
Volunteers	Anaesthetists	19	13
	Nurses	60	33
	Physicians	8	7
	Surgeons	18	18
	Technicians	8	6
	Non-Medical	73	33
Employees	Part Time	2	2
	Full Time	1	1

CCHT and the CDHB signed a Memorandum of Understanding in April 2008. This facilitated cooperation between the two organisations in a number of areas such as staff availability, training opportunities and use of resources in emergency situations. CCHT shares some costly or irregularly used resources, such as ophthalmological equipment, with other healthcare organisations.

It has been a basic tenet of CCHT that the standards of care for all services will meet and/or exceed those expected of the New Zealand public health system. Medical and surgical procedures are performed and supervised by medical, surgical and nursing specialists, working within their vocational scopes as listed with the Medical Council of New Zealand. CCHT has two independent credentialing officers, and all medical and nursing staff are subjected to a strict credentialing process, the outcomes of which are monitored by the Clinical Board.

The trustees are, of course, concerned with the long-term financial viability and sustainability of the venture. Accordingly, they intend to maintain strict expenditure disciplines and ensure that the limited available resources are expended on clinical services. CCHT is working to create a reserve fund that will provide an income to protect the long-term viability of the Canterbury Charity Hospital.

The total funds raised from 2005 until the end of the last financial year were $NZ4.3 million and came from: CCHT organized fundraising events (3.5%); donations from individuals, businesses and community groups (60%); grants from charitable trusts (33%); and interest from investments (3.5%). The initial costs of purchasing, renovating and equipping the Canterbury Charity Hospital were $NZ2.3 million. Since 2006, a further $NZ400,000 has been spent on equipment.

Many organisations and individuals donated their time, expertise and resources *pro bono* or at highly discounted rates, in order to keep the setup costs to a minimum. All major equipment was purchased from non-government grants and most of the furniture and fittings were donated. Since the Canterbury Charity Hospital became operational in July 2007, the annual operating budget has been $NZ350,000 and, each year, a progressively increasing percentage of income has been spent on patient care. From 1 April 2009 to 31 December 2009, 75% of the budget directly related to patient treatment expenses.

Whilst the primary objective of this endeavour remains the mitigation of unmet clinical need, a secondary objective has evolved—the teaching of medical and nursing staff and students. Partial or complete loss of elective surgery from the public hospitals has left a void in the training of medical[5] and nursing personnel in these procedures. This is now being addressed, at least in part, by staff and patients in the Canterbury Charity Hospital.

Junior doctors have assisted with outpatient clinics and procedures; surgical trainees have learnt procedures that are now rarely performed in the public hospital system. Third year undergraduate nurses have observed the patient clinical pathway from initial assessment to final discharge. Postgraduate nurses have gained clinical experience in their current specialty area or in other specialty areas. A large number have gained clinical experience in the perioperative setting. CCHT supported a number of volunteer postgraduate nurses, who were either retired or not employed as nurses, to regain or maintain their registration status. Such teaching and mentoring, as with the clinical services themselves, is provided *pro bono*.

Discussion

As noted by Gauld, the new system introduced by the New Zealand health reforms of 1993 "performed poorly, in keeping with problems of market failure endemic in health care".[6] The need for the Canterbury Charity Hospital arose out of this, and the data shown here suggests the need is ongoing. For example, it is unusual now for patients with haemorrhoids, inguinal hernias, and similar problems, without associated complications, to receive elective treatment in the Christchurch public hospitals, whereas patients with these afflictions were routinely operated on in the public system in 1970s and 1980s.

Of particular note, 73.3% of patients treated in the Canterbury Charity Hospital were under 65 years of age (mean age 50.6 years) and 41% were unemployed (see Results). These data suggest that there are many patients within the "working age" population in Canterbury who are unable to gain access to the public hospital system and are considered by their GP to have their quality of life and/or ability to work compromised by their medical or surgical disorder. This fact, along with the obvious point that treating such disorders early, before they become severe or complications ensue, raises the issue of whether the health reform-based restrictions of care in the public health system are cost-effective.[7,8]

Moreover, our patient eligibility criteria selectively determine the clinical and economic characteristics of the patients referred by Canterbury GPs. This population may, therefore, represent only a proportion of the existing unmet need. The fact that the Canterbury Charity Hospital saves money for the Government provides the potential for moral hazard (i.e. the incentive to save money overcomes the desire to address the healthcare needs of the whole population). It is hoped that charity hospitals do not become the default back-up, and that attention is re-focused on the provision of an effective universal public healthcare system.

The experience of CCHT is that there is excellent, sustained and sufficient goodwill within Canterbury to establish and run a charity hospital. In this regard CCHT may prove to be a template for the establishment of similar hospitals in centres elsewhere in the country. Indeed, the Auckland Regional Charity Hospital is already functioning,[9] and a somewhat similar venture has been mooted in Dunedin. Regarding practicalities, it should be noted that substantial time, funding and effort were required to establish a purpose-built hospital.

We suggest that the experience of CCHT should be the catalyst for a debate on the future of New Zealand's healthcare systems. In particular, should we now accept that the current public healthcare system is as efficient and cost-effective as is possible— in which case the need for charity-like hospitals will become necessary in cities and towns throughout the country? Alternatively, is it possible to improve the structure and functioning of the public healthcare systems, such as by reducing the burgeoning bureaucracy and the associated escalating costs.[10,11]

As far as the future of the Canterbury Charity Hospital is concerned, the trustees of CCHT intend to extend the current services according to the evolving unmet needs of the Canterbury community. In the immediate future this may include dental treatment.

Conclusion

Establishing the Canterbury Charity Hospital has proved to be a highly satisfactory endeavour as gauged by the large and increasing number of patients treated, the continuing expansion of clinical services, the high level of continued community financial support, and the substantial number and sustained commitment of the volunteer workforce. It illustrates some of the current deficiencies in the public health system that need to be addressed. If not, charity-type hospitals might be needed elsewhere throughout the country to mitigate some of these deficiencies.

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References:

1. Easton B. The New Zealand health reforms of the 1990s in context. Appl Health Econ Health Policy. 2002;1:107-12.
2. Hornblow A. New Zealand's health reforms: a clash of cultures. BMJ. 1997;314:1892-4.
3. Kelsey J. The New Zealand Experiment. 1996; p.216. Auckland University Press.
4. http://www.mobilesurgical.co.nz/company/overview.asp
5. New Zealand National Board, Royal Australasian College of Surgeons. Extending Surgical Training Opportunities in New Zealand. Submission to New Zealand Minister of Health. 9th April 2009.
6. Gauld RD. Big bang and the policy prescription: health care meets the market in New Zealand. J Health Polit Policy Law. 2000;25:815-44.
7. Lin A, Stiven P, Bagshaw P, Connor S. Cholecystectomy following acute presentation to a major New Zealand metropolitan hospital: change to the timing of surgery is needed. N Z Med J. 2006;119:U2104. http://www.nzma.org.nz/journal/119-1239/2104
8. Casillas RA, Yegiyants S, Collins JC. Early laparoscopic cholecystectomy is the preferred management of acute cholecystitis. Arch Surg. 2008;143:533-7.
9. http://www.aucklandcharityhospital.org/
10. New Zealand National Party Health Discussion Paper. Better. Sooner. More Convenient. September 2007, pp 34 & 35.
11. Woolhandler S, Himmelstein D. Competition in a publicly funded healthcare system. BMJ. 2007;335:1126-9.

Health, inequality and the politics of genes

Patrick M Whittle

Abstract

Research into the possible genetic basis of health inequalities between different ethnic or racial groups raises many scientific, ethical and political concerns. Proponents of such research point to the possible benefits for marginalised groups of understanding genetic influences on health outcomes; opponents indicate the potential social costs, citing historical use of Darwinian concepts to explain and justify inequalities between different peoples. Many health researchers may avoid the subject due to its potential for controversy—e.g. the recent media furore over the so-called 'warrior gene', and its apparent genetic explanations for negative health and social statistics among Māori. This article argues for a more nuanced account of the evolutionary history of marginalised groups such as Māori, one that accepts the possibility of relevant genetic differences between sub-populations, but which also acknowledges genuine ethical and political concerns. Such an account may assist health researchers in addressing the politically sensitive subject of 'race' and social inequality.

That health inequalities exist between ethnic or racial sub-populations in New Zealand is incontrovertible; according to recent figures, for example, the average life expectancy of Māori is 8 years shorter than non-Māori.[1] Explaining these inequalities is problematic, especially given the correlation with other social disparities (e.g. wealth, education), and the consequent need to consider poverty, deprivation and, in the case of Māori, the potential effects of a century and a half of colonisation.

Historically, of course, another explanation was often advanced for the obvious inequalities between different peoples: that this was simply the survival of the fittest, in the Darwinian struggle for existence. Indeed, Darwin himself used the declining Māori population in the 19th Century to illustrate the seemingly inevitable fate (i.e. extinction) of 'less-favoured' human races.[2]

Recent genetic research (notably, in New Zealand, the so-called 'warrior gene' and its reputed link to violent behaviour among Māori) has raised concerns that these discredited social Darwinian theories may be resurrected in modern genetic guise.[3]

By contrast, some biomedical researchers have argued that failure to acknowledge genetic differences between sub-populations may prove detrimental to the health and well-being of minority groups such as Māori.[4] Scientific and ethical reservations about the original 'warrior gene' research are widespread.[3,5,6] Little attention, however, has been paid to how health researchers could or should approach a topic as politically fraught as this; the possibility that genetic differences between sub-populations may have health or social consequences. Indeed, many health professionals may choose to simply avoid the subject due to its potential for controversy, and thus fail to provide valuable input into the social and political debate on the causes of health inequalities; debate that, in turn, frames possible policy responses to these inequalities.

This discussion will address the key questions: is an evolutionary approach to health inequalities likely to be of benefit to marginalised groups such as Māori? And: do we have reasonable grounds for concern about any such approach?

Both questions will be answered, with qualifications, in the affirmative. This will then allow a range of suggestions for how biomedical and health researchers may best deal with the politically sensitive subject of 'race' and social inequality. One major conclusion will be that an overly simplistic view of evolutionary theory (and genetics) is often presented in the debate.

A more nuanced understanding of Darwinian theory, especially of gene-environment interaction and the effects of historical-cultural processes, may prove beneficial in both improving health outcomes for marginalised groups and in allaying the fears of those who remain suspicious of Darwinian interpretations of human behaviour.

Human sub-populations or 'races' differ phenotypically; in skin colour, hair texture or facial features. One of the most contentious issues in the human sciences (indeed, in science and politics generally) is whether such differences go deeper than these observable physical characteristics—to behaviour, say, or to cognitive abilities.[7–9] An apparent consensus view among social theorists holds that human races are not biologically real, with some theorists arguing further that 'race talk' should be eliminated entirely from public discourse.[10–12] Research that openly challenges this majority opinion on race is subject to severe censure.[13]

This emphasises the fact that 'race' is a contested concept, and that it carries with it a great deal of historical baggage; most especially, the folk-biological beliefs used to justify racial bigotry.[10,12,14] A less-loaded alternative, such as 'ethnic group', is therefore often preferred. Nevertheless, race and ethnicity are not always synonymous, and a biological concept of race is sometimes of pragmatic value.

In New Zealand, for instance, identifying the 'race' of older human remains (e.g. those possibly from pre-European burial sites) may have cultural significance for Māori , due to spiritual links to ancestors;[15] at the same time, actual biological ancestry is integral to the cultural concept of whakapapa, or lineage. In such contexts, then, the physical/biological associations of 'race' may overlap with the social/cultural associations of 'ethnicity'.

In addition to possible inadvertent conflation of concepts such as race and ethnicity, a discussion such as this runs the risk of portraying minority groups, such as Māori , as 'Other'; for example, those who seemingly vary 'abnormally' from the majority population.

By contrast, genetic explanations for the high rates of melanoma among Pākehā (non-Māori) New Zealanders, for example, would be considered unremarkable; thus, while the focus here is primarily on the evolutionary genetics of Māori , it is important to acknowledge that evolutionary genetic reasoning applies to all population sub-groups. (The relative size of these sub-groups is also a factor; genetic traits associated with a small number of Pākehā families, for example, may be concealed within the larger Pākehā population; traits associated with a similar number of Māori whānau, however, may skew genetic data within the comparatively smaller Māori population.)

To return to the wider question of human difference, then: at an extreme, the consensus view on race suggests that no important differences exist between human sub-populations. Substantial medical evidence, however, links certain ailments with specific sub-groups: Sickle-cell anaemia (and partial immunity to malaria), for example, indicates West African heritage; Tay-Sachs disease is restricted almost exclusively to those of Ashkenazi Jewish descent; lactose intolerance is a feature of some sub-populations but not others; and genetic ancestry is relevant to the success or otherwise of organ transplants.[14]

Genetic information on human group differences is accumulating rapidly. However, genetic research into, say, group differences in medical conditions could appear to be at one end of a slippery slope; if biochemical differences exist between sub-populations, why not behavioural differences? And if sub-populations differ behaviourally, could racial inequalities be explained in simple genetic terms? For example, could we put the high rates of violent offending in Māori communities down simply to 'warrior genes'? Or obesity to 'thrifty genes'?

According to recent media reports, this is exactly what genetic researchers have suggested; that, say, "Māori are genetically wired to commit acts of violence". [16-17] This highlights genuine concerns about the socio-political implications of genetic research. If simplistic genetic explanations for complex social issues become established in the public mind—for example, the belief that Māori are inherently and ineluctably violent—then political support for ameliorative social policies could be eroded. Why waste taxpayer dollars on problems that cannot be fixed?

Two contrasting views are apparent among those researchers who accept the likelihood of meaningful genetic differences between human sub-populations: That the potential for social harm arising from genetic investigations are such that we may be better off abandoning these lines of enquiry altogether;[18] or that scientists' only responsibility is to the truth, no matter how politically unpalatable that truth may turn out to be.[19] Medical and health science appears caught between these two extremes.

On the one hand, health intervention that disregards possible evolved or genetic differences between sub-populations also risks ignoring factors that may help improve people's lives. On the other hand, if genetic or evolutionary research is likely to revive racial stereotypes, or cause people to view between-group inequalities as unchangeable, then the potential for wider social harm may outweigh the more restricted health benefits. How then could or should health practitioners best deal with the various scientific, political and ethical aspects of a Darwinian approach to group difference?

The 'warrior gene' controversy, which first erupted in 2006 [4,16] and re-ignited in late 2009,[3,20] illustrates the social cost/benefit dilemma posed by human genetic research. The research itself focused on monoamine oxidase (MAO) genes; those apparently linked to various behavioural disorders, including depression, mental retardation and aggression.[3,4]

A gene sub-type, MAO-A, had earlier been dubbed the 'warrior gene' due to its apparent association with aggressive behaviour in Rhesus macaque monkeys,[5] and this label was subsequently applied to the equivalent gene polymorph in humans, in relation to evolutionary speculation about its apparent prevalence among Māori

males.[21] In August 2006, this was reported in the popular media as a claim that contemporary Māori carry a 'warrior gene', making them prone to violence, criminality and risky behaviour.[16]

However, according to the epidemiologists at the centre of the controversy, Rod Lea and Geoffrey Chambers, their research agenda focussed not on aggressive traits in Māori but on the association between MAO-A and addiction; in particular, the relationship between ethnic variation in MAO-A frequency and differential patterns of alcohol and tobacco dependence. Their interpretation of the genetic data indicated that the frequency of the relevant allele was almost twice as high among Māori as Caucasian males.[4] This interpretation of the data, and in particular the small sample upon which Lea & Chambers' extrapolations were based, has been extensively criticised,[3,5,6] and the purpose here is not to legitimise this particular study.

Instead, the focus is on the medical question: could genetic information on alcohol/tobacco dependence be used in developing more appropriate treatments and better health outcomes for Māori? If so, this would illustrate one side of the dilemma suggested above: That genetic research may have positive health benefits for marginalised groups such as Māori. How then, with regard to the Lea & Chambers' study, did this benign-seeming aim become politicised?

In providing an evolutionary explanation for the apparent higher frequency of MAO-A in modern Māori, Lea & Chambers developed a 'warrior gene hypothesis', speculating that the gene may have been positively selected during the ocean voyaging and inter-tribal wars that supposedly characterised the ancestral Polynesian migrations across the Pacific. They supported this hypothesis by arguing that Māori martial prowess was historically well-recognised and that "reverence for the 'warrior' tradition remains a key part of Māori cultural structure today".[4]

While Lea & Chambers denied that this provided a biological explanation for present-day social dysfunction, their argument implied that Māori had evolved in a manner that made them genetically (and behaviourally) different from other populations. Again, the historically- and politically-dubious nature of this speculation has been emphasised in critical reviews.[3,5,6] The relevant point here is the implication that Māori are in some way inherently more aggressive than non-Māori. This illustrates the other side of the genetic difference dilemma: That any such conclusion may have a deleterious social or political impact on efforts to address high levels of violence in Māori communities.

An epidemiological study into the MAO-A30bp-rp variant, its associations with tobacco and alcohol dependence, and the variation in this gene allele's frequency between different ethnic groups would probably excite little media or political controversy, while still providing useful information for the design of diagnostic, preventative or treatment regimes. Much of the furore surrounding the Lea & Chambers' hypothesis, therefore, appears due to use of the attention-grabbing term 'warrior gene'.

For the public to believe that Māori carry a 'warrior gene' is, potentially, socially harmful; accepting the possibility of some genetic influences on tobacco/alcohol dependence among Māori appears less so. Nor is it here immediately obvious why an evolutionary explanation for apparent differences in gene frequency is necessary.

From a health perspective, the (apparent) fact that such differences exist is the relevant issue. With hindsight, or in relation to future studies, such considerations should be taken into account.

At the same time, however, an evolutionary perspective may be illuminative. A fuller understanding of the ancestral Polynesian migrations across Asia and into the Pacific may provide important insights into contemporary Pacific peoples' health and social well-being. For example, can we use commonalities/dissimilarities in health outcomes to more fully understand the causes of rising diabetes rates among disparate Pacific peoples (which appear due mainly to social and cultural factors)[22] or differences in cancer prevalence (which may combine cultural and genetic factors)? By its very nature, an evolutionary or genetic approach to human health conditions is reductionist; an attempt to describe aetiologically complex phenomena (such as behavioural conditions) in terms of other phenomena, at a simpler or more fundamental level (e.g. gene expression).

While such reductionism is necessary to distinguish possible underlying causes of observed health outcomes, it may bring with it a tendency to over-simplify human evolutionary processes, or to merely pay lip-service to the inter-relationship between genes, environment and culture. The 'warrior gene hypothesis', for example, conflates behavioural traits (risk-taking and aggression), historical-cultural practices (the supposed Māori warrior tradition) and contemporary social facts (health and social inequalities). Nonetheless, a more nuanced Darwinian approach could reveal useful insights into contemporary health and social issues, and highlight areas where evolutionary or genetic ideas may complement rather than challenge more standard sociological explanations for health or status inequalities.

The title of author Alan Duff's *Once Were Warriors* epitomises an intuitive (but suspect) notion that violence in contemporary Māori communities is a legacy of the historical Māori warrior culture. The 'warrior gene hypothesis' similarly reflects this notion, by pointing to possible genetic influences on the development of this historic culture,[4] and thereby implying a genetic basis for modern social dysfunction.[3] This, in turn, also suggests genetic determinism; that naturally selected behavioural traits have been responsible for shaping later cultural developments or present-day social behaviour. Is it the case, then, that evolutionary accounts of human behaviour inevitably imply some form of genetic determinism, the notion that humans (or distinct human groups) are, to some extent, pre-programmed to behave in certain ways?

In the case of Māori, evolutionary theory would certainly predict that genetic bottlenecks (such as small founding populations) and selective pressures over the millennia could have uniquely moulded the Māori genome. For example, it is plausible that personality traits (such as those described by the inadvertently apt acronyms OCEAN or CANOE: Openness to experience, Conscientiousness, Extraversion, Agreeableness, and Neuroticism) may have a statistical distribution among Māori that differs from other sub-populations.

Those spear-heading each new migration (such as that to Aotearoa/New Zealand), for instance, were possibly less risk-averse than those choosing to stay behind; indeed, this is perhaps true also of later Pākehā colonists, with the supposed Kiwi 'can-do' attitude potentially arising from the greater openness and resourcefulness of the early

pioneers. Thus it is conceivable that certain behavioural or personality traits, concentrated in the initial founding populations, may have influenced the development of any subsequent cultural psyche.

This argument, though, can be qualified with reference to MAO-A. As this gene has been linked to risk-taking behaviour, it initially appears plausible that it was positively selected during the Polynesian expansion across the Pacific—hence the gene's apparent frequency among Māori. In the relevant studies, however, the highest frequencies of the so-called 'warrior gene' are not found among Māori but, rather, among Chinese males.[4] This could immediately cast doubt on the evolutionary speculation behind the 'warrior gene hypothesis'.

Alternatively, one could speculate that, just as risk-taking traits may have proved advantageous in island-colonising ancestral Polynesian environments, so too may they have been in the different environments faced by ancestral Chinese. In a modern context, certainly in New Zealand, risk-taking traits may continue to be advantageous in the commercial settings stereotypically associated with Chinese; in an economically-deprived environment, stereotypically associated with many Māori, these self-same characteristics may prove disadvantageous, especially if they are expressed in drug-taking, alcohol abuse or criminal behaviour. The social consequences of such traits (if any exist) are contingent on environment.

Furthermore, the 'warrior gene hypothesis' is premised on the notion of positive selection of advantageous behavioural traits. In the successful Polynesian expansion across the Pacific, such traits would undoubtedly have included altruism/self-sacrifice, loyalty and intense cooperation. Paradoxically, these same traits also underlie another ultra-social human behaviour: warfare. Yet while intra-group cooperation is intrinsic to all human societies, inter-group conflict is a latent (but not an inevitable) human response to scarcity of, or competition for, resources. Māori cultural developments in New Zealand illustrate this.

In the period following the initial founding migration(s), cultural practices reflected changing ecological pressures, as the early Māori colonists adapted to the large and resource-rich landmass of New Zealand. The tribal culture most popularly associated with the Māori warrior tradition developed later, in the 15th and 16th Centuries, possibly as a result of population growth and exhaustion of initial food sources (e.g. large bird species such as moa).[23] Tribal organisation allowed more diverse and intensive extraction of resources, including hostile competition with other groups. Thus, while evolved behavioural traits may have influenced the resultant Māori culture, inter-tribal conflict (and the so-called warrior tradition) is more appropriately explained as a cultural response to environmental conditions.

In addition, while the specifics of any Māori warrior tradition are unique, the behaviour is not; tribal conflict is common to most (if not all) human cultures. For example, the earliest plausible date for the arrival of Māori in New Zealand, around 1000 AD, was contemporaneous with the Viking and Norman invasions of Britain; and this only centuries after the Anglo-Saxon warrior society first became established in the land that now bears its name, England (i.e. at roughly the same time that ancestral Polynesians established themselves in Hawaii and Easter Island).[24]

Thus the first Europeans to land in New Zealand, the British seaman under James Cook, were as much heirs to a warrior tradition as were the indigenous peoples with whom they came into contact. It is worth noting that Cook's British government was then engaged, closer to home, in subduing the warlike Scottish Highlanders and the Irish, both of whom were still regarded as distinct 'races' in Darwin's time.[2]

To reverse the genetic gaze away from Māori, beneath the apparent homogeneity of the Pākehā population potentially lurks genetic variation traceable to distinct sub-populations in the British Isles or elsewhere in Europe. The point is that, while the Māori society first encountered by Europeans appeared to be a warrior one, so too were contemporary or near-contemporary European societies; why then does it seem appropriate to posit simplistic genetic explanations for the former (i.e. Māori) culture but not the latter?

Nevertheless, Europeans did have one crucial genetic advantage over Māori: partial immunity to disease. Evolutionary theory explains this fact. Infectious diseases had been endemic for thousands of years in the densely populated societies of Eurasia; thus genetic changes in body chemistry that increased immunity to disease were under strong selective pressure in European populations, but not in less dense indigenous populations, such as in pre-contact New Zealand.[7] The effects of these basic genetic differences were catastrophic for indigenous peoples. Darwin, for example, described disease as one of the main causes of the "notorious" decline in the Māori population in the 19th Century, such as an estimated 33 percent fall in Māori numbers in the decades after 1850.[2]

The social dislocation wrought by this rapid population decrease must have been equally devastating (by analogy with the more well-reported influenza pandemic in Samoa in 1918,[25] those most susceptible to these diseases were Kaumatua, tribal elders whose leadership skills were likely to have been lost at precisely the time they was most needed). While such 'fatal impact' interpretations of post-contact New Zealand history are open to debate,[26] a good starting point for any evolutionary/genetic explanation for observed social inequalities between Māori and non-Māori in modern New Zealand could be with (the historical consequences of) evolved resistance to disease: A negative feedback of dramatic population decline, weakening social cohesion, adoption of European vices (alcohol and tobacco), and increasing marginalisation as European colonist numbers grew.

Evolutionary and genetic factors are, therefore, potentially relevant to contemporary health issues. On analogy with evolved immunity to disease, there may well be genetic differences in alcohol or tobacco tolerance between sub-populations (including sub-groups within the Pākehā population). Relevant genetic research, therefore, could assist attempts to address the health and social consequences of, say, drug dependence. A proviso here is that, while reductionism could indicate the possible genetic basis for behavioural traits, any appropriate conclusions could only be draw in reference to higher level cultural and historical phenomena. A broader genetic, cultural, historical approach is therefore necessary to fully understand the causes of different health and social outcomes for different peoples.

Sociological explanations for the health and social inequalities faced by groups such as Māori highlight poverty, deprivation and the impact of colonisation. This is not incompatible with an evolutionary approach. The environment in which the genome

develops, and in which any genetic influence is expressed, is of paramount importance. A broader perspective on Māori evolutionary history, for example, indicates that the Māori genome has not determined cultural and social development, rather it has allowed flexible responses to changing environmental conditions. Yet for the past 160 years, Māori have been marginalised; the resultant deprived environment, then, is the one in which (possible) genetic influences have been and are being expressed.

Popular (mis)interpretations of the results of genetic research, however, have the potential to erode support for ameliorative social policies. Awareness of these issues, and of the need to provide a broader cultural and historical perspective, may allow gene-based health research to avoid the political pitfalls highlighted by the 'warrior gene' controversy. Political opposition to genetic research, too, may be misplaced.

Whether the negative social outcomes of groups such as Māori are the result solely of environment, or of a combination of environmental and genetic factors (as seems increasingly likely), our health and social goals remain the same: Improving these groups' social and economic situation.

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References:

1. Statistics New Zealand; 2008. http://www.stats.govt.nz/browse_for_stats/health/life_expectancy/nzlifetables_mr05-07.aspx
2. Darwin C. The Descent of Man and Selection in Relation to Sex. (New edition.) London: Murray, 1901.
3. Hook GR. "Warrior genes" and the disease of being Māori. MAI Review, 2009: 2. http://www.review.mai.ac.nz/index.php/MR/article/view/222/243
4. Lea, R, Chambers, G. Monoamine oxidase, addiction, and the "warrior" gene hypothesis. N Z Med J. 2007,120(1250). http://www.nzma.org.nz/journal/120-1250/2441/
5. Merriman T, Cameron V. Risk-taking: behind the warrior gene story. N Z Med J. 2007;120(1250). http://www.nzma.org.nz/journal/120-1250/2440/
6. Crampton P, Parkin C. Warrior genes and risk-taking science. N Z Med J. 2007;120(1250). http://www.nzma.org.nz/journal/120-1250/2439/
7. Diamond J. Guns, Germs, and Steel: The fates of human societies. New York: WW Norton; 2005.
8. Flynn J. Where Have All the Liberals Gone?: Race, class and ideals in America. Cambridge: Cambridge University Press; 2008.
9. Herrnstein R, Murray C. The Bell Curve: Intelligence and class structure in American life. New York: Free Press Paperbacks; 1994.
10. Mallon R. Race: Normative, not metaphysical or semantic. Ethics, 2006;116/3:525-551.
11. Gannet L. The biological reification of race. British Journal for the Philosophy of Science, 2004;55:323-345.
12. Appiah K. Why there are no human races. In: Sober, E, editor. Conceptual Issues in Evolutionary Biology (3rd edition). Cambridge, MA: MIT Press; 2006.
13. Barash D. Review of JP Rushton, Race, 1994. In: Animal Behaviour, 1995; 49/4:1131-3.
14. Hacking I. Why race still matters. Daedalus. Winter 2005, p102-116.

15. Cox K, Tayles N, Buckley H. Forensic Identification of "Race": The issues in New Zealand. Current Anthropology, 2006; 47/5:869-874.

16. 'Warrior' gene claim slammed by Māori. Christchurch: The Press, 9 August, 2006.

17. Scientist debunks 'warrior gene'. Auckland: New Zealand Herald, September 12, 2009. http://www.nzherald.co.nz/nz/news/article.cfm?c_id=1&objectid=10596821

18. Kitcher P. Does 'race' have a future? Philosophy and Public Affairs, 2007; 35/4:294-317.

19. Kanazawa S. If the truth offends, it's our job to offend. Times Higher Education Supplement. December 15, 2006:14.

20. Maori don't have warrior gene: research. Melbourne: The Age, September 11, 2009. http://news.theage.com.au/breaking-news-world/maori-dont-have-warrior-gene-research-20090911-fk8w.html

21. Hall D, Green M, Chambers G, Lea R. Tracking the evolutionary history of the warrior gene in the South Pacific. 11th International Human Genetics Meeting, Brisbane, Australia; August 6–10; 2006. Abstract at http://www.ichg2006.com/abstract/843.htm

22. Foliaki S, Pearce N. Prevention and control of diabetes in Pacific people. BMJ. 2003; 327(7412):437-439. http://www.jstor.org/stable/25455334

23. King M. The Penguin History of New Zealand. Auckland: Penguin Books; 2003.

24. Stone L, Lurquin P. Genes, Culture, and Human Evolution: A synthesis. Oxford: Blackwell Publishing; 2007.

25. Influenza hits Samoa. Ministry for Culture and Heritage; 2009. http://www.nzhistory.net.nz/media/photo/influenza-pandemic-hits-samoa

26. Belich, J. Making Peoples: A history of the New Zealanders, from Polynesian settlement to the end of the nineteenth century. Auckland: Penguin Press; 1996.

Integrated systems to improve care for very high intensity users of hospital emergency department and for long-term conditions in the community

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Abstract

Adult patients who are very high intensity users of hospital emergency departments (VHIU) have complex medical and psychosocial needs. Their care is often poorly coordinated and expensive. Substantial health and social resources may be available to these patients but it is ineffective for a variety of reasons. In 2009 Counties Manukau District Health Board approved a business case for a programme designed to improve the care of VHIU patients identified at Middlemore Hospital. The model of care includes medical and social review, a multidisciplinary planning approach with a designated 'navigator' and assertive follow-up, self and family management, and involvement of community based organisations, primary care and secondary care. The model has been organised around geographic localities and alongside other initiatives. An intermediate care team has been established to attend to the current presenting problems, however the main emphasis is on optimising ongoing care and reducing subsequent admissions especially by connecting patients with primary health care. This whole process could be driven by the primacy care sector in due course. The background and initial experience with implementation are described.

Hospital emergency departments (ED) and adult medical services all over the world struggle to manage a workload that seems to increase inexorably. One group of ED presenters who have received particular attention are those who return repeatedly. It is assumed that, if they were somehow managed better, or differently, they might attend less frequently thus reducing the load on the ED and adult medical wards, while perhaps receiving better medical care. Useful models of alternative services have been trialled,[1-3] but no-one would claim to have 'solved' this problem.

In a parallel trend, there is an ever-growing number of people with long-term conditions, mainly due to an aging population with a heavy burden of lifestyle-related illness, and longer survival with certain chronic conditions—especially cardiovascular diseases[4]—and comorbidities are the norm.[5] Inevitably, a growing number of the ED presenters have long-term conditions as their primary or underlying problem. Meanwhile, our health care systems, both in primary care and secondary care, appear better designed to care for single, 'acute' medical conditions.[6,7] Attempts to adapt to the distinctly different and more complex demands of long-term conditions continue to challenge health systems internationally.[8 9]

We will describe attempts at Middlemore Hospital to systematically study the issue of frequent presenters at the ED, and our plans to formally trial a targeted intervention. Our main thesis is that designing systems that integrate secondary and primary care,

that are designed to support people with complex long-term conditions (often further complicated by psycho-social needs), may address at the same time both the short term challenges facing the ED and the long-term challenges of improving care for people with long-term conditions.

Definition of frequent presenter

There is no universally accepted definition of 'frequent presenter'. For example, the number of visits used as a criterion includes: four or more (in studies from Sweden,[10] the UK[11] and Ireland[12]); five or more (US,[13] Australia[14]) and 10 or more (Christchurch, New Zealand[15] and Spain[16]). Not surprisingly, a UK study noted that the profile of frequent presenters was a continuum; from about the fourth visit, the more frequently people attended the more likely they were to be male, older, attend after-hours and be more seriously ill.[11]

The current definition of frequent presenter used at Middlemore Hospital includes any adult (age 15 or over) presenting to the ED on five or more occasions in the preceding twelve months. Those under the active care of the renal or haematology service are automatically excluded; surgical, orthopaedic, obstetric and gynaecological patients may be excluded after triage. The ED computer system 'flags' those attending for the 5th or subsequent time in a year. The software behind this flag provides a first and partial filter to match our intended definition of frequent presenter.

Numbers at Middlemore Hospital

In the year ending 28 February 2010, 64,409 patients presented to the ED 88,565 times. Of these, 1711 patients age 15 or over, in 8756 presentations, were 'flagged'. Of these presentations, (5312, 61%) resulted in an overnight stay; total bed days for the year were 25,768, with a median per patient 10, interquartile range 4 to 23. According to the patient cost system, the total cost of flagged patients was approximately $31.5 million.

The median age of these 1711 patients was 56 years; 653 (38%) were age 65 or older (so eligible for Aged Care Services); 900 (53%) were women; 659 (39%) were European, 558 (33%) were Pacific, 323 (19%) were Māori, 102 (6%) were Indian, 35 (2%) were Asian, 22 (1%) were Other and 12 (1%) were or unknown ethnicity.

Fewest patients presented on Saturday (13%) and Sunday (12%), and most on Monday (16%). A total of 74% presented between 8am and 10pm (when most doctors are rostered at the hospital); and 56% presented between 8am and 6pm (when most doctors might be available in primary care). In all, 56% of presentations were on Monday to Friday between 8am and 10pm. A total of 69% of all presentations were self-referrals, suggesting a disconnection from primary care which has been noted in other studies of frequent presenters, although the literature also suggests that a subgroup are also high users of primary care.[10 12] One hundred and sixty six patients (10%) were, or had been, on the Counties Manukau DHB Chronic Care Management Programme.

Principal diagnoses on discharge ICD10 codes included diseases classified as: respiratory (18%), circulatory (14%), digestive system (10%), injury (8%), genitourinary (5%), endocrine (4%) and musculoskeletal (4%). These numbers do not

account for co-morbidities. A record audit of 30 frequently presenting patients in 2009 found them to have an average of three chronic medical conditions, most commonly respiratory, cardiovascular disease or diabetes, and taking nine regular medications. Many had psycho-social problems.

Pattern of same people over time

Table 1 shows that 77% of adults who frequent presented in a 9 month period from September 2008 to May 2009 also presented frequently in the following 9 months—no attempt has been made to adjust for seasonal variation. (The count does not include the small number who returned in the second 9 month period without meeting the frequent presenter flag criteria.) Overall, the return rate seems higher than others have found; 38% of high users in San Francisco were similarly high users the following year (using a criterion of 5 or more ED visits over the years 1993-8)[13]; and 40% of high users at Christchurch Hospital in1997 were also high users the following year (using a criterion of 10 or more ED visits).[15]

It is not clear why the numbers decrease the following year. Some die; 15% in the 18 months of our data shown in Table 1; 13% over the following 3–4 years in Christchurch;[15] 19% in Spain (10 or more ED visits), although the follow up period is unclear;[16] and frequent presenters in Sweden (4 or more visits) had a standardised mortality rate of 1.55 compared to non-frequent presenters.[10] Other plausible reasons for a decrease in attending the following year include getting better due to help from secondary care, primary care or social care. However, it is also possible that patients recovered spontaneously, moved out of the area, came to tolerate a condition or gave up their hope of help from the helping professions.

Table 1. Adults 'flagged' at Middlemore Hospital Emergency Department on their 5th or subsequent attendance in one year; comparing those who attended in one 9-month period with the subset who also attended in the following 9 months.

Variables	Presented to ED Sep 08 to May 09	Subset of first group who also presented Jun 09 to Feb 10
Visits, patients (% of baseline group)	6860, 1749	5387 (79%), 1348 (77%)
Age (median, inter-quartile range)	56 (34 to 73)	57 (35 to 73)
Female	914 (52%)	704 (52%)
European	686 (39%)	519 (39%)
Māori	332 (19%)	263 (20%)
Pacific	560 (32%)	440 (33%)
Asian	36 (2%)	23 (2%)
Indian	104 (6%)	80 (6%)
Other	17 (1%)	13 (1%)
Unknown	14 (1%)	10 (1%)

Note: The software behind the flag is intended to exclude those recurrently attending renal, haematology and gastroenterology day stay services. No attempt is made to adjust for seasonal variation.

Solutions proposed in the literature

Pope et al in Canada found that case management of 24 patients—chosen for their complex or chronic medical conditions, drug use, or their violent or abusive

behaviour—reduced their number of admissions from a median of 27 to 7 visits per year.[2]

On the other hand, when Phillips et al implemented case management for 60 patients who had averaged over 20 ED visits in the previous year to a tertiary hospital in Australia[1] they found that in the subsequent year these patients increased their use of both the ED and primary care. Perhaps this serves as a warning that some of the decrease in attendance in subsequent years seen at Middlemore and elsewhere may indeed be due to patients deciding we cannot meet their needs.

Both these studies used a before and after study design without a contemporaneous control, which is problematic given the apparent 'natural history' of decreased attendance over time (Table 1). Shumway et al used a randomised control design to study 165 patients under case management and 85 under usual care. Their patients had 5 or more visits in the previous year, and 'psychosocial problems that could be addressed by case management'.[3] Case management reduced ED use at a cost similar to usual care over 2 years.

Very high intensive users programme at Middlemore

In Counties Manukau a service has evolved over several years to coordinate care between ED, secondary care and primary care. Elements of this approach has already been tested with promising results.[17] A pilot programme called the Very High Intensity Users (VHIU) project has been allocated $800,000 for 1 year by the DHB. Ongoing funding will depend on the results of an evaluation. The business case states that the VHIU project aims to improve patient care co-ordination, and to establish, or re-establish, effective care in the community and general practice.

The multidisciplinary programme team includes part time input from general medicine Senior Medical Officers, Clinical Nurse Specialists, a Clinical Pharmacist, Primary Care Nurse Specialists and Locality Coordinators (who are employed by Primary Health Organisations (PHOs)). The role of the Clinical Nurse Specialists and pharmacist ensures early input for patients with the social worker, psychiatry liaison, Needs Assessment Coordination and associated allied health professionals as required. The role of the Primary Care Nurse Specialists and Locality Coordinators is to ensure safe integration and early collaboration with general practitioners, practice nurses and other community services.

For each patient who is 'flagged' when they attend ED, one of the VHIU staff uses a short checklist to review the electronic patient records for current and previously identified problems. See Figure 1 for a flow diagram of the 'filtering' process. Patients who are still included have an interview structured around a risk assessment guide (RAG, see Box 4), a tool modified from Degeling's observations in the United Kingdom.[8] The RAG tool is primarily used before the patient has left hospital but is otherwise done in the patient's home. The current RAG tool is a paper form that consists of 31 prompt items, space for free text comment, and a checklist for actions or referrals. The prompt items are grouped into six domains: demographic, social (including cultural and linguistic), health service related, mental health, pharmacy and clinical. The interview typical lasts about 20 minutes.

Figure 1. Initial screening tool for 'flagged' patients at Middlemore Emergency Department
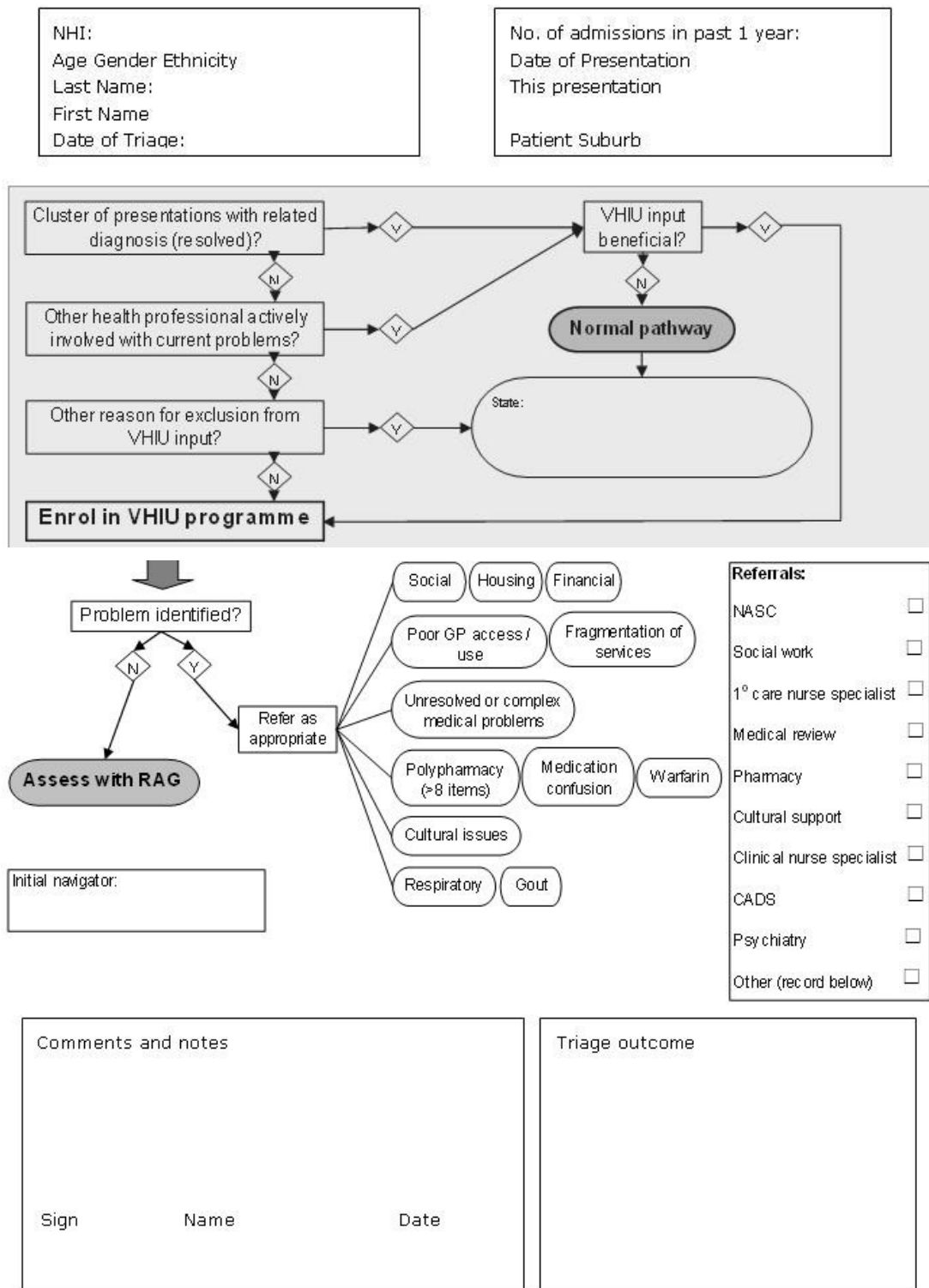
Experience suggests that the RAG supports a structured approach to identifying important patient issues that may have been missed, on repeated occasions, by a focussed clinical approach. Specialist Nurses and/or the Pharmacist will visit the patient either in the ward or at home and 'hear' the patient's concerns, understandings and expectations about their health issues. Navigation of the health system, literacy, cultural beliefs, coping mechanisms at home and various other barriers will become better understood as a consequence.

After the RAG assessment, each patient is considered by a wider multi-disciplinary group and one team member is delegated as the 'navigator' who then arranges any or all of: a more extensive review of secondary care or primary care medical records, a home visit to assess needs and living circumstances in more detail, a multi-disciplinary meeting with primary care or referral to secondary or primary service or social service agencies. The 'navigator' then 'follows' the patient—frequently and assertively if needed—and will advocate for them with other health or social agencies, either in person or by phone or other communication.

Patients remain enrolled in the VHIU programme until a decision is made collaboratively that they are receiving effective care and support from community services and have been integrated back into community care. In practice this process lasts from a few days to a few weeks. The programme has a small fund to reimburse short term costs of primary care, such as costs of waiving patient charges, or costs to have a GP attend a family conference.

Examples of the range of services and outcomes credited to the VHIU team include: effective advocacy for better housing; acceleration of outpatient review or medical investigations; medical case note review revealing clinically important disconnections of knowledge between medical services; better coordination of health care; shifting a Pacific man from a rest home where no Pacific languages were spoken into one were staff spoke his language; and sorting medication confusion and arranging for medications to be 'blister packed'.

Any time a patient is 'flagged' the same process is repeated, so that a patient not enrolled in the VHIU programme on the first occasion may be on another, or a patient who has been 'discharged' from the programme may be re-enrolled. Anecdotes within the VHIU programme to date and from the evaluation of the earlier pilot which tested the underlying concepts,[17] suggest that this programme may reduce the return visits to ED by as much as half. It is important to note that the VHIU focus is on preventing the next admission rather than early discharge at the index admission.

The proposed evaluation includes a randomised controlled trial to assess the effect of the programme on acute hospital demand; a process evaluation to evaluate programme delivery; measures of patient outcomes; and measures of costs.

Beyond VHIU

The VHIU programme was not developed in isolation—it is one of several programmes within Counties Manukau DHB addressing the problems of ED workloads and management of long-term conditions. It is in line with continuing experience at Counties Manukau which confirms the need for primary-secondary care integration and integration with social support for long-term care management.[17-19]

Other ongoing initiatives include Chronic Care Management,[20] Care Plus, improvement programmes in Aged Related Residential Care,[21] Year of Care, a Clinical Networks project (integrated Continuous Quality Improvement groups in primary care) and nurse-led clinics. A consortium of the three Auckland regional DHBs and eleven PHOs is currently developing a detailed project in response to the 'Better, Sooner, More Convenient' Request For Proposal.

We look forward to the day when long-term condition management will be centred on family and patient self monitoring and will be organised from community-owned, locality-based organisations that will hold budgets for long-term condition managements; when Continuous Quality Improvement will be the hallmark of care provision; and when information technology will provide the backbone for an integrated care system that supports a single shared patient record.

This vision can be achieved, but requires local pilots which are carefully evaluated and infrastructure developed around successful pilots, both requiring funding at a level that does not doom the pilots to failure. The VHIU project may be the first such pilot.

Box 1. People who have been involved in this work in Counties Manukau, and who have endorsed the concerns and the proposals outlined here

(Alphabetically) Jacqui Adair, Alex Boersma, Tom Bracken, Linda Bryant, Fay Burke, Ria Byron, Sam Cliffe, Janine Cochrane, Alan Cumming, Helen Duyvesten, Debbie Eastwood, Priya Francis, Jeff Garrett, Meg Goodman, Brad Healey, Denise Kivell, Chris Lash, Christine Lynch, Mangere Community Health Centre, Mangere Family Doctors, Tina McCafferty, Shirley Miller, Allan Moffitt, Helen Morrish, Primary Health Organisation Group (GPHO), Karyn Sangster, Vanessa Thornton, Sarah Tibby, Vanessa Whiu, Anne Williamson

Box 2. The archetypical VHIU patient in CMDHB is:

- A woman of 50–55 years old, obese with type 2 diabetes, cellulitis, gout and COPD or CHF.
- She will be as disabled as an 85 year old but will not be getting the services available to aged care such as case conferencing, care plans and home support.
- In fact, she will have dependent children and possibly parents, one reason she seeks her own medical care in EC after 10pm is that for a few hours the family do not need her.
- She will have appointments at 5 sub specialty outpatients clinics, and she will be labelled non compliant / DNA and will have no continuity of care with her GP.
- She will not be helped by being told that she must look after herself or she will not see her children grow up—she knows this.
- Health literacy will be zero as will self efficacy, and she will be unable to navigate the health / social welfare systems.
- She will be in a state of medication confusion and will be seeing 6 health care workers none of whom knows what the others are doing

Box 3. Elements of the proposed intervention in Counties Manukau

1. Risk Assessment Guide (RAG) - global assessment of needs to include patient and family/whanau perspective	7. One co-ordinator / navigator / key worker
2. Multidisciplinary input	8. Rigorous follow up
3. Home visits	9. Care plan
4. Relationship building	10. Self / family management
5. Clinical review	11. Crisis management
6. Case conference of health professional and family meeting, preferably in primary care	12. Advocacy for social services
	13. Evaluation
	14. Continuous improvement

Box 4. Data collected in the Risk Assessment Guide (RAG). Modified with permission from Degeling et al[8]

Patient sticker			Date		
Ethnicity			First Language		
Age 65+	Y	N	Uses multiple pharmacies	Y	N
Housing issues	Y	N	Poly-pharmacy (>8 items)	Y	N
Risk at home	Y	N	Compliance (meds)	Y	N
Living alone	Y	N	Progression of disease	Y	N
Living alone	Y	N	Multiple co-morbidities	Y	N
Living with dependent	Y	N	CNS involved*	Y	N
Support services	Y	N	Psychological issues	Y	N
Family/friend support	Y	N	E.g. health-seeking behaviour, personality disorder		
Independently mobile	Y	N	Compliance (clinics)	Y	N
Independent with ADLs*	Y	N	**Referrals made:**		
$ as health barrier	Y	N	Cultural support	Y	
Able to express needs	Y	N	Chronic care pharmacist	Y	
Cultural support desired	Y	N	NASC*	Y	
Can speak English	Y	N	Social worker	Y	
Literate (English)	Y	N	Primary Care Nursing	Y	
Poor health literacy	Y	N	Psychiatry	Y	
Service fragmentation	Y	N	Community Alcohol & Drug	Y	
Poor GP access	Y	N	Medical review	Y	
CCM enrolled*	Y	N	VHIU team*	Y	
Mental health diagnosis	Y	N	Other (specify)	Y	
Alcohol/drug misuse	Y	N			

* ADLs = activities of daily living, CCM = Chronic Care Management programme, CNS = clinical nurse specialist, NASC = needs assessment coordination service, VHIU = very high intensity users,

Competing interests: None known.

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References:

1. Phillips GA, Brophy DS, Weiland TJ, et al. The effect of multidisciplinary case management on selected outcomes for frequent attenders at an emergency department.[see comment]. Medical Journal of Australia 2006;184(12):602-6.

2. Pope D, Fernandes CM, Bouthillette F, Etherington J. Frequent users of the emergency department: a program to improve care and reduce visits. CMAJ Canadian Medical Association Journal 2000;162(7):1017-20.

3. Shumway M, Boccellari A, O'Brien K, Okin RL. Cost-effectiveness of clinical case management for ED frequent users: results of a randomized trial. American Journal of Emergency Medicine 2008;26(2):155-64.

4. National Health Committee. People with Chronic Conditions: a discussion paper. Wellington: National Health Committee, 2005.

5. Starfield B, Lemke KW, Bernhardt T, et al. Comorbidity: implications for the importance of primary care in 'case' management. Annals of Family Medicine 2003;1:8-14.

6. National Health Committee. Meeting the Needs of People with Chronic Conditions. Wellington: National Health Committee, 2007. http://www.nhc.health.govt.nz/moh.nsf/indexcm/nhc-people-with-chronic-conditions

7. Wagner EH, Austin BT, Michael Von K. Organizing Care for Patients with Chronic Illness. The Milbank Quarterly 1996;74(4):511-544.

8. Degeling P, Close H, Degeling D. Re-Thinking Long Term Conditions. A Report on the Development and Implementation of Co-Produced, Year-Based Integrated Care Pathways to Improve Service Provision to People with Long Term Conditions. Durham: The Centre for Clinical Management Development, Durham University, 2006.

9. Singh D, Ham C. Improving Care for People with Long Term Conditions: A review of UK and International Frameworks. HSMC. Birmingham: HSMC, University of Birmingham & NHS Institute for Innovation and Improvement, 2006.

10. Hansagi H, Olsson M, Sjoberg S, et al. Frequent use of the hospital emergency department is indicative of high use of other health care services.[see comment]. Annals of Emergency Medicine 2001;37(6):561-7.

11. Moore L, Deehan A, Seed P, Jones R. Characteristics of frequent attenders in an emergency department: analysis of 1-year attendance data. Emergency Medicine Journal 2009;26(4):263-7.

12. Byrne M, Murphy AW, Plunkett PK, et al. Frequent attenders to an emergency department: a study of primary health care use, medical profile, and psychosocial characteristics.[see comment]. Annals of Emergency Medicine 2003;41(3):309-18.

13. Mandelberg JH, Kuhn RE, Kohn MA. Epidemiologic analysis of an urban, public emergency department's frequent users. Academic Emergency Medicine 2000;7(6):637-46.

14. Jelinek GA, Jiwa M, Gibson NP, Lynch AM. Frequent attenders at emergency departments: a linked-data population study of adult patients. Medical Journal of Australia 2008;189(10):552-6.

15. Kennedy D, Ardagh M. Frequent attenders at Christchurch Hospital's Emergency Department: a 4-year study of attendance patterns. New Zealand Medical Journal 2004;117(1193):U871.

16. Salazar A, Bardes I, Juan A, et al. High mortality rates from medical problems of frequent emergency department users at a university hospital tertiary care centre. European Journal of Emergency Medicine 2005;12(1):2-5.

17. Sheridan N, Kenealy T, Parsons M, Rea H. Health Reality Show: Regular Celebrities, High Stakes, New Game. Integrated care in two general practices: a future model for managing complex patients in primary health care New Zealand Medical Journal 2009;122(1301):31-42.

18. Rea H, Kenealy T, Wellingham J, et al. Chronic Care Management evolves towards integrated care in Counties Manukau, New Zealand. New Zealand Medical Journal 2007;120(1252):1-11.

19. Rea H, McAuley S, Stewart A, et al. A chronic disease management programme can reduce days in hospital for patients with chronic obstructive pulmonary disease. Internal Medicine Journal 2004;34(11):608-14.

20. Kenealy T, Carswell P, Clinton J, Mahony F. Report of the evaluation of Chronic Care Management in Counties Manukau:Phase One. Auckland: Uniservices, University of Auckland, 2007.

21. Sankaran S, Kenealy T, Adair A, et al. A complex intervention to support 'rest home' care: a pilot study. New Zealand Medical Journal 2010;123(1308). http://www.nzmj.com/journal/123-1308/3948/content.pdf

Sulphasalazine lung toxicity: report of two cases

Gonesh C Karmakar, Conroy A Wong, Fiona Horwood, Graeme Anderson

Sulphasalazine has been used widely for treatment of ulcerative colitis as an anti-inflammatory agent since the 1940s. Later on, its use was extended to treat rheumatoid arthritis as a disease-modifying agent (DMARD[1]).

Sulphasalazine may cause clinically important adverse effects such as nausea, vomiting, headache, skin rashes, fever, arthralgias, abnormal liver function tests and less commonly agranulocytosis, haemolytic anaemia and neurotoxicity.[1,2] Pulmonary toxicity is less common and there are only few reports in the literature.[1,3]

We report findings in two cases of sulphasalazine-induced pulmonary toxicity.

Case 1—A 45-year-old lady with Crohn's disease and bronchiectasis developed enteropathic spondyloarthropathy involving both sacroiliac joints. She was started on sulphalazine as a DMARD. Four weeks later she developed shortness of breath, dry cough and hypoxic respiratory failure. A peripheral blood eosinophil count was normal. A plain radiograph showed bilateral airspace opacification most prominent in the left upper lobe with evidence of bronchiectasis on the right.

The high resolution CT scan (HRCT) demonstrated widespread ground glass opacification especially in the left upper lobe. There were some small centrilobular nodules and appearances were those of an acute hypersensitivity pneumonitis on a background of chronic changes of bronchiectasis (Image A). She was admitted to the intensive care unit for observation and high flow oxygen. She continued to deteriorate on broad-spectrum antibiotic treatment. Sulphasalazine was then discontinued and she was treated with high dose methylprednisolone. She improved clinically over the next few days and returned to her baseline level of functioning after 3 weeks.

Case 2—A 35-year-old lady was recently diagnosed with rheumatoid arthritis. She developed symptoms of breathlessness, dry cough and mild fever 10 days after commencing sulphasalazine. On discontinuation of sulphasalazine her symptoms improved. Sulphasalazine was recommenced one week later and she developed similar symptoms with more severe breathlessness after 4 days. Eosinophil counts were normal in peripheral blood and BAL.

The chest radiograph showed bibasal consolidation with septal thickening and thickening of the fissures. The HRCT demonstrated dense bibasal consolidation with thickening of the interlobular fissures throughout the lungs but worse at the bases. There were small pleural and pericardial effusions. The appearances were of an acute interstitial pneumonia with a differential diagnosis of infection (Image B). No infective aetiology was identified on culture of sputum or bronchial washings. Sulphasalazine was stopped but symptomatic improvement was slow. She was given a short course of oral corticosteroids with rapid resolution of symptoms.
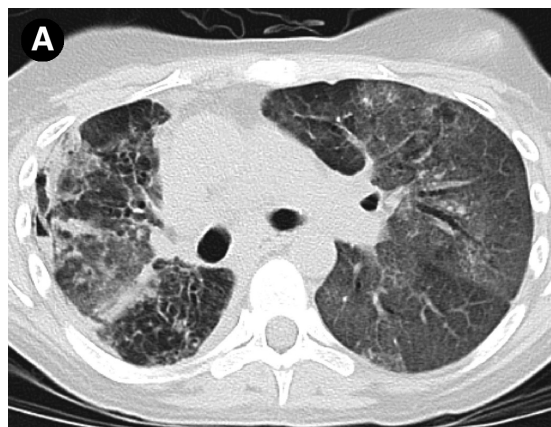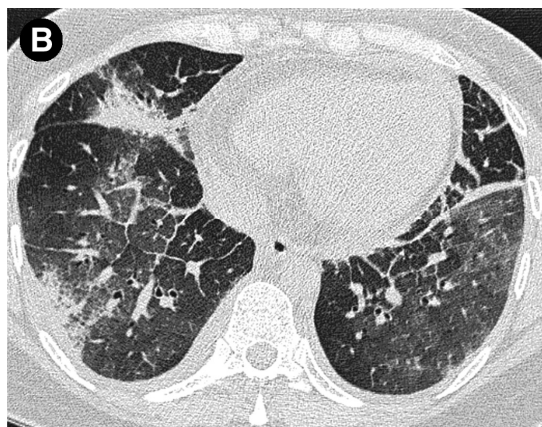
Image A

Image B

Discussion

Pulmonary toxicity from sulphasalazine is an uncommon but potentially serious adverse effect. The exact mechanism of sulphasalazine pulmonary toxicity is unknown. Various pulmonary pathologies have been reported including pulmonary eosinophilia, interstitial pneumonia[4] and hypersensitivity pneumonitis.[1] We described cases of hypersensitivity pneumonitis and acute interstitial pneumonia.

Pulmonary toxicity from sulphasalazine should be considered in patients who develop respiratory symptoms and radiographic changes. From our experience, discontinuation of the drug and treatment with corticosteroids facilitate rapid and complete recovery.

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References:

1. Parry SD, Barbatzas C, Peel ET, Barton JR. Sulphasalazine and lung toxicity: Eur Respr J 2002;19:756-764.

2. Moseley RH, Barwick KW, Dobular K, DeLuca VA. Sulphalazine-Induced Pulmonary Disease: Digestive Diseases and Sciences. 1985;30:901-904.

3. Hamadeh MA, Atkinson J, Smith LJ. Sulphalazine-induced Pulmonary disease. Chest. 1992;101:1033-1037.

4. Tydd TF, Dyer NH. Sulphasalazine Lung. The Medical Journal of Australia. 1976;1:570-573.

# A case of acupuncture-induced pneumothorax

Brian Kennedy, Lutz Beckert

Mrs P, a 54-year-old woman, presented with chest pain and increasing shortness of breath. She suffers chronic left-sided musculoskeletal chest pain, for which she sought out acupuncture therapy.

It was during her second acupuncture session that she became acutely short of breath, following introduction of an acupuncture needle into the right side of her chest posteriorly. She developed "tightness" in the right apical area and associated chest pain. She immediately left the acupuncture clinic, returned home and called an ambulance when her shortness of breath and pain worsened.

On arrival to hospital she was dyspnoeic and distressed. She described her chest discomfort as being pleuritic in nature and exaggerated by movement. She has a 25-pack year smoking history, having stopped smoking about 10 years prior. On clinical examination, absent air entry was noted in the right hemithorax with hyper-resonance to percussion.

Her chest X-ray confirmed a moderate-sized right pneumothorax (Figure 1).

Figure 1. Pneumothorax (lung border identified by arrow)
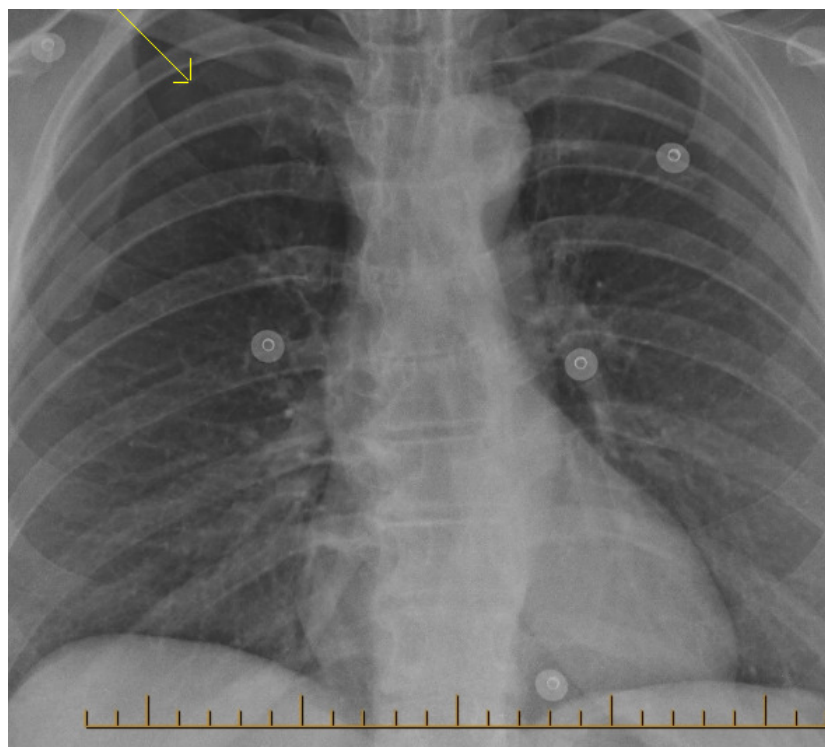
Her pneumothorax was aspirated and 450 ml of air removed from the pleural space. Her symptoms improved following the drainage. However the following morning Mrs P was noted to be increasingly dyspnoeic after mobilising to the bathroom.

A repeat chest film demonstrated a recurrence of the right pneumothorax extending to the right base. She was treated with a 12-gauge chest drain into the fourth intercostal space anterior axillary line. Her lung reinflated, the chest drain was removed and she was discharged home the next morning.

Discussion

Acupuncture is described as the insertion of one or more dry needles into the skin and subcutaneous tissue into acupuncture points. The term is derived from the Latin words "acus" meaning needle and "punctura" meaning penetration. Having originated in China over 2000 years ago, it remains a popular therapy for a variety of conditions today including chronic pain, nausea and vomiting, headache and hypertension. Its efficacy has proven difficult to ascertain.

A meta-analysis of randomised controlled trials of acupuncture for pain that included both sham acupuncture and no treatment arms found that the superiority of acupuncture over sham acupuncture, if real, appeared to be too small to be clinically important.[1]

Multiple models have been derived attempting to explain the perceived effects of acupuncture. Its most common use is in pain relief and this remains the most studied application. A popular theory is that of endorphin release. According to this theory, acupuncture stimulation is associated with neurotransmitter effects such as endorphin release at both the spinal and supraspinal levels.[2,3] It has been shown that opioid antagonists block the analgesic effects of acupuncture, supporting this theory.

Complications are infrequently observed with acupuncture treatment; however as with any form of needle use, adverse events can occur. These include transmission of diseases, needle fragments left in the body, nerve damage, pneumothorax, pneumoperitoneum, organ puncture, cardiac tamponade and osteomyelitis. Local complications include bleeding, contact dermatitis, infection, pain and paraesthesias.[4,5]

Despite the variety of listed complications and the occasional case reports, major adverse events are exceedingly rare and are usually associated with poorly trained unlicensed acupuncturists.[6] A prospective investigation in Germany of 97,733 patients constituting 760,000 treatment sessions reported that the two most frequently reported adverse events were needling pain (3.3 percent) and haematoma (3.2 percent).[7] Potentially serious adverse events included two cases of pneumothorax.

In conclusion, patients seeking acupuncture treatment should be directed to see only acupuncturists who are experienced and licensed. Despite the low risk of pneumothorax, all patients should be advised of the risk of pneumothorax when needles are being introduced into the thoracic region.

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References:

1. Madsen MV, Gotzsche PC, Hrobjartsson A. Acupuncture treatment for pain: systematic review of randomised clinical trials with acupuncture, placebo acupuncture, and no acupuncture groups. BMJ 2009;338:a3115.

2. Han JS, Terenius L. Neurochemical asis of acupuncture analgesia. Annu Rev Pharmacol Toxicol 1982; 22:193.

3. Andersson S, Lundeberg T. Acupuncture- from empiricism to science: functional background to acupuncture effects in pain and disease. Med Hypotheses 1995;45:271.

4. Lao L, Hamilton GR, Fu J, Berman BM. Is acupuncture safe? A systematic review of case reports. Altern Ther Health Med 2003; 9:72.

5. Ernst E, White AR. Prospective studies of the safety of acupuncture: a systematic review. Am J Med 2001;110:481.

6. Vickers A, Wilson P, Kleijnen J. Acupuncture. Qual Saf Health Care 2002;11:92.

7. Melchart, D, Weidenhammer, W, Streng A, et al. Prospective investigation of adverse effects of acupuncture in 97,733 patients. Arch Intern Med 2004; 164:104.

A case of nonresolving pneumonia

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

Clinical—A 64-year-old man presented with a low-grade fever (maximum temperature 37.5 degrees Celsius), malaise, nonproductive cough and dyspnoea for 1 week. Physically, fine crackles were heard at bilateral lung bases. Blood tests revealed a haemoglobin of 13.7 g/dl and a leukocyte count of 8,890/µl (neutrophil 65.8%, lymphocyte 28.9% and monocyte 3.5%). Smear of sputum specimens were negative for acid-fast bacilli and malignant cells.

A chest radiograph (Figure 1) showed consolidations at bilateral lower lung fields. The patient was treated with oral amoxicillin/clavulanate plus clarithromycin for 14 days. However, pneumonia did not resolve in the follow-up chest radiograph. Computed tomography (CT) of his chest (Figure 2) revealed bilateral lung airspace consolidations with a predominantly peripheral distribution in the lower and middle zones.

Figure 1

What is the diagnosis and how could it be confirmed?

Figure 2
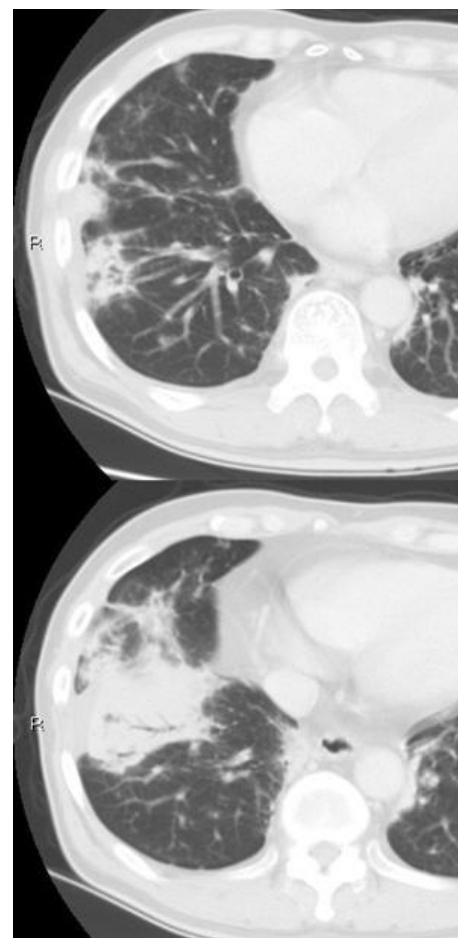
Answer—*Cryptogenic organising pneumonia (COP).* A thoracoscopic lung biopsy confirmed the diagnosis of COP. Resolution of symptoms and pulmonary infiltrates was achieved after systemic corticosteroid therapy (Figure 3).

Figure 3

Discussion

Unusual infectious diseases and noninfectious mimics of pneumonia should be considered in patients with a presumptive diagnosis of pneumonia who fail to response to antibiotic therapy. Despite the diagnosis is usually made after performance of invasive procedures,[1] the imaging pattern of idiopathic interstitial pneumonias would be diagnostic in a relevant clinical context.[2]

The characteristic appearances of COP on CT scan are areas of consolidation with a subpleural or peribronchial distribution, and a lower lung predominance.[3] In addition, ground-glass opacities are present in most cases and the lung architecture is relatively maintained without fibrotic destruction. The differential diagnosis of these findings includes collagen vascular disease, infection, vasculitis, sarcoidosis, lymphoma and bronchioloalveolar carcinoma.[3]

The suspicion for COP should be raised in those with a distinct clinicoradiological syndrome of subacute pneumonia and typical alveolar patches on imaging. The definitive diagnosis of COP requires surgical lung biopsy and the response to corticosteroid treatment is generally remarkable.

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References:

1. Alves dos Santos JW, Torres A, Michel GT, et al. Non-infectious and unusual infectious mimics of community-acquired pneumonia. Respir Med. 2004;98(6):488-94.
2. Johkoh T, Müller NL, Cartier Y, et al. Idiopathic interstitial pneumonias: diagnostic accuracy of thin-section CT in 129 patients. Radiology. 1999;211(2):555-60.
3. Lynch DA, Travis WD, Muller NL, et al. Idiopathic interstitial pneumonias: CT features. Radiology. 2005;236(1):10-21.

A case of recurrent hypoglycaemia

Scott Swendsen, Lester Layfield, Douglas G Adler

Clinical—A 59-year-old female presented to diabetes clinic with a 1.5-year history of episodes of confusion and syncope with two random blood glucose measurements found to be <40 mg/dL (normal 70–99 mg/dL). A triple phase pancreatic protocol CT and abdominal MRI were negative.

Upper endoscopic ultrasound (EUS) was performed and a mass seen (Figure 1). Endoscopic fine needle biopsy obtained abnormal cells (Figure 2).

Figure 1. 7.5MHz Radial EUS image showing an 7mm hypoechoic lesion in the body of the pancreas (arrow)
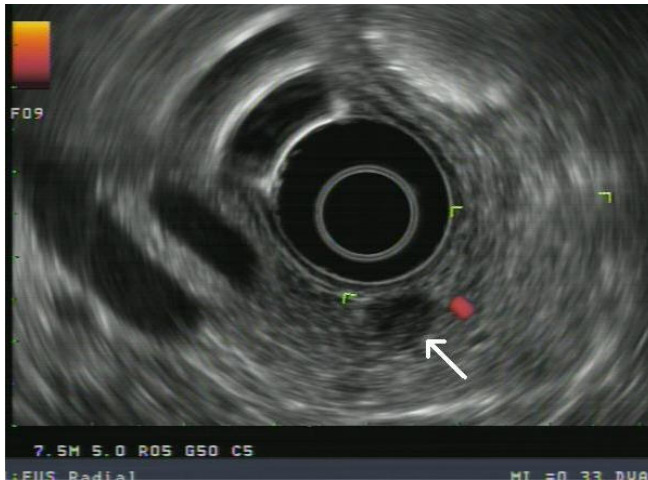
Figure 2. Cytologic specimen with a cluster of cells containing ovoid nuclei with a fine chromatin pattern (Romanovsky stain, ×600)
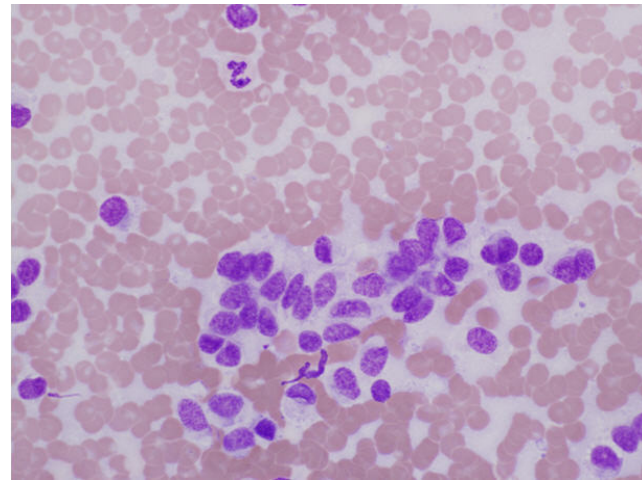
What is the diagnosis?

Answer—*Insulinoma*

Discussion—Insulinomas can be difficult to detect. Abdominal CT is considered the first line imaging study with sensitivity between 65–94%. MRI has a sensitivity of 85%. EUS is 82–94% sensitive for the detection of insulinomas.[1–5] Compared to CT and MRI, EUS has a greater sensitivity for detecting tumours <3 cm.[6,7] Insulinomas are often not seen on octreotide scans.[8] This case serves to illustrate that if MRI and CT studies are negative but a high clinical suspicion persists, there is merit in performing EUS.

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References:

1. Ichikawa T, Peterson MS, Federle MP, et al. Islet cell tumor of the pancreas: biphasic CT versus MR imaging in tumor detection. Radiology. 2000;216(1):163–171.

2. Kirkeby H, Vilmann P, Burcharth F. Insulinoma diagnosed by endoscopic ultrasonography-guided biopsy. J Laparoendosc Adv Surg Tech A. 1999;9(3):295-298.

3. Proye C, Malvaux P, Pattou F, et al. Noninvasive imaging of insulinomas and gastrinomas with endoscopic ultrasonography and somatostatin receptor scintigraphy. Surgery. 1998;124(6):1134-1143; discussion 1143-1144.

4. McLean A. Endoscopic ultrasound in the detection of pancreatic islet cell tumours. Cancer Imaging. 2004;4(2):84-91.

5. Ardeng JC, Rosenbaum P, Gang JA, et al. Role of EUS in the preoperative localization of insulinomas compared with spiral CT. Gastrointest Endosc. 2000;51(5):552–555.

6. Rosch T, Lightdale CJ, Botet JF, et al. Localization of pancreatic endocrine tumors by endoscopic ultrasonography. N Engl J Med. 1992:326(26):1721-1726.

7. Muller MF, Meyenberger C, Bertschinger P, Schaer R, Marincek B. Pancreatic tumors: Evaluation with endoscopic US, CT, and MR imaging. Radiology. 1994;190(3):745-751.

8. Mougey AM, Adler DG. "Neuroendocrine Tumors: Review and Clinical Update." Hospital Physician. 2007;43(11):12-20, 51.

Interprofessional learning in medical education in New Zealand

Judy McKimm, Dale Sheehan, Phillippa Poole, Mark Barrow, John Dockerty, Tim J Wilkinson, Andy Wearn

Abstract

This article considers interprofessional learning initiatives in the context of undergraduate and postgraduate education and the continuing professional development of doctors and other health professionals. The evidence for and challenges to delivering interprofessional education are discussed along with current interprofessional education initiatives in Aotearoa/New Zealand and Australia.

Many opportunities exist for health professionals to work together more effectively. We all want the best outcomes for our patients and good working relationships, but often we work and learn in professional silos. This paper explores the policy drivers for interprofessional learning (IPL), provides evidence for what works, identifies some of the challenges and shares examples of how health professionals in New Zealand are implementing IPL initiatives: aimed at improving health outcomes and facilitating well-functioning workplaces for all members of the health care team.

What is interprofessional learning?

The literature uses a number of terms relating to interprofessional interactions. Box 1 clarifies some of these terms.

Box 1. Some definitions

- Interprofessional Education (IPE): occasions when members (or students) of two or more professions learn with, from and about one another to improve collaboration and the quality of care.[1]
- Interprofessional Learning (IPL) is often used interchangeably with IPE, but is not such a prescriptive definition, referring both to planned interprofessional education / learning activities and those that arise more spontaneously in the workplace or in education.[2–4]
- Multiprofessional education or shared learning may involve different professions learning material together but not necessarily learning from or about one another.
- Interprofessional collaboration, (IPC) occurs when multiple health workers from different professions work together to provide comprehensive services to deliver high quality health care.
- An Interprofessional (IP) Team comprises different professionals who deliver services and coordinate care / improvement programmes. An IP team:
- Sets goals collaboratively through consensual decision making
- Has activities that result in an individualised care plan / service / programme delivered by one or more team members
- Maximises the value of shared expertise
- Minimises barriers of professional autonomy.[5]

Successful IP teams understand where professional boundaries intersect and end, respect all members of the health workforce, disrupt hierarchies and activate all members of the health care team. [6, 7] IPL is just one of many educational approaches that can contribute to improved teamwork and collaboration and improved patient care. A powerful way to learn about IPC is through experiences gained by working in teams in the workplace. However, much of this learning is serendipitous and role models may not always be ideal. Consequently, such opportunistic learning may not produce learners who are of a benchmark standard or who have had similar core learning experiences that meet defined educational outcomes. [8]

If we want to ensure that our future health professionals are equipped to work in integrated services, function effectively across professional and organisational boundaries and genuinely work collaboratively with other health workers, then we need to formally educate them to do so. Some of this formal education is best delivered by IPL but there are also other modalities such as debriefing sessions by mentors and team building exercises for individual clinical teams. The advantage of IPL is that it requires the learners to work in teams or groups to explore similarities and differences between professions and to learn from one another about health care with the aim of functioning more effectively in IP work-based teams.

Why IPL?

Since the late 1970s, interprofessional learning has had high level policy impetus from international bodies, such as the World Health Organisation (WHO) and governments, emphasising the need for health workers to work together for effective health care. Two key international political drivers were the WHO report [9] that revealed an urgent need to enhance human resources for health and the WHO 5th World Assembly Resolution calling for rapid scaling up of health workforce production and the use of innovative approaches to teaching.[10]

As one innovation to help tackle this problem, the WHO launched a study group on Interprofessional Learning and Collaborative Practice. This group conducted an international 'environmental scan' and an assessment of the current research in this area and synthesised it within an international context. [11]

The group collated the evidence from six systematic reviews on IPE, six systematic reviews related to collaborative practice plus collaborative practice case studies from ten countries. The major strength of the WHO Framework is that it was a study carried out by international experts, based on a wider international consultation than any previous studies.

The study group concluded that "*After almost 50 years of inquiry, there is now sufficient evidence to indicate that interprofessional education enables effective collaborative practice, which in turn optimizes health services, strengthens health, and improves health outcomes.*"[11(p18)]

This is not a direct causal relationship and IPL is clearly not the only factor in IPC but the WHO report highlights the link between IPL and IPC through the development of *collaborative, practice-ready health care professionals*; stating that interprofessional education can be a key contributor in the development of a collaborative practice-ready health workforce. This relationship is represented in Figure 1.[11(p13)])

Figure 1. IPL and collaborative practice: the links

Independent of the IPL agenda, standards defined by professional and statutory bodies responsible for medical students, trainees and practising doctors are increasingly emphasising the need for team-working skills, collaboration and communication skills.[12–15] Health professional education programmes have introduced a range of IPL initiatives, from single projects through to major curriculum interventions. They involve students as champions and leaders, university and health service teachers, and managers and practitioners working in shared health contexts.[2,11,12,16] Outcomes from these initiatives are discussed in the following section.

What IPL works?

As Hammick et al[16] point out in the BEME systematic review of the IPE literature, the wide range of IPL interventions use different educational methods, involve different professional groups and employ different evaluation methods. Most of the published reports are descriptive and evaluative, and some report mixed results on the impact of IPL on improved practice, rather than providing robust research-based evidence on the effectiveness—or not—of IPL.[17,18] However, emerging empirical evidence cited by recent reports indicates the positive benefits of IPL on learner satisfaction, increased knowledge and skills about collaborative practice and changed perceptions of others in the health care team.[11,12]

Drawing from the wide range of published literature, IPL that contributes effectively to improving collaborative practice should include:

- An authentic, realistic IPL experience linked to the needs of all professionals involved in the learning;[19,20]
- IPL activities customised to meet the needs of learners and the educational and health context; [21]
- Expert and specifically trained staff to facilitate IPL activities, drawn from a range of professional backgrounds [22, 23, 24]
- IPL as a core learning modality within the curricula and not as a bolt-on;[11,25,26]
- Clear learning outcomes around content and process;[27–29]
- A shared vision of IPL and how it should be implemented in the organisation;[12,25]
- Resources (rooms, equipment, teachers) to enable IPL to occur;[25,30]
- Leadership for IPL through champions and educational strategies.[25,31,32]

What is happening in New Zealand?

In New Zealand, planning for improved integrated family health centres and restructuring of secondary and tertiary health organisations requires acknowledging that interprofessional teams are a key plank of the health workforce. [33] Many IPL initiatives exist, involving a range of health professionals, in undergraduate, postgraduate and continuing education settings. Examples include the first international IPE conference in Australasia in 2010; the Australasian Interprofessional Practice and Education Network (AIPPEN) and the NZ National Centre for Interprofessional Education and Collaborative Practice Consultative Group.

In this article, we focus on some specific examples involving medical students, trainees and qualified doctors. However it is first important to distinguish between multidisciplinary learning and IPL. The Universities of Auckland and Otago undergraduate medical programmes both offer a common first year to prospective medical students. Students learn in class sizes of over 1000, and include those aiming for physiotherapy, pharmacy, medicine, dentistry, nursing and health science programmes. Although these students learn alongside and with one another, this is not structured IPL: IPL means "learning about" and "from" rather than simply "learning in the same room as" other future health care professionals. In addition, the majority of these students have not yet even entered an identified health professional programme.

The undergraduate context—During the "Early Learning in Medicine" phase of the Otago medical curriculum, students work as carers in residential care facilities. This initiative is currently the subject of extensive evaluation but the hope is that valuable lessons around the roles of other health professionals will be learnt during these experiences. A recent new development at the Dunedin School of Medicine happens during ambulatory care learning for fourth year medical students which involves a simulation session with new graduate nurses with a view to benefiting both professions. The medical students work through a clinical scenario with a "patient" (SIM Man™) that involves the nurse in making an initial assessment. An example of a smaller-scale initiative at the University of Otago, Christchurch, asks students during the Health Care of the Elderly module to work with and document the skills and attributes that other health professionals bring to their older patients. [34]

Since 2000, the Faculty of Medical and Health Sciences (FMHS) at the University of Auckland has offered two core IPL modules for medicine, nursing and pharmacy students: Māori Health Week runs in year 2 and a two-day quality and safety activity runs in year 3.[35] Where possible, the IP group membership remains the same for the year 3 activity to build on the relationships developed in year 2. [36]

The learning outcomes for Māori Health Week include IPL components as well as those of improving Māori healthcare. Seminars and case-based scenarios provide triggers for facilitated group learning about health disparities between Māori and other New Zealanders. Trained facilitators from a range of professions work with the groups to facilitate the sharing of experience from various professional backgrounds.

This approach fosters students from the different programmes working in pairs or small groups on projects, and generates shared group learning around Māori Health. Changing perspectives and any resulting tensions are actively managed by the facilitators and the groups themselves.

During the quality and safety module, interprofessional student groups work with facilitators from the three professional programmes plus hospital-based quality managers. They learn from a range of experts and undertake root cause analyses of real adverse events, exploring actions healthcare teams might take to prevent a recurrence. This highly structured initiative was positively evaluated both for content and for the interdisciplinary approach that provides training for clinical teamwork [37] and complements the more opportunistic learning that occurs in the workplace.

Although evaluations from both modules consistently show that students value IP group learning and state that they learn from and about the other professions, the effectiveness of IPL in improving clinical practice and patient care is yet to be measured.

Since 2008, reflecting an example of a more strategic and systematic approach to IPL which aims to address structural barriers,[21,38] IPL has been formally embedded into the Educational Strategy of the FMHS at the University of Auckland.'. A working party audited activities across the undergraduate health professional programmes and developed an IPL strategy and capabilities framework (see Box 2).

The Framework is used by programme leaders to ensure that activities are in place to facilitate and measure the achievement of the capabilities. As at Otago, educational interventions include mixed models of learning to work interprofessionally, where some groups will learn together or students will learn about the other professions through clinical placements or e-learning activities.

Box 2. FMHS Interprofessional capabilities framework (The University of Auckland)

Interprofessional capabilities
KNOWLEDGE On graduation, students will be able to demonstrate understanding of:
➢ Health services and systems; roles of players and components including changes and the drivers of change
➢ The roles and responsibilities of other health professionals and professions supporting health and social care, including the role of advanced practitioners
➢ The ethical and legal frameworks underpinning healthcare and professional practice
➢ Determinants of health and health inequalities
➢ The concepts and practice of interprofessionalism and integrated care
➢ A range of models and paradigms of health and wellness
➢ Models to improve quality and safety in healthcare, including to reduce error
SKILLS On graduation, students will be able to demonstrate effective collaboration and co-operation with health care team members, professionals, patients and community groups supporting health and social care in a range of contexts and settings. This will incorporate:
➢ An awareness and application of appropriate role boundaries
➢ Verbal and written communication with and referrals to other health professionals and agencies
➢ Cultural understanding, competence and safety
➢ Leading and managing teams
➢ The application of different evidence paradigms to healthcare practice
➢ A proactive approach to ensuring safe healthcare delivery

ATTITUDES/BEHAVIOURS
On graduation, students will be able to demonstrate that they:
➢ Value and show respect for a range of health care professions and professionals (and associates—e.g. social workers)
➢ Actively and proactively collaborate with others to improve advocacy and patient care, and improve health outcomes
➢ Articulate and maintain a distinctive and authentic professional identity relative to own role and role in the team
➢ Practice as a constructive and collaborative health care team member with respect for complementary skills and competencies

The postgraduate context—Postgraduate courses across New Zealand, delivered in the tertiary education and clinical setting provide many varied examples of students learning from, about and with one another. To offer just one example, within the Department of Preventive and Social Medicine at Otago, courses in Public Health and Research Methods attract students from wide a range of health disciplines. Students have opportunities to work together in small interprofessional groups which allow pooling of students' rich backgrounds of professional expertise. Learning how to work collaboratively together is an integral part of their learning experience.

Continuing professional development

IPL also occurs in programmes of Continuing Professional Development (CPD), most commonly for practitioners supporting patient pathways involving multidisciplinary teams which cross organisational and professional boundaries, such as family medicine, palliative care, care of the elderly, maternal and child health and mental health. The University of Auckland runs specific modules on IPE in its Masters' programmes in Clinical Education and nursing to support clinical educators in developing and delivering IPE and IPL.

In 2007, Canterbury District Health Board (CDHB) established an Interprofessional forum to promote IP collaboration in clinical postgraduate education with representatives from a range of health and social care disciplines. This was formally supported by Directors of Nursing and Medicine, key allied health professionals across the CDHB and the Training and Development Unit.

In 2008 and 2009, symposium topics included 'supervision models and tools used in different disciplines' which resulted in the establishment of an interprofessional module for clinical supervisors teaching clinical/portfolio assessment within the DHB. The group also hosted expert practitioners from CAIPE (the UK Centre for the Advancement of IPE) to share expertise with national and local groups. In March 2010, the group (including representatives from nursing, medicine, social work, occupational therapy, physiotherapy, speech language therapy, pharmacy and dietetics) piloted an 'interprofessional ward' simulation exercise (see Box 3).

The evaluation of the ward simulation (developed using the Sheffield University CILASS impact evaluation framework)[38] provided extensive information at a number of levels, from tips for the future on IPL delivery, to running ward simulations and future projects. Although participants' feedback indicated that they felt that they achieved the individual learning outcomes, the facilitators (who may have had higher expectations around IPL) felt that more interprofessional collaboration could have been achieved.

The pilot provided a number of strategies for overcoming this and recommended that an IP learning experience be part of orientation for all new graduates. The simulated

ward experience is a viable and worthwhile approach to introducing IPL and collaborative practice to both undergraduates and new graduates.

Box 3 Interprofessional Ward Simulation

Requires new practitioners from nursing, medicine and allied health disciplines to jointly prioritise the care of simulated patients and compile collaborative patient records. Aims were to:

1. Work collaboratively as an interprofessional (IP) team in a simulated healthcare environment
2. Integrate their clinical skills in this setting
3. Jointly prioritise the care of simulated patients
4. Socialise interprofessionally early in their careers
5. Compile collaborative patient records

Anticipated individual learning outcomes

1 Enhance confidence in working in an IP team
2 Enhance understanding of the roles and responsibilities of each professional group taking part in the scenario
3 Gain realistic expectations of other professionals on the team

Challenges to delivering IPL

The challenges surrounding IPL are not unique to practice in New Zealand, have been highlighted by others, and include:[3,8,11,12,16–18,23,34,39–41]

- Determining the right stage of readiness for students to engage in IPL;

- Being sure that it does no harm. For example, uninformed interactions with other health professionals could, in theory, lead to reinforcement of stereotypes. This may be a particular risk if professional identities are still relatively immature;

- The logistics of timetabling small group sessions with increasing student numbers;

- Managing teaching within timetable constraints, particularly those around differing requirements for clinical placement activities and detailed curriculum requirements from professional bodies;

- Being able to bring together students with varying authentic experiences so that the experience is meaningful to all;

- Identifying appropriate activities (with or without real patients) and learning interventions;

- Willingness of senior management to commit to investment in IPL with increasing pressure on resources and ever-crowded curricula;

- Recruiting, training and supporting expert facilitators and IPL 'champions' who are comfortable with and competent at facilitating interprofessional groups;

- Unhelpful and entrenched stereotypes and attitudes and perceived threats to professional identity;

- Lack of attention to language (different terminology, same terms different meaning).

With scarce resources, some of the challenges can be very difficult to overcome and require commitment throughout the organisation(s), the establishment of IPL champions and attention to ensuring that learning is contextualised for the educational and service environment.[11,16,25]

Summary and conclusions

Drivers and opportunities exist to guide increased local structured IPL, collaborative practice initiatives and research in IPE. These include intensive planning for integrated family health centres and continuing financial pressures in health and education, the publication of international policy guides (such as the WHO Framework[11]) and networking and conference opportunities.

Calls for increased interprofessional collaboration in healthcare are becoming more insistent. The educational and financial drivers to diversify the workforce, to develop new roles and to extend existing roles are gaining more ground in New Zealand. [33] Alongside this, there is an urgent need for rigorous evaluation of any initiatives as we have an incomplete understanding of what works, what does not work and what may do harm.

Although more research is needed to assess the extent to which learning together results in better collaborative practice and improved patient outcomes, the evidence to date shows that students enjoy and value IPL and believe it improves their capacity to work with other professionals. Links between teaching innovations and patient care outcomes may be the 'Holy Grail' of medical education but are very difficult to demonstrate and a challenge not just for IPL but all areas of education; generalised clinical outcomes may not even be a realistic goal given that context is always a dependant variable. [42]

While well planned IPL may lead directly to improved health outcomes, [2,11,12] the evidence from the 2010 WHO study [11] shows that well-planned IPL at all stages of training can help to address the challenges in preparing professionals for an increasingly complex global healthcare environment.

Key points:

- IPL provides opportunities for students and qualified health professionals to learn to work with other professionals collaboratively;

- Key policy drivers around the world emphasise team-working and collaborative practice as key components for improved health care;

- There are many challenges to delivering IPL, especially in undergraduate education;

- Many examples exist, in New Zealand and around the world, of effective IPL;

- More research is needed to evaluate the effectiveness of IPL in improving health outcomes and patient safety.

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References:

1. Centre for the Advancement of Interprofessional Education (CAIPE). www.caipe.org.uk; 2002.

2. Freeth D. Interprofessional Learning. Edinburgh: Association for the Study of Medical Education, 2007.

3. Centre for the Advancement of Interprofessional Education (CAIPE) workshops. www.caipe.org.uk, 2008.

4. Reeves S. An overview of continuing interprofessional education. JCEHP. 2009; 29: 142-6.

5. Towards Unity for Health. Position paper – Interprofessional Education and Practice, http://www.the-networktufh.org/home, 2007.

6. Laugesen, R. Just checking. NZ Listener, March 6-12, 2010:16-20

7. Hayne AB, Weiser TG, Berry WR, et al. A Surgical Safety Checklist to Reduce Morbidity and Mortality in a Global Population, New Eng J Med. 2009;360:491

8. Illing J, Morrow G, Kergon C, et al. How prepared are medical graduates to begin practice? A comparison of three diverse medical schools. Report to General Medical Council, September 2008.

9. World Health Organization. World Health Report 2006: Working together for health. Geneva: World Health Organization; 2006.

10. World Health Organization. WHA59.23: Rapid scaling up of health workforce production. Fifty-Ninth World Health Assembly. Geneva: World Health Organization, 2006;37-8.

11. Health Professions Network Nursing and Midwifery Office within the Department of Human Resources for Health. Framework for action on interprofessional education & collaborative practice. Geneva: World Health Organization; 2010.

12. World Health Organization. WHO Patient Safety Curriculum Guide for Medical Schools. Geneva: World Health Organization, 2009.

13. Australian Medical Council. Assessment and accreditation of medical schools: Standards and procedures, 2009. http://www.amc.org.au/images/Medschool/standards.pdf

14. General Medical Council (UK) Tomorrow's Doctors 2009 . London: General Medical Council. 2009. http://www.gmc-uk.org

15. Royal College of Physicians and Surgeons of Canada. The CanMEDS Physician Competency Framework: Better standards, better physicians, better care. http://rcpsc.medical.org/canmeds/index.php

16. Hammick M, Freeth D, Koppel I, et al. A best evidence systematic review of interprofessional education. Med Teach 2007;29:735-51.

17. Thistlethwaite, J, Nesbit, G. Interprofessional education. What's the point and where we're at The Clin Teach. 2007;4:67-72.

18. Wakefield A, Boggis C, Holland M. Team working but no blurring thank you! The importance of team work as part of a teaching ward experience. Learn Health Soc Care. 2006;5:142-54.

19. Kilminster S, Hale C, Lascelles M, et al. Learning for real life: patient-focused interprofessional workshops offer added value. Med Educ. 2004:38:717-26.

20. Ketola E, Sipils R, Makel M, et al. Quality improvement programme for cardiovascular disease risk factor recording in primary care. Qual Saf Health Care. 2000;9:175-80.

21. Shafer MA, Tebb KP, Pantell RH, et al. Effect of a clinical practice improvement intervention on Chlamydial screening among adolescent girls, JAMA. 2002;288:2846-52.

22. Steinert Y. Learning together to teach together: interprofessional education and faculty development. J Interprof Care. 2005;19(Suppl 1):60-75.

23. Oandasan I, Reeves S. Key elements for interprofessional education, Part 1: the learner, the educator and the learning context. J Interprof Care. 2005;19:S21-38.

24. Hawkins E, Bray J. Preparing for Interprofessional Teaching. New York: Radcliffe Press. 2008.

25. Gilbert JHV. Interprofessional learning and higher education structural barriers. J Interprof Care. 2005;19(Suppl 1):87-106.

26. Stone M. Coming in from the interprofessional cold in Australia. Aust Health Review. 2009;31:332-40.

27. Freeth, D, Nicol, M. Learning clinical skills: an interprofessional approach. Nurse Educ Today. 1988;18:455-61

28. Ker J, Mole L, Bradley P. Early introduction to interprofessional learning: a simulated ward environment. Med Educ. 2008;37:248-55.

29. Davidson M. Interprofessional pre-qualification education: a systematic review. Aust Health Review. 2008; 32:111-12.

30. Plimott J. Building Clinical Education Capacity with Interprofessional Clinical Education Units. Paper presented at the All Together Better Health Conference 5, International Interprofessional Conference Sydney, Australia, 6-9 April 2010.

31. Reeves S, Freeth D. The London training ward: an innovative interprofessional leaning initiative. J Interprof Care. 2002;16 (1):41-52.

32. Morey JC, Simon R, Jay GD, et al. Error reduction and performance improvement in the emergency department through formal teamwork training: evaluation results of the MedTeams project. Health Services Research. 2002;37:1553-81.

33. Boyd MA, Horne W. Primary Health Care in New Zealand: Teamworking and Collaborative Practice. Auckland: Waitemata District Health Board. 2008.

34. Wilkinson TJ. Teaching teamwork to medical students: goals, roles and power. Medical Education. 2002;36:1089-90

35. Horsburgh M, Lamdin R. Māori health issues explored in an interprofessional learning context. J Interprof Care. 2004;18:279-87.

36. Horsburgh M, Merry A, Seddon M, et al. Educating for healthcare quality improvement in an interprofessional learning environment: A New Zealand initiative. J Interprof Care. 2006;20:555-7.

37. Garden S, Bernau S, Robinson G, et al. Undergraduate education to address patient safety. N Z Med J. 2008;12:119-21.

38. CILASS impact evaluation framework. In: http://www.sheffield.ac.uk/cilass/students/san.html Sheffield University, 2010.

39. Lindqvist SM, Reeves S. Facilitators' perceptions of delivering interprofessional education: a qualitative study. Med Teach. 2004;29:403–5.

40. Koppel I, Barr H, Reeves S, et al. Establishing a systematic approach to evaluating the effectiveness of interprofessional education. Issues in Interdisc Care. 2001;3:41-9

41. Sheehan D, Robertson L, Ormond T. Comparison of language used and patterns of communication in interprofessional and multidisciplinary teams. J Interprof Care. 2007; 21:17-30.

42. Regehr G. Trends in medical education research, Academic Medicine; 2004;79:939-9.

The Annual Meeting at Auckland

The annual meeting of the New Zealand Branch of the British Medical Association was held in Auckland on February 28th, 1911, and following days. All who attended say that the meeting was an unqualified success, and though there were not quite so many visitors from the south as were expected, the meetings were well attended, and the papers, several of which are published in this issue of the JOURNAL, were of more than usual interest. We print an appreciation by a country doctor, which gives a fair idea of the importance and usefulness of these annual meetings.

Impressions of the Annual Meeting

[By "MEDICO."]

Those of us who were fortunate enough to attend the Annual Meeting held this year in Auckland have new memories of a profitable and pleasant time, and a happy combination of business and pleasure.

The Auckland doctors and their charming wives seemed to do all in their power to give the visiting members and their wives a bully time, to use an American expression. Who will forget the dinner and the amusing "tom-cat incident." Then it was pleasant to see medical men renewing old acquaintances and friendships—also making new friendships. You would see two medical men foregathering for the first time—perhaps they had heard of one another, but had never met before ; one had formed a mental picture of the other as being perhaps a tall, stout, elderly man, and lo, to his surprise, he finds the person in question is a thin, lean, shaven, youthful-looking individual of somewhat small stature.

Then the business part of the proceedings were certainly very instructive. Some excellent papers were read, and the discussions were also very good. Now and again the seriousness of the discussions would be relieved by the "fulminating" Dr. C., the amusing part being that the more he fulminated, the less seriously were his remarks treated. Dr. Valintine was present at one or two of the meetings, and explained his views on the proposed Nursing Scheme, having trained nurses in the back-blocks. This should, I think, meet with our hearty approval. Then he mentioned the proposed Notification of Venereal Disease. This is a matter that wants very careful consideration.

Dr. Valintine made a decided *faux pax* when he said that hospital abuse was a thing of the past. The remark made several of those present squirm, and one or two spoke very strongly on the subject,, and several more would have spoken on the same matter but for the consideration of Dr. Valintine's feelings. Hospital abuse is very prevalent, especially in the smaller townships, where all classes of people, rich and poor alike, go to the general hospital for operations, etc.

Thus as I have said we had a pleasant and profitable time, and the week passed all too quickly.

Some of us would have liked to have heard some discussion on the vexed Lodge Practice, but time did not permit. This is a matter that is of importance to the average general practitioner. Then another important matter affecting the welfare of the general practitioner in some smaller townships which there is a hospital, is the so-called Resident-Surgeon (with free house, etc., and salary) engaging in general practice. It is most unfair.

I would like to see these three important matters brought up at every annual meeting of the British Medical Association in New Zealand, *i.e.*, (I) hospital abuse; (II) lodge practice, and (III), resident surgeons engaging in private practice. These cannot be discussed too often.

Increased *Clostridium difficile* virulence in North America

The Infectious Diseases Society of America informs us that there is an epidemic of *Clostridium difficile* (*C. difficile*) infections in both Canada and the USA. The incidence now exceeds the incidence of MRSA infection and is as common as hospital-acquired bloodstream infections. Furthermore, the virulence has increased as mutation of the bacteria has resulted in more harmful toxins.

This epidemic of more virulent *C. difficile* is ascribed to the widespread use of fluoroquinolones in North America. These antibacterial drugs are the most common first choice drugs in the North American community hospital environment. Fortunately they are not our first choice in New Zealand and we should refrain from frivolous use of these valuable back-up antibacterials. New *C. difficile* treatments are being sought and these include monoclonal antibodies (of course) and a *C. difficile* toxin vaccine. A new biotherapeutic possibility is also being investigated—colonising the gut with non-toxin producing *C. difficile* strains which will keep out the toxigenic strain!

JAMA 2010;303:2017-9.

More about sutures vs staples in wound closure

Our abstract in the 16 July 2010 *NZMJ* reported a less than satisfactory meta-analysis which concluded that orthopaedic surgeons should close wounds with sutures rather than staples as this would minimise wound infections. This has attracted a flurry of criticism—seven letters pointing out the flaws in the meta-analysis. The points made include selection bias, only 6/195 papers included and not mentioning the recent guidelines on surgical site infection from the National Institute for Health and Clinical Excellence (NICE), particularly their conclusions and recommendations on using staples to close the skin. Other pertinent points include the fact that not all sutures are nylon and dissolving sutures are known to initiate foreign body reactions which may predispose to infection. The relative skills of senior surgeons and junior colleagues also receive mention. One anti-stapler points out that staples are more expensive, are painful when removed and produce broader scars. In their reply the authors of the meta-analysis acknowledge the criticisms. Their revised conclusion is that well-designed randomised trials are still needed before considering changing clinical practice.

BMJ 2010;340:1097-8.

Treatment of restless legs syndrome with gabapentin enacarbil

The restless legs syndrome (RLS) is characterised by an urge to move the legs, usually accompanied or caused by uncomfortable sensations in the legs. Treatment with dopamine agonists may help but side effects are a problem. Gabapentin has been reported as useful in some but irregular absorption limits efficacy. This study reports

on the use of the pro-drug gabapentin enacarbil which is reputed to produce sustained gabapentin levels with single daily dosing. In a preliminary study nearly 60% of patients with the RLS benefitted from the use of gabapentin enacabil. These 194 patients were randomised in a double blind trial to either 1200mg of gabapentin enacarbil once daily (5 p.m.) or placebo. At 36 weeks a significantly smaller proportion of patients treated with gabapentin enacarbil (9/96 {9%}) experienced relapse compared with the placebo-treated patients (22/97 [23%]). Apparently the drug was well-tolerated with no adverse effects recorded. Interestingly three adverse effects were recorded in the placebo cohort. Neither gabapentin or the pro-drug are approved for treatment of the RLS in the USA (or NZ).

Testosterone therapy in men with androgen deficiency—maybe not a good idea

It is known that elderly men with androgen deficiency may lose muscle mass and strength. This study assessed the merits of androgen replacement in a placebo-controlled trial in such men. 290 men (mean age of 74 years) with limitations in mobility and a total serum testosterone of 100 to 350 ng per decilitre (3.5 to 12.1 nmol per litre) or a free serum testosterone level of less than 50 pg per decilitre (173 pmol per litre) were randomly assigned to receive placebo gel or testosterone gel, to be applied daily for 6 months. At 6 months the testosterone cohort had significantly better muscle strength with improved mobility. This is the good news. The bad news is that the trial was terminated prematurely on safety grounds as 23 of the testosterone group developed cardiovascular adverse events, compared with 5 in the placebo group. I note that the testosterone treated group at baseline included significantly more men being treated with antihypertensive medication. In addition, the numbers with hyperlipidaemia and statin therapy were significantly greater in the testosterone group. A wise move to stop the trial. It would have been even wiser to have excluded the men with increased cardiovascular risk factors before starting the trial.

Can vancomycin-coated tympanostomy tubes prevent the formation of methicillin-resistant *Staphylococcus aureus* biofilm?

We have been on a bit recently about bacterial biofilm and the problem that antibiotics have in gaining access. Apparently tympanostomy tubes also develop biofilm which leads to chronic middle ear suppuration. This study compares tympanostomy tubes coated with silver oxide or vancomycin or nothing after they have been inoculated with methicillin-resistant *Staphylococcus aureus*. Electron microscopy several days later showed *S. aureus* biofilm had developed on both the uncoated tympanostomy tubes and the silver oxide tubes. The vancomycin coated tubes showed no biofilm development. Promising, however, this is an in-vitro study involving small numbers (5 in each group).

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<div align="right">N Engl J Med 2010;363:109–22.</div>

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<div align="right">The Journal of Laryngology & Otology 2010;124:594–98.</div>

The Unfortunate Experiment debate: Manning response to Chalmers

I write to respond Professor Iain Chalmers' letter in the 30 July 2010 issue of the *Journal*. Chalmers is critical of the medical advisers to the Inquiry for not seeking 'systematically analysed and published evidence of (i) international consistency of gynaecological practice in … respect [of the treatment of carcinoma *in situ*]; and (ii) empirical research evidence justifying such consistency.'

Chalmers suffers from the misunderstanding that I was one of the medical advisers to the Inquiry. I took no part in the Inquiry. I am an academic medical lawyer and the editor of *The Cartwright Papers*.

Chalmers is very critical of the medical advisors to the Cervical Cancer Inquiry, who incidentally included Professor Eric MacKay, gynaecologist and Professor Linda Holloway, pathologist as well as Professor Charlotte Paul, epidemiologist, for the fact that Judge Cartwright relied on expert evidence to form an opinion about *conventional* treatment. Chalmers' criticism betrays a fundamental misunderstanding of the judicial process.

First, the Judge's terms of reference charged her with determining whether 'there was a failure adequately to treat cervical CIS at NWH, and if so, the reasons for that failure and the period in which the failure existed'. In order to do so, she was required to determine what constituted 'adequate' treatment for CIS in 1966 and thereafter during the period of Green's study. Hence the evidence Judge Cartwright sought was a definition of *adequate* treatment. Chalmers cites pages in the Report where the evidence for conventional treatment is summarised, complaining that '[t]hese short passages do not cite scientific articles, but simply quote the opinions of four witnesses (and one interviewee).'

Prompted by Professor Jones' criticism, Chalmers has apparently 'now consulted the Cartwright Report.' Clearly, he has still not read the whole Report. It is tempting to dismiss him as unqualified to participate knowledgeably in these debates until he has done so. In any event his reading was clearly only 'in search of the evidence [he] needed' of a definition of conventional treatment for CIS, for he misses the whole chapter called 'Adequate management of CIS' (page 103 and following).

Chalmers states he 'felt sure that [Paul] would have pointed [him] to the relevant parts of the Cartwright Report if it contained the evidence [he] sought from her.' I am happy to refer him now to the relevant part of the Report he missed. On page 106 the Judge concluded, on the basis of expert advice from eight world leaders in the gynaecological field and consideration of other views canvassed during the Inquiry, that: 'the appropriate treatment of CIS, if invasive cancer is to be avoided, is to remove the lesion. The patient must then be monitored so that further treatment can be offered if there is persisting disease or a recurrence, as evidenced by positive cytology.' (See also page 107, under the heading 'The aim of treatment').

Secondly, a judge relies on the advice of experts who can be cross-examined. It is they who cite the published evidence for their opinions. The Judge stated: 'Their evidence was derived from an examination of the medical literature, a review of research projects and personal experience in practice at various periods from the early 1950s.' (page 106) Even if the Judge had asked the experts called to give evidence to conduct a systematic review, as Chalmers thinks ought to have been done, he produces no evidence that it would have been different from the advice of the experts and the conclusion of the Judge (page 107)—that all treatments at that time were aimed at eradicating the lesion, be they by local destructive treatments, local excision, conization or hysterectomy. What Green was doing was not treating.

Chalmers criticises Paul for using uses the term 'current best practice' without defining it in *The Cartwright Papers*. He omitted to notice that Paul was quoting *Bryder's* view of what Green was doing, that the 1966 proposal was for 'conservative treatment that was 'current best practice'' (pages 23–24 of *History*). Paul disagreed with this characterisation by Bryder of Green's 1966 proposal (pages 121–122 of *The Cartwright Papers*).

Far from being an 'intemperate attack', Paul's careful analysis is based on an unimpeachable grasp of the scientific evidence. I urge the Journal's readers to read it for a full understanding of the true scale and seriousness of Bryder's errors in her *History*, rather than to rely on Chalmers' misunderstandings both of the Report and of the proper way evidence is gathered and factual findings are made on the basis of it in judicial inquiries.

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A response to Professor Bryder's comments on 'Consequences in women of participating in a study of the natural history of cervical intraepithelial neoplasia 3'

In her recent *NZMJ* Editorial,[1] Professor Linda Bryder raised six points regarding this paper which was published in *Aust & NZ J Obstet & Gynaecol*.[2] These points are listed below, each with an explanation.

1. **The definition of 'treatment of curative intent'**

    We deemed 'curative intent' to encompass procedures designed to eradicate all tissue with cervical intraepithelial neoplasia, grade 3 (CIN3), an outcome that could only be achieved with some certainty by hysterectomy, amputation of the cervix or cone biopsy. Dr Green himself pointed out in 1962 that ring biopsy, a slightly less extensive procedure than cone biopsy, was not 'definitive therapy' for CIN3.[3] We also provided a textbook reference[4] supporting this definition as it applied to international practice at the time. This is not to deny that lesser procedures sometimes, in the case of ring biopsy quite often, effect a 'cure'; moreover, there is no guarantee of 'cure' with treatment of curative intent, as can be verified by careful examination of Figure 1 of our *Lancet Oncology* paper.[5]

2. **Why was 1974, rather than 1970, chosen as the last year of the 'clinical study'?**

    Initially, we examined the data from 1955-76 (the period covered by the McIndoe paper[6]) in four 5-year periods and one 2-year period; the 1st and 2nd, and the 3rd and 4th periods, were combined to simplify presentation of the data. Figure 1 of the *ANZJOG* article[2] does indeed show that there was a lower proportion of women in Green's 'core group' in the years 1971-74 (just under 20%) than in 1966-1970 (about 50%). However, the effect of choosing 1974 instead of 1970 as the last year was to **reduce** the difference in outcomes for the women according to the period of CIN3 diagnosis (Tables 2a and 3a) but to make little difference to outcomes according to initial management (Tables 2b and 3b). 1975 was the year an internal (National Women's Hospital) inquiry released its report into the conduct of the clinical study.

    Our data show that, of women newly diagnosed with CIN3 in 1975-76, 14 (5.5%) received no more than a punch or wedge biopsy as initial management (Table 1) and 30 (11.7%) had untreated positive smears during follow-up (Table 2a). Clearly, the practice of withholding or delaying curative treatment was abandoned gradually, not abruptly.

3. **No evidence that patient records reviewed were Green's patients**

    Our analysis included all women who were newly diagnosed with CIN3 at National Women's Hospital in the years 1955 to 1976, irrespective of their clinician, provided they satisfied the inclusion criteria outlined in the paper.[2]

We did not record the name of the treating doctor. However, it is fair to assume that whenever curative treatment was withheld or delayed, it was in accordance with the policy of Dr Green's clinical study. Our aim was to ascertain the effect on women of different forms of initial management rather than to compare outcomes according to individual clinicians. It should be noted that we analysed follow-up interventions only for 10 years after initial diagnosis of CIN3 (i.e. no later than 1984 for women included in the clinical study).

4. Were women in the early 1960s more likely to be unscreened than in the period 1965–74?

As pap smears were introduced into New Zealand only in 1955,[7] the cytological abnormality that prompted the histopathological diagnosis of CIN3 would have been picked up in the first pap smear for nearly all women diagnosed in the 1955–64 period, at least in the first few years. However, as time went on, an increasing proportion of women would have had a previous pap smear.

5. A retrospective study cannot prove unethical behaviour which implies *intent* not to do the best for one's patients

In our *ANZJOG* paper,[2] we did not make the claim that its findings proved unethical behaviour. What we did acknowledge in the Background of the Abstract and in the Introduction was the unethical nature of the clinical study from which our data were derived, and supported this with independent references. The wording was included to satisfy requirements pertaining to publication of findings from studies considered unethical.

In my view, clinical research that involves giving or withholding a medical intervention cannot be deemed ethical on good intentions alone. In 1964 the Declaration of Helsinki[8] stated:

(i) "Clinical research cannot legitimately be carried out unless the importance of the objective is in proportion to the inherent risk to the subject" (section 1, paragraph 3) and "Every clinical research project should be preceded by careful assessment of inherent risks in comparison to foreseeable benefits to the subject or to others" (section 1, paragraph 4).

(ii) "… the doctor should obtain the patient's freely given consent after the patient has been given a full explanation" (section 2, paragraph 1).

We did not record information relating to informed consent. However, our results show that the inherent risk of invasive cervical or vaginal cancer was high, not low as claimed by Dr Green. Moreover, Figure 2(b) in our *ANZJOG* paper shows that half of the cancers in women managed initially by no more than a punch or wedge biopsy were diagnosed within 5 years of the CIN3 diagnosis. These are data by which to judge whether the importance of the objective (not having a cone biopsy) was in proportion to the inherent risk (invasive cancer).

6. **Where is the authors' evidence that 'follow-up' biopsies were often intended to exclude invasive cancer rather than to diagnose and treat CIN3'?**

Our wording was: "These observations are *consistent with the findings* of the judicial inquiry that follow-up biopsies often were intended to exclude invasive cancer rather than to diagnose and treat CIN3, …" (Discussion, 2nd paragraph). In relation to our own findings, we stated: "…inclusion in this clinical study subjected women to many medical interventions designed to observe rather than treat their cervical intraepithelial neoplasia,…" (Discussion, 1st paragraph). The evidence for this was, in the 'core group', the approximately 4-fold increase in frequency of positive smears (indicating persistent or recurrent CIN3) that were not followed by a treatment with curative intent, and of follow-up biopsies (Table 2b). Of the follow-up biopsies taken from women in this group in the 10-years 1965–74, only one-quarter were 'with curative intent' (unpublished data).

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References:

1. Bryder L. A response to criticisms of The History of the 'Unfortunate Experiment' at National Women's Hospital. N Z Med J 2010; 123(1319). http://www.nzma.org.nz/journal/123-1319/4240/content.pdf

2. McCredie MRE, Paul C, Sharples KJ, Baranyai J, Medley G, Skegg DCG, Jones RW. Consequences in women of participating in a study of the natural history of cervical intraepithelial neoplasia 3. Aust & N Z J Obstet & Gynaecol. 2010. (epublication ahead of print)

3. Green GH. Carcinoma in situ of the uterine cervix: conservative management in 84 of 190 cases. Aust NZ J Obstet Gynaecol 1962;2:49–57.

4. O'Dowd MJ, Philipp EE. The History of Obstetrics and Gynaecology. New York: Parthenon, 1994.

5. McCredie MRE, Sharples KJ, Paul C, et al. Natural history of cervical neoplasia and risk of invasive cancer in women diagnosed with cervical intraepithelial neoplasia 3: a retrospective cohort study. Lancet Oncol 2008; 9: 425–434.

6. McIndoe WA, McLean MR, Jones RW, Mullins PR. The invasive potential of carcinoma in situ of the cervix. Obstet Gynecol 1984;64:451–458.

7. Jones RW, Fitzgerald N. The development of cervical cytology and colposcopy in New Zealand: 50 years since the first cytology screening laboratory at National Women's Hospital. NZ Med J 2004;117(1206). http://www.nzma.org.nz/journal/117-1206/1177/

8. World Medical Association. Declaration of Helsinki: Recommendations guiding doctors in clinical research. Adopted by the 18th World Medical Assembly, June 1964.

Response to the Missing Manuscript editorial

I read with considerable disquiet the editorial about the "missing manuscript" in the debate on the Unfortunate Experiment.[1]

Three things concerned me about this editorial. The first was the suggestion that Professor Paul's career had been "built at least in part on these issues". The same could be said about any academic who publishes on any issue. Thus, in my view this is an inappropriate statement, and the reason for making it is unclear.

The second issue of concern is the statement that Professor Paul's manuscript was "very wordy". It is not usual for peer reviewers to report their views on a manuscript publically, and I believe it sets a disappointing precedent.

The third issue of concern is that the editorial seems to advocate airing the two sides of a debate, even though the scientific evidence supports only one side; that expressed by Professor Paul. The New Zealand Press Council identified a similar issue, stating that while it was obliged to uphold the freedom of publications to hold to a position against the weight of informed opinion, this could damage the credibility of the publication:

> "The Council is obliged to uphold the freedom of publications to take a position, and hold to it if they choose, against the weight of informed opinion. No publication operates in a vacuum. All are vulnerable to criticism of their material in other media and all can suffer if their conduct costs them credibility."[2]

In my opinion, the risk to credibility is even greater for a scientific journal, and the credibility of the New Zealand Medical Journal has been damaged.

Ann Richardson
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References:

1. Frizelle FA. The missing manuscript. NZ Med J 2010; 123: 11-13.
   http://www.nzma.org.nz/journal/123-1319/4252/
2. New Zealand Press Council. Case number 2110. Professor Charlotte Paul against the New Zealand Listener. March 2010
   http://www.presscouncil.org.nz/display_ruling.php?case_number=2110

Congratulations to the NZMJ for the Unfortunate Experiment theme issue

We would like to congratulate the *New Zealand Medical Journal* on an excellent and thought-provoking issue, covering the controversy and reawakened interest in Dr Herbert Green's "unfortunate experiment" at the National Women's Hospital.

Dr Graeme Overton[1] offers a wonderfully clear explanation of the key problem with the 1984 paper by McIndoe and colleagues.[2] He points out that, not only were the two groups retrospectively divided on the basis of persistent abnormal cervical cytology during follow-up and not prospectively as experimental groups for comparison of different treatment strategies, but that the paper reports absolutely no discernible differences in the initial treatments received by these two groups, with similar proportions having initially undergone hysterectomy (an unthinkably radical treatment by today's standards), cone biopsy and other procedures.

Unfortunately, the same level of attention has not been paid to the similar problem in the 2008 paper by McCredie and colleagues.[3] This paper states clearly that the authors divided their sample into adequately and inadequately treated groups. However, a major problem in their methods is that they use the **outcome following treatment** as part of the classification system:

> "Any procedure followed by a positive smear in the following 6–24 months was classified as inadequate treatment (Figure 1). Four women who developed cancer within 2 years of CIN3 diagnosis, but who had no follow-up cytology, were assumed to have had inadequate treatment."

Given that they partially base the classification of adequacy of treatment on outcomes, it is totally unsurprising and uninformative that the authors should find that women who are classified as "inadequately treated" have poorer outcomes. It is difficult to follow exactly what this paper was trying to prove, but as a means to demonstrate that conservative treatment led to worse outcomes, the methods are wholly inadequate.

We therefore suggest that the *New Zealand Medical Journal* editorial[4] errs in citing this paper as evidence that women were harmed. It provides no such evidence.

It is regrettable that Professors Paul and Jones, co-authors of the McCredie paper, should have turned down the opportunity to provide a scientific defence of their approach for this issue of the *New Zealand Medical Journal* and we applaud Professor Frizelle for his decision to continue with publication in the face of those difficulties.[4]

(We declare we have no conflicts of interests.)

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References:

1. Overton GH. The 1987 National Women's Hospital (NWH) 'Unfortunate Experiment'. Accusations of unethical experiments and undertreatment, resulting in excess deaths from cervical cancer. Facts and fables. N Z Med J. 2010;123(1319):101-104. http://www.nzma.org.nz/journal/123-1319/4244/content.pdf

2. McIndoe WA, McLean MR, Jones RW, Mullins PR. The invasive potential of carcinoma in situ of the cervix. Obstet Gynecol. 1984 Oct;64(4):451-8.

3. McCredie MRE, Sharples KJ, Paul C, Baranyai J, Medley G, Jones RW, Skegg DCG. Natural history of cervical neoplasia and risk of invasive cancer in women with cervical intraepithelial neoplasia 3: A retrospective cohort study. Lancet Oncol. 2008 May;9(5):425-34.

4. Frizelle FA. The missing manuscript. N Z Med J. 2010;123(1319):11-13. http://www.nzma.org.nz/journal/123-1319/4252/content.pdf

Smoker (mis)perceptions associated with pack colouring: national survey data

Background—Several studies have concluded that "light" and "mild" descriptors on cigarette packs lead smokers to assume that cigarettes labelled in this way pose a lower health risk than "full flavour" or "regular" cigarettes.[1–4]

In response to the bans several countries have imposed on these descriptors, the tobacco industry has introduced "colour coded" packs and specific pack colours for different brand variants,[5] a pattern that is also evident in New Zealand.[6,7] As a result, smokers have been conditioned to interpret lighter pack colours (e.g. white, silver or blue) to signify "lighter" cigarettes.[5]

This is a health issue given that smokers mistakenly believe cigarettes from lightly coloured packs are less harmful and less addictive.[8 9] We therefore aimed to determine how New Zealand smokers interpret cigarette pack colouring.

Methods—The New Zealand arm of the International Tobacco Control Policy Evaluation Survey (ITC Project) derives its sample from the New Zealand Health Survey (a representative national sample with boosted sampling of Māori, Pacific and Asian NZers). In wave two, conducted between March 2008 and February 2009 (n=923 respondents), we asked about perceptions of tobacco packaging. Further detail on the survey methods are available in an online *Methods Report*[10] and in related publications.[11 12]

Results—Around a third of smokers said that they obtained at least some "useful information on how cigarettes taste" from the pack colour (35.3%, 95%CI=30.9–39.7). This was less than the equivalent ratings for tar and nicotine levels of the brand (43.5%); brand descriptor words such as "smooth" and "ultra" (50.4%), and "light" and "mild" (65.1%). The latter is notable since at this time of this wave two survey most tobacco packaging no longer used "light" and "mild" descriptors even though the Commerce Commission Inquiry into this matter did not report until September 2008.[13]

Obtaining information about taste from pack colour was reported more frequently in younger age groups (p-value for trend: p<0.00001) (Figure 1). This was also the pattern for increasing individual deprivation scores (p-value for trend: p=0.0002) (Figure 2). Māori and other ethnic groups also showed this pattern compared to European smokers, but not at a statistically significant level (Figure 3).

In two multivariate models, younger smokers were significantly more likely to report that the pack colour provided useful information on taste (but in the fully-adjusted "Model 3" this was only statistically significant for the comparison between the 35–49 age group and the 50+ age group; i.e. adjusted odds ratio=1.88; 95%CI=1.07–3.30). This model adjusted for variables relating to demographics, socioeconomic position, mental health, and smoking-related beliefs/behaviours (the full table of results is available on request).

Figure 1. Percentage of New Zealand smokers who believe that pack colouring provides useful information about cigarette taste by age group
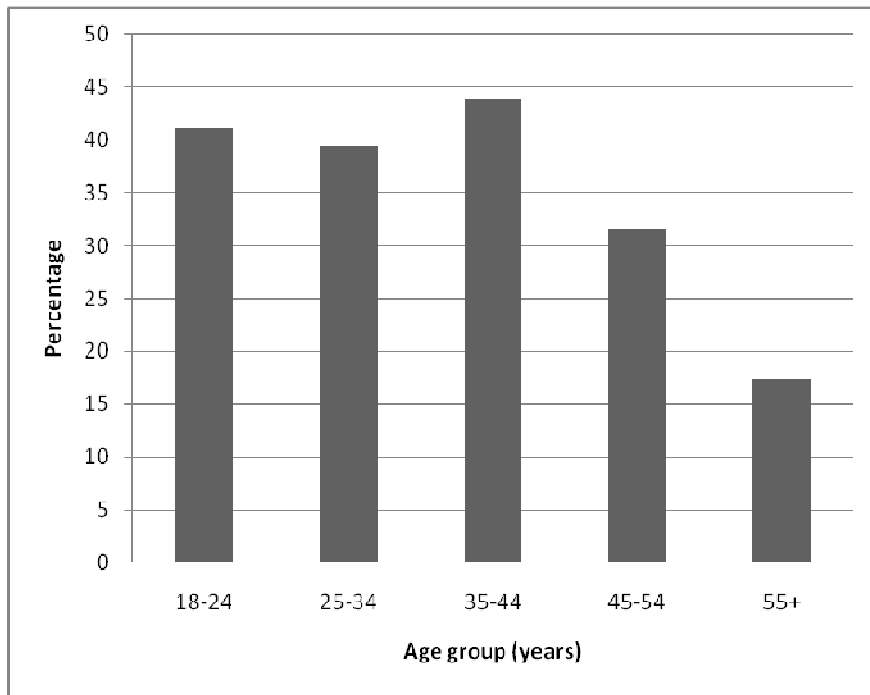

Figure 2. Percentage of New Zealand smokers who believe that pack colouring provides useful information about cigarette taste by individual deprivation level (NZiDep)
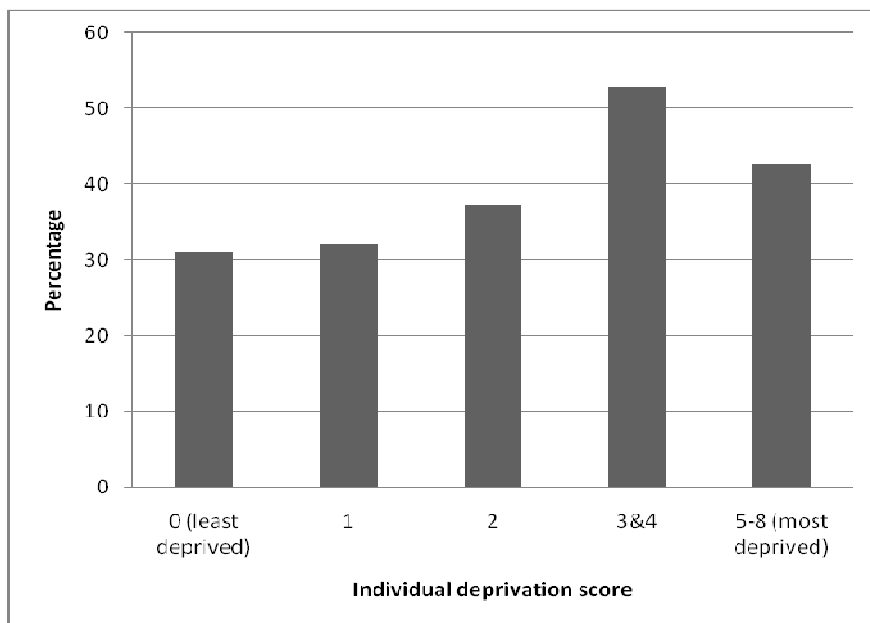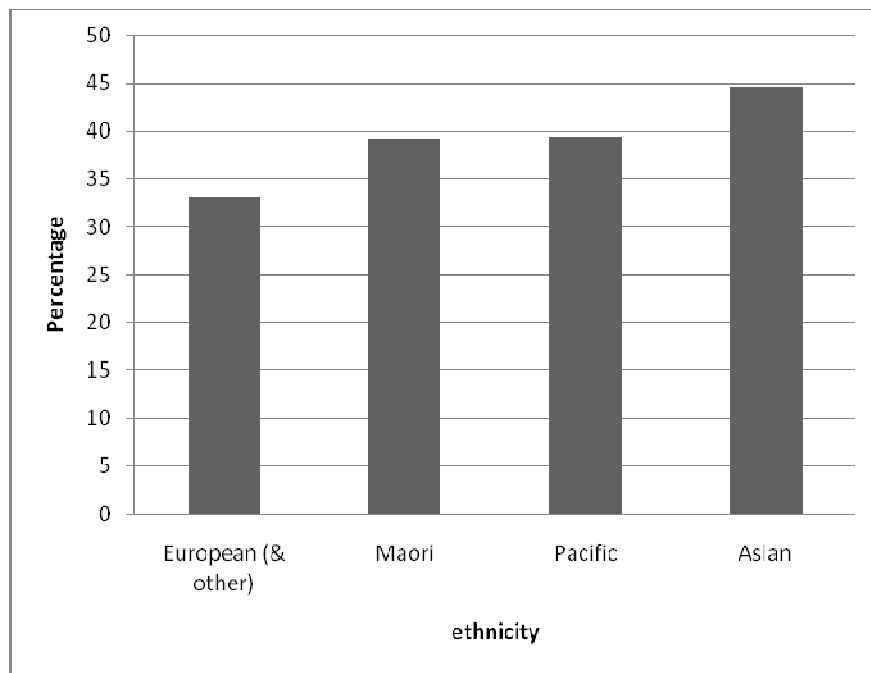
Figure 3. Percentage of New Zealand smokers who believe that pack colouring provides useful information about cigarette taste by ethnicity

Discussion—These findings indicate that New Zealand smokers (and especially younger smokers) commonly say that pack colour gives them useful information about cigarette taste. Such findings are consistent with the international literature that is more specifically focused on pack colour and perceptions of health risk.[8,9] Given this picture along with the evidence of misperceptions concerning "light" cigarettes (including for New Zealand[14]) and findings that some smokers believe that cigarettes tasting "less strong" are less harmful,[2] differential pack colouring is likely to maintain and reinforce these false beliefs.

Together with evidence that New Zealand smokers frequently hold various other health risk misperceptions,[15] these findings add further weight to calls for precautionary policy responses. These could include regulations introducing plain packaging of tobacco products (as planned by the Australian Government[16]) with all packs mandated to be a single colour. Alternately, regulations could require larger pictorial health warnings on packs, which would displace coloured surfaces and branding imagery.

Such policies could play an important supplementary role in a clear endgame strategy that rapidly halts the tobacco epidemic in New Zealand (e.g. by phasing out sales; see references[17–19] for further details).

Given the current inquiry into the tobacco industry by the Māori Affairs Select Committee, we suggest the established body of evidence regarding deceptive tobacco packaging merits a robust policy response.

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References:

1. Borland R, Yong HH, King B, et al. Use of and beliefs about light cigarettes in four countries: findings from the International Tobacco Control Policy Evaluation Survey. Nicotine Tob Res. 2004;6 Suppl 3:S311-21.

2. Shiffman S, Pillitteri JL, Burton SL, et al. Smokers' beliefs about "Light" and "Ultra Light" cigarettes. Tob Control. 2001;10 Suppl 1:i17-23.

3. Shiffman S, Pillitteri JL, Burton SL, et al. Effect of health messages about "Light" and "Ultra Light" cigarettes on beliefs and quitting intent. Tob Control. 2001;10 Suppl 1:i24-32.

4. Kropp RY, Halpern-Felsher BL. Adolescents' beliefs about the risks involved in smoking "light" cigarettes. Pediatrics. 2004;114:e445-51.

5. Wakefield M, Morley C, Horan J, et al. The cigarette pack as image: new evidence from tobacco industry documents. Tob Control. 2002;11:i73-i80.

6. Peace J, Wilson N, Thomson G, et al. Recent changes in cigarette packaging in New Zealand may continue to mislead smokers. N Z Med J. 2008;121:U2908.

7. Peace J, Wilson N, Hoek J, et al. Survey of descriptors on cigarette packs: still misleading consumers? N Z Med J. 2009;122:90-6.

8. Hammond D, Dockrell M, Arnott D, et al. Cigarette pack design and perceptions of risk among UK adults and youth. Eur J Public Health. 2009;19:631-7.

9. Hammond D, Parkinson C. The impact of cigarette package design on perceptions of risk. J Public Health (Oxf). 2009;31:345-53.

10. Wilson N. Methods report for the New Zealand arm of the International Tobacco Control Policy Evaluation Survey (ITC Project) (Updated 2009). Wellington: University of Otago, Wellington, 2009. http://www.wnmeds.ac.nz/itcproject.html

11. Wilson N, Blakely T, Edwards R, et al. Support by New Zealand smokers for new types of smokefree areas: national survey data. N Z Med J. 2009;122:80-9.

12. Wilson N, Weerasekera D, Edwards R, et al. Characteristics of smoker support for increasing a dedicated tobacco tax: National survey data from New Zealand. Nicotine Tob Res. 2010;12:168-73.

13. Commerce Commission. Consumers warned 'light' and 'mild' tobacco likely to be just as deadly as regular strength (Media Release, 24 September). Wellington: Commerce Commission, 2008. http://www.comcom.govt.nz/media-releases/detail/2008/consumerswarnedlightandmildtobacco/

14. Wilson N, Weerasekera D, Peace J, et al. Misperceptions of "light" cigarettes abound: national survey data. BMC Public Health. 2009;9:126.

15. Wilson N, Thomson G, Weerasekera D, et al. Smoker misperceptions around tobacco: national survey data of particular relevance to protecting Maori health. N Z Med J. 2009;122:123-7.

16. Sweet M. Australia to mandate plain packs for cigarettes as part of new tobacco control offensive. BMJ. 2010;340:c2401.

17. Wilson N, Edwards R, Blakely T, et al. Submission to the: Inquiry into the tobacco industry in Aotearoa and the consequences of tobacco use for Māori. Wellington: University of Otago, , 2010. http://www.parliament.nz/NR/rdonlyres/FA7DEFFA-0AFF-4CC8-85C6-CD5B805D34C9/128156/49SCMA_EVI_00DBSCH_INQ_9591_1_A31755_UniversityofO.pdf

18. Thomson G, Wilson N, Blakely T, et al. Ending appreciable tobacco use in a nation: Using a sinking lid on supply. Tob Control (In press).

19. Laugesen M, Glover M, Fraser T, et al. Four policies to end the sale of cigarettes and smoking tobacco in New Zealand by 2020. N Z Med J. 2010;123(1314):55-65. http://www.nzma.org.nz/journal/123-1314/4107/content.pdf

Legal blood alcohol limit: has New Zealand missed a golden opportunity?

The New Zealand Government recently announced (26 July 2010) that it will decide "...whether or not to lower the legal blood alcohol limit after conducting New Zealand-specific research on the level of risk posed by drivers with a blood alcohol limit of between 0.05 and 0.08." This research will be carried out over a period of 2 years" beginning after a law change due to be implemented by early next year.[1]

If the results of any New Zealand-specific research indicate lives will be saved by lowering the blood alcohol concentration (BAC), then the current limit could presumably be lowered shortly afterwards. Thus, a change in the law regarding BAC could be seen in around 3 years.

This would seem like a considered rather than a knee-jerk response, were it not for the fact that in the NZ Ministry of Transport's 2009 discussion document 'Safer Journeys' it was stated that "New Zealand and international research has consistently demonstrated the benefits associated with BAC levels of 0.05, or lower, in saving lives and preventing serious injuries—analysis suggests that we would see similar improvements here if we lowered the BAC to 0.05. It is estimated that between 15 and 33 lives could be saved and 320 to 686 injuries prevented every year. This corresponds to between $111 million and $238 million."[2]

Given that consistent evidence already suggests lives may be saved and that the potential costs of wrongly doing nothing (failing to lower the BAC when so doing may save upwards of 40 lives over the next 3 years) appear to far outweigh the potential costs of wrongly doing something (lowering the BAC when so doing will not lower the number of alcohol related road deaths), it is hard to understand why the legal BAC will not be lowered at the earliest possible opportunity.

By failing to take into account the relative costs of the two ways in which their decision may be wrong, New Zealand may have missed a golden opportunity to save lives by failing to lower the BAC at the same time as other changes are made to laws regarding recidivist drink drivers and drivers under the age of 20 years.

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Jose Perezgonzalez
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References:

1. Joyce, S. Tackling New Zealand's drink driving problem. 26 July 2010.
   http://www.beehive.govt.nz/release/tackling+new+zealand%e2%80%99s+drink+driving+problem
2. Ministry of Transport. Discussion Document: Have your say on our next road safety strategy. August 2009.
   http://www.transport.govt.nz/saferjourneys/Documents/SaferJourneysfull_web.pdf

Laurence Allan Malcolm

Seventeen years in New Guinea proved pivotal in the life of doctor and health activist Laurence Malcolm. Working with isolated highland tribes, and then with health policy makers in Port Moresby, altered his perspectives, says his wife Lyn. "The experience reprogrammed him, " she says.

He set a new course, away from bureaucracies, towards empowering people to do things for themselves.

He left the religious faith that had been important to him, but retained its values of justice and compassion, until his death on 22 June 2010.

Christchurch associate Dr Murray Laugesen says Malcolm's experience in New Guinea "laid the foundation for the way he looked at healthcare in New Zealand", leading to his pioneering and leadership roles in planning for general practice.

Health researcher Professor Ross Barnett says Malcolm was most influential in improving the spread of New Zealand's GP workforce and in moving medical planning to a horizontal integration of groups and services.

Malcolm grew up in a Brethren family at Richmond, Nelson. He won a Junior Scholarship at Nelson College and studied medicine at Otago University. His years in Dunedin fostered a love of tramping and mountaineering. He married Irene Hodge in 1953. He served as house surgeon at Christchurch and Blenheim hospitals in 1954 and 1955, and then as a registrar at Christchurch Hospital.

For the next 17 years he worked for the Australian Government in New Guinea. Lyn says a wish to be unconventional, with a sense of missionary fervour, drove him. He, Irene and their children were the first white people that many highland tribesfolk had seen. He led the building of churches and swimming pools in two villages. Moving about the jungle brought him into the crossfire of warring tribes. He survived a plane crash in which the pilot and passenger were killed.

Medical care involved treatment of diseases new in remote areas and wounds inflicted by bows and arrows. Lack of protein in the local diet led Malcolm to initiating the growing of peanuts and sparked a lifelong interest in nutrition and growth. In his final years in the country, Malcolm developed national health plans for New Guinea, working in Port Moresby.

His stay in New Guinea was broken by a study trip to Britain, where he qualified as a Fellow of the Royal College of Physicians, in Edinburgh. Research in New Guinea brought him a Doctor of Medicine degree from Otago. He added qualifications in tropical medicine, at Sydney, and public health administration, at Massey.

He returned to New Zealand in 1974 determined to revolutionise public health planning. He persuaded the Director-General of Health to set up a Health Planning and Research Unit in Christchurch. While heading this unit, he married Lyn Wright, in 1982. He took a consultancy position with the Department of Health in Wellington in 1984 and became Professor of Community Health at the University of Otago's Wellington School of Medicine in 1985.

Malcolm moved back to Christchurch in 1995 and, with Lyn, established Aotearoa Health, a consultancy service in health policy, management and research. He wrote papers for medical journals and articles for The Press on such issues as primary and clinical care of the elderly, Māori and under-privileged sections of society. Barnett says Malcolm was "more than an academic—he was an activist: he never let go". He was a model for academics, as "the critic and conscience of society".

Malcolm was elected to the Canterbury District Health Board in 2004 but was defeated three years later, a victim of voters' disillusionment with the Christchurch 2021 political movement, of which he was part. Lyn says he planned to stand again this year but as an independent. He was a member of many national and international boards and committees. Laugesen says: "Malcolm's main passion was primary healthcare in Canterbury". He was tenacious in his research and a pioneer in planning. The unit he headed in Christchurch was the first of its kind. His ideas were "ahead of his time".

Christchurch GP Dr George Chisholm says Malcolm was a forward thinker and "very committed to the development of good integrated healthcare". He was the "backbone" of planning for aged people's care. "He was meticulous in research. He challenged a lot of medical assumptions and clarified a lot of issues, " Chisholm says. "He was committed to the absolute, critical place of primary care in the health system. He was ahead of his time in many areas. He could be controversial but he stuck to the facts as he knew them, " Chisholm says.

Lyn says her husband loved a challenge. His fearlessness in tackling the health establishment served him also in his leisure activities—from conquering high mountain peaks, to yachting in many countries, to playing the finest baroque music on his violin, to building a garden at his hillside home.

Laurence Allan Malcolm, born Richmond 8 November 1929; died Christchurch 22 June 2010. Survived by wife Lyn, daughter Anne, sons Chris, David and Geoff, seven grandchildren and one great-grandchild.

Mike Crean wrote this obituary; it first appeared in *The Press* newspaper (Christchurch).

Additional tribute by colleagues George Salmond and Ross Barnett

As longstanding research colleagues we seek to supplement the preceding general account of the life and work of Laurence Malcolm.

Malcolm's involvement with clinical work, health policy activities and service planning in New Guinea provided him with knowledge and tools that fuelled his ambition to engage in similar work when he returned to New Zealand in 1974.

At that time structured information gathering and health services research activities were just getting started in New Zealand. There was little appreciation of the contribution that could be made by the disciplined gathering, sharing and analysis of data about the allocation and use of health resources. At the time the then Department of Health has a small operations research, later a management services and research, unit based in its Wellington head office. The unit was small with limited capability and capacity.

For personal reasons Malcolm wished to work in Christchurch where with a small team of researchers he set up a subsidiary health policy and planning unit.

From the outset Malcolm's ideas and concepts about health services planning and delivery challenged conservative ways of thinking and established interests. In particular he was an early champion for moving the focus of health care delivery from hospital to community settings and, by advocating service planning, a way of improving coordination across different parts of the health sector.

Using research evidence he also demonstrated unfairness and inequalities in the way health resources were allocated and used to meet the needs of disadvantaged groups and geographically diverse populations in New Zealand.

He recognised that in a country like New Zealand with a slowly growing economy, an aging population, rising health care costs and increasing health and health care expectations, it would not be possible to sustain existing patterns of health care delivery. In future such care would simply not be affordable. This prompted his active interest in and support for service and workforce innovation and development particularly in general practice and other primary health care settings.

From modest beginnings in Christchurch Malcolm broadened and deepened his information gathering and research activities as Professor of Public Health at the Wellington School of Medicine and later in private consultancy practice based in Christchurch.

Malcolm's thinking was always ahead of its time, not only in New Guinea but also in New Zealand. Lack of research resources and information limitations sometimes made it difficult for him to convincingly justify the ideas and concepts he wanted to promote. Often challenging, and sometimes controversial, he was frequently a burr under the saddle of the health sector establishment. He was not just an academic researcher and a planner he was an activist widely promoting his work and that of his colleagues. If he felt that he was on sound ground he was a difficult man to budge, regardless of what opposition was ranged against him.

Malcolm pushed the margins. He was an explorer, a navigator and an innovator and as such he was not always right. He was a man of spirit and courage in the pursuit of those causes he had evidence for and in which he fervently believed.

He is sorely missed.

THE NEW ZEALAND MEDICAL JOURNAL

Journal of the New Zealand Medical Association

GRANTS AWARDED JULY 2010

At the July meeting of the Scientific Advisory Group of the National Heart Foundation, a total of 29 grants were awarded. The awards included 5 Project Grants, 6 Fellowships/Scholarships, 8 Small Project Grants, 1 Grant-in-Aid and 9 Travel Grants. A total of 7 Summer Studentships were also awarded to the Medical Schools at the University of Otago and the University of Auckland.

PROJECT GRANTS

Professor Timothy Buckenham

Department of Academic Radiology, University of Otago, Christchurch

Characterisation of atherosclerotic plaque using multispectral CT (MARS).

$134,134 for 3 years.

Associate Professor Ngaire Kerse

Department of General Practice & Primary Health Care, University of Auckland

At the heart of healthy ageing.

$147,191 for 2 years.

Dr Richard Troughton

Department of Medicine, University of Otago, Christchurch

The PEOPLE Study: Prospective evaluation of outcome in patients with heart failure with preserved left ventricular ejection fraction.

$163,016 for 1 year.

Dr Leigh Ellmers

Christchurch Cardioendocrine Research Group, University of Otago, Christchurch

Uroguanylin: Prognostic/therapeutic potential in cardiovascular disease.

$148,962 for 2 years.

Dr Barry Palmer

Department of Medicine, University of Otago, Christchurch

The VEGF system and survival in coronary heart disease.

$140,864 for 2 years.

FELLOWSHIPS

Dr Khang Li Looi

An Overseas Training & Research Fellowship (for 1 year) was awarded to Dr Khang Li Looi, Department of Cardiology, North Shore Hospital. Dr Looi will work as an electrophysiological fellow at Papworth Hospital, Cambridge, UK.

Dr Wan Yiu Wandy Chan

The Canham Research Fellowship (for 3 years) was awarded to Dr Wan Yiu Wandy Chan, Department of Medicine, University of Otago, Christchurch

Ms Karen Peebles

A Research Fellowship (for 3 years) was awarded to Ms Karen Peebles, Department of Surgery & Anaesthesia, University of Otago, Wellington.

Dr Maithri Siriwardena

A Research Fellowship (for 2 years) was awarded to Dr Maithri Siriwardena, Christchurch Cardioendocrine Research Group, University of Otago, Christchurch.

Mr Alexander Anderson

A Postgraduate Scholarship (for 3 years) was awarded to Mr Alexander Anderson, Auckland Bioengineering Institute, University of Auckland.

Ms Catherine Lizamore

A Postgraduate Scholarship (for 3 years) was awarded to Ms Catherine Lizamore, Department of Social Science, Parks, Recreation & Sport, Lincoln University.

SMALL PROJECT GRANTS

Professor Philip Bones

Department of Electrical & Computer Engineering, University of Canterbury

Fast methods for magnetic resonance angiography (MRA).

$14,800 for 2 years.

Dr Paul Young

Intensive Care Unit, Wellington Hospital

A double-blind randomised controlled trial of remote ischaemic preconditioning in high risk adult cardiac surgery.

$15,000 for 1 year.

Ms Lana Jago

Department of Psychology, University of Auckland

Assessment of an online self-management programme to improve adherence in heart failure patients.

$12,016 for 1 year.

Dr Denis Loiselle

Department of Physiology, University of Auckland

The efficiency of fish oil-fed hearts.

$14,860 for 2 years.

Dr Ralph Maddison

Clinical Trials Research Unit, University of Auckland

Development of an m-health physical activity module.

$14,965 for 6 months.

Dr Anna Pilbrow

Christchurch Cardioendocrine Research Group, University of Otago, Christchurch

Genetic risk for heart disease – a role for small regulatory RNAs.

$14,735 for 1 year.

Dr Paula Skidmore

Department of Human Nutrition, University of Otago, Dunedin

Physical fitness and its relationship to body composition, food choice and activity in Dunedin adolescents. The Otago School Students Lifestyle Survey 2 Fitness Testing Study.

$14,928 for 18 months.

Professor Janet Hoek

Marketing Department, University of Otago, Dunedin

Defining and estimating informed choice among young adult smokers and non-smokers.

$14,895 for 5 months.

GRANT-IN-AID

Associate Professor Michael Hamlin

Department of Social Science, Parks, Recreation, Tourism & Sport, Lincoln University

Elite Ageing: How does lifelong high intensity exercise influence physiological decline?

$5,155.

TRAVEL GRANTS

Ms Denise Barlow

Cardiac Care Department, The National Heart Foundation

Attendance at Motivational Interviewing Network of Trainers (MINT) Training for New Trainers (TNT) programme, Singapore.

Associate Professor Vicky Cameron

Christchurch Cardioendocrine Research Group, University of Otago, Christchurch.

European Society of Cardiology Congress, Stockholm, Sweden.

Mr Carlos Cheung

Department of Biological Science, University of Auckland

15th Annual Meeting of the European Council for Cardiovascular Research, Nice, France; and 1st International Diabetes & Obesity Forum, Athens, Greece.

Dr Bridget Leonard

Auckland Bioengineering Institute, University of Auckland

CSANZ Annual Scientific Meeting, Adelaide, Australia.

Dr Christopher Pemberton

Department of Medicine, University of Otago, Christchurch

European Society of Cardiology Congress, Stockholm, Sweden.

Ms Katrina Poppe

Department of Medicine, University of Auckland

European Society of Cardiology Congress, Stockholm, Sweden.

Ms Elizabeth Theakston

Auckland Bioengineering Institute, University of Auckland

IEEE Engineering in Medicine & Biology Conference 2010, Buenos Aires, Argentina.

Dr Anne von Zychlinski-Kleffman

Department of Biochemistry, University of Otago, Dunedin

9th HUPO (Humane Proteome Organisation) 2010 Annual World Congress.

Mr Andrew Waa

Department of Public Health, University of Otago, Wellington

Asia Pacific Conference on Tobacco or Health: FCTC in the Asia Pacific: Change, Challenge & Process, Sydney, Australia.

Mosby's Dictionary of Medicine, Nursing, and Health Professions: 2nd Australian and New Zealand Edition

P Harris, S Nagy, N Vardaxis (eds). Published by Mosby (Elsevier Australia), 2010. ISBN 9780729539098. Contains 2015 pages & over 2400 high quality full-colour illustrations and photographs. Price AU$83.70 (online price at Elsevier Australia)

This is an attractive comprehensive medical and nursing dictionary that has special application to Australasia.
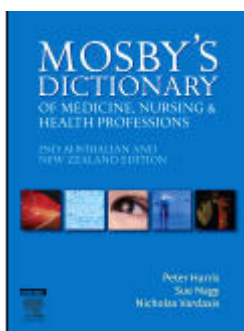
New Zealand specific names such as PHARMAC are included. It comprises some 2000 pages including appendices on units of measurement, medical terminology, medication—clinical calculations, drug interaction, and infection control.

There is access to additional appendices on line. These include an Assessment Guides, a guide to complementary and alternative medicine, and comprehensive information about health organizations in Australia and New Zealand.

My review copy did not provide web access so I am not able to comment on the content or quality of these. The dictionary is very clear, is written in easily understood language with many colour photographs and illustrations. A colour atlas of anatomy is included. This serves only as a general guide, and is not detailed, but for which there is an appendice with web access.

For those requiring an excellent dictionary, I have no hesitation in recommending it.

Jim Reid
Head of Department
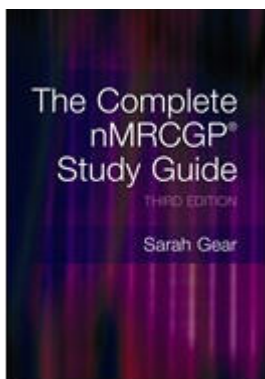Department of General Practice
University of Otago, Dunedin

The Complete nMRCGP Study Guide (3rd edition)

Sarah Gear. Published by Radcliffe Publishing, 2008. ISBN-13: 9781846192968. Contains 352 pages. Price £27.95

This was an interesting book—as the title states it is a guide to candidates for the MRCGP examination which of course is UK based.

This examination has some parallels with the Primex of the FRNZCGP, and the clinical component of this book (which in this section deals with common conditions presenting and being managed in general practice) would be of benefit to candidates for this, and they could do worse than at least leaf through the 200 pages which provides a perspective on what should be covered in study. As well as listing these conditions the book provides immediate source of further information – guidelines, source papers, studies etc where the reader can find web addresses and journal references.

As it is written for UK candidates, it is very much oriented in that direction, and much of the non clinical section (125 pages) has little direct application for New Zealand, except for an excellent short chapter on applied statistics—all you need to know in 10 pages (almost)!

This is an excellent well written book—well worth buying for a RNZCGP Primex candidate, but it is of little use beyond that stage as the GPEP2 is very different from the English model.

Jim Reid
Head of Department
Department of General Practice
University of Otago, Dunedin

## Comprehensive Review for the NCLEX-PN Examination (4th edition)

Linda Anne Silvestri. Published by Saunders Elsevier, 2010. ISBN 9781416047308. Contains 1040 pages. Price AU$54.00 (online price at Elsevier Australia)

This book is written for nurses preparing to sit the National Council Licensing Examinations (NCLEX), a prerequisite for obtaining a practicing certificate to work in most of the USA.
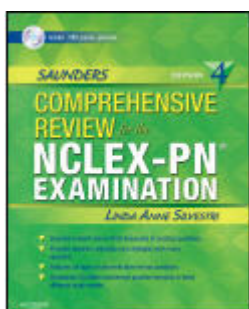
This version is for those seeking license to work as an LPN, therefore it is not at RN level. However, it is a very useful resource for nurses in many areas of practice and would be a valuable addition to any library or facility, from tertiary hospitals, medical centres, smaller hospitals, schools of nursing libraries and individuals. The content is comprehensive in scope; material is well presented, easy to follow and covers all areas of practice.

It is suitable for a beginning nurse, yet is equally useful for experienced nurses to review and update their knowledge base. It would also be useful for nurses changing their area of practice for it provides excellent information to enable a quick mastering of the new information.

The book's strength stems from the experienced writers of the Units; experts in their own fields. The way it is set out, makes it easy to follow. Each Chapter in a Unit identifies the specific relevant points covered in that chapter; includes multichoice questions to test learning and then provides the answers with an accompanying rationale.

The questions are structured in a way that reflects Bloom and requires not just recall but actual application of knowledge, encouraging critical thinking in the process. Learning is clearly focused on the needs of the patient or client in any particular scenario and what the nurse would be expected to do.

The accompanying CD provides a range of opportunities to test knowledge, reviewing the various categories of focus and the correct answers to reinforce learning. The CD includes a 100 question practice exam.

I will find this book useful in my work as a Nurse Educator.

Sue Bye
Nurse Educator
Christchurch Hospital

Caribbean Diseases: Doctor George Low's Expedition in 1901-02

Gordon C Cook. Published by Radcliffe Publishing, 2009. ISBN-13: 9781846193453. Contains 238 pages. Price £65

George Carmichael Low (1872–1952), following a distinguished undergraduate and early post-graduate career, joined Dr (later Sir) Patrick Manson in 1899, in the newly-founded London School of Tropical Medicine.
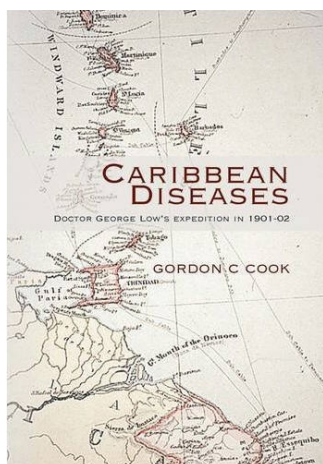
In 1901–02, at the height of his research powers, Low undertook a demanding tour of the Caribbean where he made important contributions to the understanding of the filariases and assisted in malaria eradication. He contributed significantly to disease prevention strategies, not then widely appreciated and often resisted.

Low kept Manson well-informed of his work in the Caribbean islands. In his book, Caribbean Diseases, Gordon C Cook gives a remarkably detailed analysis of 31 letters Low wrote to Manson that record Low's epidemiological observations and work sectioning mosquitoes to delineate life-cycles.

The letters paint a picture of a man, with unbridled energy, unravelling the mysteries of disease transmission. Letters 25–28 give his accounts of an outbreak of severe jaundice at Castries, St Lucia. Not yet 30 years old and inexperienced in disease prevention, Low showed remarkable maturity in the measures he introduced. Was it malignant malaria (*P. falciparum*) or yellow fever? Low's careful analysis showed clear evidence that this outbreak involved both malaria and yellow fever. These letters alone make reading the book well-worthwhile.

In addition to the letters, Cook summarises the considerable contributions Low made to scientific knowledge and disease prevention that resulted from his Caribbean experience.

Cook asks the question, 'Why so little is known about George Carmichael Low?' In his lifetime, Low was a towering figure in the field of tropical medicine. However, Cook asks the question: 'Why was Low underrated both then and now?' Living in the shadow of Manson was certainly not helpful. In his book, Cook has done much to right this.

This book will be of interest to historians, especially of tropical medicine, and those seeking inspiration in research.

H Bramwell Cook
Formerly Gastroenterologist at Christchurch Hospital