THE NEW ZEALAND MEDICAL JOURNAL
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THE NEW ZEALAND MEDICAL JOURNAL
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This Issue in the Journal

Implication of using estimated glomerular filtration rate (GFR) in a multi ethnic population of diabetes patients in general practice
Grace Joshy, Tesa Porter, Clem Le Lievre, Jane Lane, Mike Williams, Ross Lawrenson

This study looked at the prevalence of chronic kidney disease among diabetes patients in New Zealand. Estimated Glomerular Filtration Rate (eGFR) is increasingly used as a marker for identifying early renal failure. This study measured the agreement between two equations commonly used in the calculation of eGFR. There were significant differences in agreement between the two equations: the MDRD and the CG. While CG equation identifies more European of both genders, more Māori females were identified by MDRD. MDRD equation may be overestimating CKD among Māori females. Each ethnic subpopulation may need to be validated separately, and by gender.

Ethnic disparities in causes of death among diabetes patients in the Waikato region of New Zealand
Grace Joshy, Chanukya Kamalinie Colonne, Peter Dunn, David Simmons, Ross Lawrenson

Maori diabetes patients experienced significantly higher risk of mortality compared with Europeans with diabetes. Compared with European diabetes patients, Maori diabetes patients are more likely to die from cardiovascular disease, cancer and renal disease. Maori with diabetes are more likely than Europeans with diabetes to have diabetes reported on death certificates. The results suggest that the under-coding of diabetes on death certificates in New Zealand has not improved and continues to be a major limitation for routine mortality analysis solely based on these codes. Mortality among diabetes patients in New Zealand would need to be compared with that among people without known diabetes, to estimate the true burden due to diabetes.

Exploring physical and psychological wellbeing among adults with Type 2 diabetes in New Zealand: identifying a need to improve the experiences of Pacific peoples
Charlotte A M Paddison

In this study we explored levels of physical and psychological wellbeing among adults with Type 2 diabetes in New Zealand. Our results showed that the majority (58%) of people in this study had difficulty achieving good control of their diabetes and did not meet the clinical targets set by health professionals. We already know that physical health outcomes for people with Type 2 diabetes vary by ethnicity; our study shows that psychological outcomes such as diabetes-related distress also differ across ethnic groups—with Pacific peoples worst affected. Our findings show that we need

to improve the experiences of Pacific peoples with Type 2 diabetes. This includes improving metabolic control, and also working with Pacific patients to address any concerns about medication and to reduce emotional distress about diabetes.

Standardisation of reporting haemoglobin A1c: adoption of the New Zealand Society for the Study of Diabetes (NZSSD) position statement
Chris Florkowski, Michael Crooke

New Zealand, in line with other countries has now implemented a system of new units for reporting HbA1c for monitoring long-term glucose control in diabetes. Our paper traces the background and rationale for this important change.

How well does routine hospitalisation data capture information on comorbidity in New Zealand?
Diana Sarfati, Sarah Hill, Gordon Purdie, Elizabeth Dennett , Tony Blakely

Comorbidity refers to diseases or disorders that coexist with a disease of interest. Comorbidity is common and it is being increasingly recognised as being important in health-related research and policy. This paper compares routinely collected comorbidity data (collected by coders based in District Health Boards and sent electronically to NZ Health Information Systems) with comorbidity data collected directly from patients' hospital notes by a physician. We found that although (expected) differences in these two sources of data exist, administrative data provides a useful and relatively accessible source of information on comorbidity in New Zealand.

A population-based approach to the estimation of diabetes prevalence and health resource utilisation
James Smith, Gary Jackson, Brandon Orr-Walker, Rod Jackson, Siniva Sinclair, Simon Thornley, Tania Riddell, Wing Cheuk Chan

This study showed that diabetes is common in Counties Manukau and throughout the northern region, particularly in Māori and Pacific communities. It combined data that is normally used for administrative purposes with two other sets of data, to look at diabetes rates and the use of healthcare resources. All data were adjusted to avoid identification of individuals. This method has the advantage of providing comprehensive information about diabetes in a way that is timelier and less expensive than traditional survey methods, allowing better planning for the diabetes epidemic.

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Coming to grips with chronic kidney disease

John Collins

The 2002 classification of renal dysfunction as chronic kidney disease (CKD) independent of the underlying cause—with 5 stages based on glomerular filtration rate estimates—was an important advance in the diagnosis and management of progressive kidney disease.[1] Subsequent reports from a number of countries, including Australia and the USA,[2] have documented a CKD prevalence in excess of 12% in their populations although some have questioned the validity of these estimates.[3] No national study has been undertaken in New Zealand and there is no national CKD surveillance programme under consideration as has been proposed elsewhere.[4]

Joshy et al in this edition of the *Journal* report on the prevalence of CKD stages 3–5 in a cohort of primary care patients with diabetes.[5] They used the older Cockcroft-Gault and newer MDRD formulae to estimate GFR utilising the serum creatinine measurement and demographic measures.

They found differences in CKD estimates, as have others,[6] emphasising the imprecision of these screening tools. Some of this imprecision, particularly in stage 3 CKD, will be addressed by the future introduction of newer formulae such as the CKD-EPI formula which has been validated in a large US population.[7] Nevertheless a degree of imprecision will remain, but is clinically tolerable, given that the role of a screening tool is to focus strategies of diagnosis and therapy.

The simplest way to identify kidney injury is to estimate the degree of albuminuria. Recent large longitudinal studies have shown that the presence of albuminuria is an important prognostic factor for CKD progression, cardiovascular events and mortality.[8]

After considerable international debate[9] a recent Nephrology Consensus Conference held in 2009 determined that levels of albuminuria will be part of a reclassification of CKD likely to be promulgated by the International Renal Guideline Group KDIGO in the near future.[10]

Joshy et al found the presence of albuminuria in 51% of Māori with diabetes suggesting a high level of kidney injury present in this ethnic group. This prevalence of kidney injury signals the inevitability of a high future incidence of end stage kidney failure, other serious comorbid events and premature mortality.[11] There is already a 14-fold higher incidence of diabetic end stage kidney disease amongst Māori compared to those of European origin.[12]

While it is well established that effective antihypertensive therapy, particularly utilising blockers of the renin angiotensin aldosterone system, targeting a blood pressure (BP) of <130/80 mmHg is associated with a reduction in progression of CKD and reduction in the incidence of CVAs and cardiovascular events,. these targets were achieved in <30% of patients in the study by Joshy et al. When proteinuria exceeds 1

gram/24 hours the recommended target BP is even lower at 125/75 mmHg[13] and fewer patients still will be achieving that goal.

Effective innovative strategies to improve BP management in this population are urgently needed if we are to reduce the high mortality and comorbid event rates. Such approaches will require a shift in the standard paradigm of consultation-based health care with a strong emphasis on finding ways to ensure that BP targets are being consistently achieved in most patients. While screening strategies need to be broadly focused there is a need to tailor management to individual patient circumstances and clinical need.

Changes from the current approach could include more frequent clinic visits, practice nurse community contact and visits, wider utilisation of home BP monitoring, a closer relationship with pharmacies to ensure medications are being accessed appropriately, a closer collaborative partnership between primary health care and the DHB Diabetes and Renal Specialist Services along with a renewed focus on the importance of lifestyle modification with an emphasis on minimising salt intake to optimise BP control.

Business as usual will not address these important issues.

Competing interests: None known.

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Implication of using estimated glomerular filtration rate (GFR) in a multi ethnic population of diabetes patients in general practice

Grace Joshy, Tesa Porter, Clem Le Lievre, Jane Lane, Mike Williams, Ross Lawrenson

Abstract

Aim To estimate the prevalence of chronic kidney disease (CKD) among diabetes patients in New Zealand, using estimated Glomerular filtration rate (eGFR); to measure the agreement between the Modification of Diet in Renal Disease (MDRD) and Cockcroft-Gault (CG) equations in identifying CKD among Europeans and Māori; to review the metabolic control and treatment in patients with evidence of CKD.

Method Diabetes patients were identified though general practice records of diagnosis codes, diabetes annual reviews, prescriptions and laboratory results. The MDRD and CG equations were used to calculate the eGFR. Agreement between the two equations was expressed using Kappa statistics and was tested using McNemar's chi-square test. Logistic regression model was used to identify the predictors of CKD (eGFR<60 ml/min/1.73m^2).

Results Overall prevalence of CKD among diabetes patients was 19.5% (MDRD) and 23.5% (CG). Māori were significantly more likely to have CKD [Odds-ratio 1.8(1.2–2.8)]. There were significant differences between the MDRD and the CG equations in identifying patients with CKD. While CG equation identifies more European of both genders, more Māori females were identified by MDRD.

Conclusion Patients with decreased eGFR who do not have proteinuria or microalbuminuria might benefit from more intensive management of blood pressure. MDRD equation may be overestimating CKD among Māori females. Each ethnic subpopulation may need to be validated separately, and by gender.

Chronic kidney disease (CKD) among diabetes patients is increasing in incidence globally.[1] CKD is classified into five stages based on severity as below (Table 1)[2] using glomerular filtration rate (eGFR).

Table 1. The five stages of chronic kidney disease

Stage	Description	eGFR ml/min/1.73m2
1	Kidney damage with normal or ↑ GFR	≥ 90
2	Kidney damage with mild ↓ GFR	60–89
3	Moderate ↓ GFR	30–59
4	Severe ↓ GFR	15–29
5	Kidney failure	<15 (or dialysis)

It is estimated that 16% of New Zealanders have some form of kidney damage. Diabetes has been the primary cause of 36 to 45% of cases of end stage kidney disease (CKD stage 5) in New Zealand patients between 1999 and 2004,[3] with significant ethnic differences in incidence.[4] Surveys in primary care patients with diabetes have identified 24%[5]–31%[6] with evidence of CKD (eGFR <60 ml/min/1.73m^2). CKD remains the second most likely cause of death and morbidity after cardiovascular disease in diabetes, and CKD is a major independent risk factor for cardiovascular disease.[7]

Evidence suggests that progression to kidney failure in patients with diabetes can be delayed or prevented by controlling blood sugar levels and blood pressure and by treating proteinuria.[8–10] The key is detecting chronic kidney disease in its earliest, most treatable stages. Primary care physicians have been encouraged to test for macro and micro albuminuria and to estimate the albumin-creatinine ratio (ACR). It has also been suggested that estimating the glomerular filtration rate is a more sensitive method of identifying early renal failure.[5]

The eGFR, calculated by using the MDRD equation (named after the US Modification of Diet in Renal Disease Study[11]), detects chronic kidney disease more accurately than does the serum creatinine level alone. The eGFR rate also is used for disease staging.

Using the MDRD equation, laboratories are now able to routinely report eGFR derived from the serum creatinine concentration, age and gender. It does not require body surface-area measurements. In their position statement, the Australasian Creatinine Consensus Working Group, recommended that an eGFR based on the abbreviated MDRD formula be reported with every request for serum creatinine in patients over the age of 18 years.[12]

Over 69% of New Zealand laboratories report eGFR results with most requests for serum creatinine in patients aged >18 years.[13] New Zealand Guidelines Group[14] recommends calculation eGFR using CG[15] method which uses age, serum creatinine, gender, body weight and height or using the MDRD formula.[11] There is concern over the validity of either method in Māori.

The MDRD calculation makes an adjustment for ethnicity in the case of black Americans, but no such adjustment factor has been developed for other non-European ethnicities including Māori.[16] Consequently, we wanted to investigate the prevalence of CKD in a population of New Zealand patients with diabetes and measure the agreement between the MDRD and CG formulae in identifying CKD among both Europeans and Māori in New Zealand.

Key indicators of quality treatment in patients identified with early CKD include good glycaemic control, management of blood pressure to agreed targets, the use of ACE inhibitors to reduce progression of renal disease and use of statins to reduce the risk of cardiovascular disease.[14,17] We have reviewed the management of diabetes in patients with evidence of CKD by comparing blood pressure control, glycaemic control, the use of ACE and the use of statins among patients with or without evidence of renal disease.

Method

A cross-sectional survey was conducted on all patients registered with 10 practices within the Rotorua General Practice Group in Rotorua New Zealand. The survey identified all patients registered with the practices on 1 July 2007. Patients with diabetes were identified by searching electronic patient records for diagnostic code for diabetes, "Get Checked" diabetes annual review (DAR), prescription of insulin or oral hypoglycaemic, laboratory records of HbA1c greater than 6.5%.

Records were reviewed, for patients who were identified from prescriptions or laboratory records but did not have a diabetes code, to confirm the diagnosis of diabetes. Information on metabolic control, blood pressure, body measurements and treatments (Statin or ACE prescription) were extracted either from the DAR database or from patient records where it was not otherwise available. We excluded newly diagnosed patients as they may not have had time to be fully assessed or optimum treatment to be instituted. Both MDRD and CG formulas were used to calculate eGFR.

$$\text{MDRD eGFR} = 186 \times \left[\frac{\text{Serum Creatinine}}{88.4}\right]^{-1.154} \times \text{Age}^{-0.203} \times (0.742 \text{ if female})$$

$$\text{CG eGFR} = \frac{(140 - \text{Age}) \times \text{Weight} \times 1.04 \text{ x } 1.73}{\text{Serum Creatinine}} \div (0.007184 \times \text{Weight}^{0.425} \times \text{Height}^{0.725}) \text{ if female}$$

$$= \frac{(140 - \text{Age}) \times \text{Weight} \times 1.23 \text{ x } 1.73}{\text{Serum Creatinine}} \div (0.007184 \times \text{Weight}^{0.425} \times \text{Height}^{0.725}) \text{ if male}$$

Estimates of eGFR could only be made in patients where age, gender, ethnicity, weight and serum creatinine were all available. Those with missing data in any of these categories have been excluded from this analysis. Microalbuminuria [ACR 2.5–29.9 mg/mmol creatinine (men), 3.5–29.9 (women)] and proteinuria (ACR ≥30) were defined as per local guidelines.[14] Ethnic and gender specific prevalence of clinically significant CKD (eGFR <60 ml/min/1.73m^2) has been calculated.

Chi-squared test was used to test differences in proportions and ANOVA was used to test differences in means. Agreement between the two formulas in identifying patients with eGFR <60 ml/min/1.73m^2 was tested using McNemar's Chi-squared test. Kappa statistics for agreement has also been reported. Logistic regression model was used to identify predictors of clinically significant CKD. All statistical analyses were performed using SAS, 9.1 (SAS Institute, Cary, NC, USA).

Results

The total population in the 10 practices was 48,545 (34,051 aged 18 or above). Of the 1819 (3.74%) diabetes patients identified, 1353 (74%) had a DAR in the last 2 years. 342 (19%) patients did not attend a DAR. 124 (6.8%) were newly diagnosed (36 Māori, 74 NZ European, 14 Others). 1796 were aged 18 or above. Glomerular filtration rate could be estimated using both MDRD and CG equations for 942 adult patients aged 18+, who had serum creatinine, body weight and height data available from DAR records. Among them, 772 (82%) were recorded as having Type 2 diabetes and 65 (7%) as having Type 1 diabetes. 105 (11%) did not have type of diabetes recorded. Clinical and demographic characteristics of these patients are summarised in Table 2.

Table 2. Demographic and clinical characteristics of diabetes patients by ethnicity and gender

Variables	European Female	European Male	Māori Female	Māori Male
N	244	331	172	195
Age (years)	66.1±13.5	64.5±13.1	58.5±13.0	59.9±11.7
Duration of diabetes (years)	8.5±6.9	8.1±6.3	9.3±8.4	8.9±8.2
BMI (kg/m^2)	30.4±7.1	30±5.3	33.4±7.1	33.2±5.9
HbA$_{1c}$ (%)	7.5±1.5	7.5±1.6	8.5±2.2	8.0±1.8
HbA$_{1c}$ (%) > 8 mmol/l	73 (29.9%)	98 (29.6%)	77 (44.8%)	76 (39.0%)
High blood pressure (mmHg)	190 (77.9%)	227 (68.8%)	126 (73.7%)	143 (73.7%)
Current smoking	31 (12.7%)	36 (10.9%)	51 (29.7%)	45 (23.1%)
Statin treated	151 (61.9%)	226 (68.3%)	103 (59.9%)	124 (63.6%)
ACE treated	139 (57.0%)	180 (54.4%)	119 (69.2%)	133 (68.2%)
Serum creatinine (umol/l)*	70 (62–82.5)	90 (78–103)	71 (61.5–89.5)	88 (76–101)
Albumin creatinine ratio (ACR)*	1.3 (0.5,3.4)	0.9 (0.4,3.7)	2.4 (0.9–12)	4.2 (1.1–21.1)
Microalbuminuria	44 (18.9%)	75 (24.1%)	44 (28.4%)	71 (39.0%)
Proteinuria	14 (6.0%)	18 (5.8%)	23 (14.8%)	35 (19.2%)
Normal	175 (75%)	218 (70%)	88 (57%)	76 (42%)

* Serum creatinine and ACR are reported as median (interquartile range). Other data are mean±SD or n (%).

Compared with Europeans, Māori patients were on average 5.9 years younger ($p<0.0001$), had higher BMI (+3.1 kg/m^2, $p<0.0001$), significantly higher rates of microalbuminuria /proteinuria (51% versus 28% among Europeans, $p<0.0001$) and higher HbA$_{1c}$ levels (42% with HbA$_{1c}$ >8% versus 30% among Europeans, $p<0.0002$). The extent of Statin and ACE therapy among Māori patients was similar to that in Europeans, but their prevalence of smoking was substantially higher (26% versus 12% among Europeans, $p<0.0001$).

Overall prevalence of CKD among diabetes patients as identified through eGFR<60 ml/min/1.73m^2 was 19.5% using MDRD and 23.5% using CG. Prevalence of CKD, among European (18.8% using MDRD, 25.9% using CG) and Māori (20.4% using MDRD, 19.1% using CG) diabetes patients, in various subgroups of clinical characteristics is outlined in Tables 3 and 4 respectively. The prevalence of eGFR <30 ml/min/1.73m^2 among European and Māori was 2% and 3% respectively using MDRD, 3% and 3% respectively using CG.

There are significant differences in the agreement between MDRD and CG equations in identifying patients with eGFR <60 ml/min/1.73m^2 for Māori females, European females and European males (Table 5). While CG equation identifies more European of both genders, more Māori females are identified by MDRD.

Table 3. Prevalence of renal disease (eGFR<60 ml/min/1.73m^2) among European diabetes patients, eGFR estimated using MDRD and Cockroft-Gault (CG) equations

Variables		Female (n=244)		Male (n=331)	
Subgroup		MDRD	CG	MDRD	CG
		49 (20.1%)	64 (26.2%)	59 (17.8%)	85 (25.7%)
Age (years)	<60	3 (4.2%)	0 (0.0%)	5 (4.6%)	3 (2.8%)
	60+	46 (26.7%)	64 (37.2%)	54 (24.3%)	82 (36.9%)
	<10	20 (19.6%)	26 (25.5%)	23 (14.1%)	36 (22.1%)
Duration of diabetes (years)	10+	16 (25.0%)	20 (31.3%)	19 (26.8%)	28 (39.4%)
BMI*	Normal	16 (30.2%)	27 (50.9%)	15 (30.0%)	26 (52.0%)
	Obese	18 (16.8%)	11 (10.3%)	23 (15.5%)	24 (16.2%)
	Overweight	14 (18.2%)	25 (32.5%)	21 (16.8%)	34 (27.2%)
HbA_{1c} (%)	≤ 8	38 (22.2%)	50 (29.2%)	46 (19.7%)	66 (28.3%)
	> 8	11 (15.1%)	14 (19.2%)	13 (13.3%)	19 (19.4%)
High blood pressure†	No	12 (22.2%)	14 (25.9%)	22 (21.4%)	27 (26.2%)
	Yes	37 (19.5%)	50 (26.3%)	37 (16.3%)	58 (25.6%)
Microalbuminuria		10 (22.7%)	23 (25.6%)	24 (32.0%)	25 (20.2%)
Proteinuria		5 (35.7%)	16 (36.4%)	8 (44.4%)	31 (41.3%)

Data are number of people [n (%)] with eGFR<60 ml/min/1.73m^2 in each subgroup; * The BMI cut-off points as in the 2002/03 New Zealand Health Survey were used to classify overweight and obesity (25 and 30 respectively in European, 26 and 32 respectively in Maori); † Systolic BP ≥ 130 mmHg or Diastolic BP ≥ 80 mmHg

Table 4. Prevalence of renal disease (eGFR<60 ml/min/1.73m^2) among Māori diabetes patients, eGFR estimated using MDRD and Cockroft-Gault (CG) equations

Variables		Female (n=172)		Male (n=195)	
Subgroup		MDRD	CG	MDRD	CG
		45 (26.2%)	35 (20.3%)	30 (15.4%)	35 (17.9%)
Age (years)	<60	14 (15.2%)	8 (8.7%)	9 (9.5%)	6 (6.3%)
	60+	31 (38.8%)	27 (33.8%)	21 (21.0%)	29 (29.0%)
	<10	19 (23.8%)	13 (16.3%)	12 (12.5%)	15 (15.6%)
Duration of diabetes (years)	10+	14 (30.4%)	12 (26.1%)	8 (14.8%)	12 (22.2%)
BMI*	Normal	5 (26.3%)	6 (31.6%)	2 (16.7%)	5 (41.7%)
	Obese	24 (27.6%)	14 (16.1%)	16 (17.4%)	12 (13.0%)
	Overweight	14 (24.1%)	14 (24.1%)	10 (12.7%)	16 (20.3%)
HbA_{1c} (%)	≤ 8	27 (28.4%)	23 (24.2%)	19 (16.0%)	22 (18.5%)
	> 8	18 (23.4%)	12 (15.6%)	11 (14.5%)	13 (17.1%)

High blood pressure[†]	No	11 (24.4%)	8 (17.8%)	6 (11.8%)	9 (17.6%)
	Yes	34 (27.0%)	27 (21.4%)	24 (16.8%)	26 (18.2%)
Microalbuminuria		11 (25.0%)	4 (9.1%)	10 (14.1%)	4 (8.0%)
Proteinuria		11 (47.8%)	10 (22.7%)	15 (42.9%)	11 (15.5%)

Data are number of people [n (%)] with eGFR<60 ml/min/1.73m^2 in each subgroup; * The BMI cut-off points as in the 2002/03 New Zealand Health Survey were used to classify overweight and obesity (25 and 30 respectively in European, 26 and 32 respectively in Maori)' [†] Systolic BP ≥ 130 mmHg or Diastolic BP ≥ 80 mmHg.

Table 5. Agreement between MDRD and CG equations in identifying patients with eGFR<60 ml/min/1.73m^2

Patient group	n	MDRD	CG	Diff (%)	Kappa	P value
European Male	331	59 (17.8%)	85 (25.7%)	-7.9% (-11.5–-4.2)	0.65 (0.55–0.75)	<0.0000
European Female	244	49 (20.1%)	64 (26.2%)	-6.1% (-10.6– -1.7)	0.64 (0.53–0.76)	0.0107
Māori Male	195	30 (15.4%)	35 (17.9%)	-2.6% (-6.4–1.3)	0.72 (0.59–0.86)	0.3018
Māori female	172	45 (26.2%)	35 (20.3%)	+5.8% (1.1–10.6)	0.71 (0.58–0.83)	0.0309

Statin and ACE prescriptions among CKD patients were higher in the presence of microalbuminuria / proteinuria (71% vs. 57%, p=0.02 and 79% vs. 61%, p=0.001 respectively). CKD patients with normal ACR levels had better control of HbA_{1c} (80% with HbA_{1c}<8% vs. 66%, p=0.01) and blood pressure (34% with BP<130/80 vs. 20%, p=0.01) compared with CKD patients with microalbuminuria / proteinuria. (Table 6).

Table 6. Differences in management of diabetes patients with evidence of CKD compared with diabetes patients with normal renal function

Variables	Number (%)	% with HbA1c < 8%	% with BP < 130/80	% Prescribed Statin	% Prescribed ACE
eGFR <60 & Microalb/Proteinuria	128 (13.7%)	66%	20%	71%	79%
eGFR <60 & no Microalb/Proteinuria	125 (13.8%)	80%	34%	57%	61%
eGFR ≥60 & Microalb/Proteinuria	218 (23.3%)	50%	22%	68%	75%
Normal renal function	463 (49.6%)	70%	31%	62%	49%
Total	**934**	**66%**	**28%**	**64%**	**61%**

After adjustment for age, gender and BMI, Māori diabetes patients were significantly more likely to have clinically significant CKD compared with Europeans [odds ratio 1.8 (1.2, 2.8) using MDRD equation]. Similar results were yielded using CG equation.

Discussion

Māori and Pacific people with Type 2 diabetes have significantly higher rates of End Stage Renal Failure (ESRF), proteinuria and microalbuminuria than Europeans.[19] They are have higher rates of risk factors; obesity, smoking and poorer metabolic

control.[4] Differences in rates of proteinuria and microalbuminuria and degree of glomerular hyperfiltration are seen within 5 years of diagnosis.[20]

The increased risk of diabetic nephropathy among Māori and Pacific people is thought to be related to a family history of nephropathy rather than family history of diabetes.[21]

We investigated the prevalence and associations of CKD in this general practice based study of diabetes patients aged 18 and above, with high Māori representation. Overall prevalence of clinically significant CKD (eGFR<60 ml/min/1.73m^2) using eGFR was similar to that found in general practice populations in Australia.[6] (24.3% using CG), but lower than that in the UK[5] (31.3% using MDRD). The prevalence of proteinuria and microalbuminuria were similar to previous studies.

We have found a higher prevalence of clinically significant CKD (as indicated by an eGFR<60 ml/min/1.73m^2) among those with longer duration of diabetes (10+ years).Raised HbA_{1c} and blood pressure was associated with microalbuminuria/proteinuria and those patients were more likely to be prescribed statin or ACE.

Among people with clinically significant CKD, those without microalbuminuria/proteinuria were less likely to be prescribed statin or ACE than those with microalbuminuria / proteinuria, although they were also at high risk of cardiovascular disease. Interestingly, this group of patients tended to have good metabolic control, but only 34% were recorded as having blood pressure <130/80 mmHg. Routine monitoring of eGFR along with serum creatinine would identify this group of patients to their general practitioner as being in need of more intensive treatment of their blood pressure as well as glycaemic control.

Treatment with ACE inhibitors was much higher compared with figures from the UK where only one-third of diabetes patient with CKD stages 3–5 were ACE treated.[5] Only those patients with completed data were included in the analyses, which could possibly introduce a selection bias favouring regular attendees, raising the proportion with ACE / Statin prescriptions.

The MDRD equations were derived from patients with varying degrees of renal impairment employing a stepwise regression technique, where GFR was measured from the renal clearance of [^{125}I] iothalamate.[11] On the other hand, Cockcroft-Gault formula was constructed from hospitalised patients to predict creatinine clearance from the serum creatinine in the absence of urine collection.[15]

It has been shown that MDRD equation consistently underestimates GFR, whereas the CG equation consistently overestimates GFR in people without kidney disease.[22] In contrast, a New Zealand study with predominantly Europeans subjects found that the MDRD formula produced a statistically significant overestimation of GFR and the CG prediction equation gave a statistically significant underestimation of GFR,[23] but there was no significant difference in performance in estimating GFR between the two prediction equations.

A validation study in patients with ESRD showed that the MDRD equation is more accurate than the Cockcroft-Gault formula in predicting the group mean.[24] However, the predicted GFR using either formula was related to the basal GFR and percentage

body fat. MDRD is said to be preferable to the CG method in patients with diabetes.[25] However, our results indicate that while CG will identify more European diabetes patients at risk of CKD, it seem to miss some Māori women with diabetes.

The National Kidney Foundation in the US[26] and the National Service Framework for Renal Services in the UK[27] have recommended routine eGFR reporting. It has been endorsed by several other counties including New Zealand, Australia, Canada.[28,29] A recent review has shown the increasing use of eGFR in America, Europe, Asia and Australia, in population based studies which look at the prevalence of CKD.[30] Automatic reporting of eGFR, which constitutes *de facto* screening for chronic kidney disease is of concern,[31] given that and the validity of eGFR for this purpose has not been appropriately tested.[32,33]

Given the higher rates of renal complications among Māori, robust screening tools are needed to identify complications at an early stage. Automatic reporting of MDRD eGFR serves as a useful screening tool for kidney disease, although clinicians should recalculate it using the patient's actual body surface area for patients with extreme body size.[12]

The MDRD equation has a correction factor for black ethnicity. Given the high obesity rates, a similar correction factor may be required for Māori and other high risk ethnic minorities. Australasian Creatinine Consensus Working Group's recommends that laboratories continue to automatically report eGFR (MDRD) in Aboriginal and Torres Strait Islander peoples and other ethnic groups, pending publication of ethnic specific validation studies.[28]

More research is needed to develop a modified equation with a correction factor for Māori and similar high risk ethnicities. It appears that a generic approach will be unsuccessful in considering the validity of the eGFR in ethnic subpopulations. Each such subpopulation may need to be validated separately, and by gender.

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Ethnic disparities in causes of death among diabetes patients in the Waikato region of New Zealand

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Aim Assist health service planning by: (1) estimating the causes and disparities in mortality among people with diabetes in Waikato and (2) examining the differences in recording of diabetes.

Method A retrospective cohort study of diabetes patients registered with the Waikato Regional Diabetes Service. Deaths from 2003-2007 were identified among patients diagnosed with diabetes before 2003. Causes of death were obtained from the NZHIS. Mortality rates were compared with the general New Zealand population. Cox's proportional-hazards-model was used to estimate the all-cause and cause-specific mortality risk.

Results 921 deaths were observed among 9043 diabetes patients. Compared with Europeans, Māori had nearly double the age-adjusted mortality rates. SMRs for male-Europeans, female-Europeans, male-Māori and female-Māori aged 25+ were 1.16 (1.05-1.28), 1.10 (0.98–1.24), 2.49 (2.06–3.01), 3.12 (2.56–3.80) respectively. Of the 441 deaths with causes available, 268 (61%) had diabetes mentioned on the NZHIS-coding. Māori were more likely than Europeans to have diabetes reported on NZHIS-coding. They were more likely to die from cardiovascular disease, cancer and renal disease [Hazard-ratios 2.31 (1.6–3.3), 1.83 (1.1–3), and 11.74 (4.8–29) respectively].

Conclusion Māori diabetes patients experienced significantly higher risk of mortality compared with Europeans. Studies on diabetes related mortality using the national mortality database needs to take the increased recognition of diabetes on NZHIS coding for Māori into account.

Diabetes is associated with increased mortality rates, when compared to people without diabetes.[1–3] Some studies have suggested a reduction in excess mortality.[4] In New Zealand, Māori have been shown to have excess mortality associated with diabetes.[5] Health service planners use official mortality statistics as an indicator of health needs. NZHIS mortality records are routinely analysed, looking at deaths coded with diabetes as the primary cause of death. This approach has been shown to be missing out important information on deaths due to comorbidities among diabetes patients.

Routine mortality analysis is further limited by the under-coding of diabetes on death certificates.[6–8] Waikato DHB's Health Needs Analysis Report 2008 highlighted disparities in mortality for diabetes patients.[9] This could be biased if ethnic differences in the level of under coding differed, an issue not previously studied. To overcome coding biases, a comprehensive analysis of mortality in people with diabetes is best undertaken using a population based diabetes register.

The Waikato Regional Diabetes Service (WRDS) provides specialist diabetes services and performs retinal screening for people living within the Waikato DHB region. Patients are referred to the service by their general practitioner and the WRDS diabetes register is complied from the retinal screening register and those referred for other complications. The register is thought to be almost 90% complete for Waikato.[10]

The aims of this study are

(1) To estimate the causes of death and disparities in mortality among people with diabetes in Waikato by ethnicity and gender on the WRDS diabetes register.

(2) To examine the ethnic differences in the recording of diabetes on NZHIS coding.

Method

The WRDS database was established in 1997, to record secondary diabetes service utilisation. This is a retrospective cohort study of diabetes patients registered with the WRDS database before 2008. Patients diagnosed before 2003, and alive as of 1.Jan.2003, were identified and retrospectively followed for 5 years until death or end of 2007. The National Health Information Service (NZHIS) Mortality Collection classifies the underlying cause of death for all deaths registered in New Zealand, using the ICD-10-AM 2nd Edition and the WHO Rules and Guidelines for Mortality Coding.

Deaths registered in New Zealand from 1988 onwards are held in this national mortality database. Loss of follow-up due to within country migration was not an issue in this study, due to the availability of national mortality data. It was not possible to track migration out of New Zealand. The (NZHIS) Mortality Collection had causes of deaths available for deaths until 2005 at the time of the study in 2008. The unique National Health Index (NHI) number in New Zealand allows linkage between health information systems. Causes of death information for deaths from 2003–2005 was obtained from the NZHIS using NHI linkage. Patient status information (alive/deceased) is also available from WRDS database. In case of mismatch between national mortality data and WRDS data, deaths were verified by manually reviewing patient records and then by contacting the diabetes educators and general practitioners.

Causes of death were classified into cardiovascular disease (CVD), cancer, renal, cerebrovascular, gastrointestinal, respiratory, diabetes/complications and other. Two people coded the data independently and the two sets of codes were compared to minimise coding errors. The concordance between single ethnicity on WRDS database and prioritised ethnicity recorded on hospital patient management system was examined.

Crude mortality rates per 1000 person-years were calculated by ethnicity and gender. Segi world population, used in national mortality reports, was used to standardise mortality rates. The 95% confidence intervals for age-standardised mortality rates have been calculated using the Keyfitz method.[11] Mortality rates for Type 1 and Type 2 diabetes patients were age-adjusted using direct standardisation to the corresponding study population structure.

Standardised mortality ratios (SMRs) in relation to the national death rates were calculated using the 2004 national data from the Ministry of Health.[12] SMR is the ratio of observed number of deaths in the diabetic population to the expected number of deaths. Expected deaths were calculated by applying the age (5-year group) and gender specific mortality rates of the general population applied to the number of person-years of follow-up in each group. National ethnicity specific death rates were available for Māori population. SMRs for Māori diabetes patients in relation to national age and gender specific rates for Māori have been calculated.

Confidence intervals for SMRs were calculated using the Boice-Monson method.[13] Fisher's exact test was used to determine whether diabetes was more likely to be recorded on NZHIS coding for Māori compared with Europeans. Cox proportional hazards model was employed to identify the risk factors for all cause and cause-specific mortality. Data were analysed using SAS® version 9.

Results

9043 diabetes patients diagnosed with diabetes before 2003 were identified. Patients were of mean age 59±16 years, 69% Europeans, 21% Māori, 8% Other and 2% Unknown. The majority (7,501) had Type 2 diabetes and 1,391 had Type 1 diabetes. A small proportion of patients [151 (1.7%)] did not have type of diabetes recorded. Duration of diabetes at start of follow-up could be calculated for 8664 (95.8%) who had year of diagnosis of diabetes recorded.

8485 (94%) of patients had demographic information available on the hospital system due to secondary service contact. Of these 7575 (89%) had only a single ethnicity recorded, even though the hospital system can store up to three ethnicities. 568 (7%) had two ethnicities recorded, 7 (0.1%) had 3 ethnicities and 335 (4%) did not have any ethnicity recorded. While 91% of the 1915 people identifying themselves as Māori on the WRDS database had the same prioritised ethnicity on the hospital system, 129 (7.6%) were recorded as non-Māori. Similarly 120 people recorded as non-Māori on the WRDS database had prioritised Māori ethnicity on the hospital system. Ethnicity recorded on the WRDS database has been used for further analysis.

921 deaths were observed during the 5-year follow-up period with 46261 person-years of follow-up (Table 1). Compared with European diabetes patients, Māori had nearly double the age-adjusted mortality rates (Table 2). SMRs in relation to national general population rates for male-Europeans, female-Europeans, male-Māori and female-Māori aged 25+ were 1.16(1.05–1.28), 1.10(0.98–1.24), 2.49(2.06–3.01), 3.12(2.56–3.80) respectively. Age, gender and ethnicity specific SMRs have been calculated diabetes patients in general as well as for people with Type 2 diabetes (Table 3). Age-specific SMRs decreased with age among all subgroups of ethnicity and gender.

Of the 921 observed deaths, 441 deaths until end of 2005 (26581 person-years of follow-up) had cause of death information available. 268/441 (61%) had diabetes mentioned on the death certificate. Among the 441 deceased patients, 98% of patients recorded as Māori on the WRDS database had a matching ethnicity on NZHIS mortality database, where as 96% had the same prioritised ethnicity on the hospital system. Māori are more likely than Europeans to have diabetes reported on NZHIS coding (p value 0.0098), but cause specific differences were not statistically significant (p value 0.0760 and 0.6414 for cardio vascular disease and cancer respectively).

Due to the small number of observed deaths among Pacific Islands people and Asians (18 and 12 respectively), they are not analysed as separate ethnicity categories but are included in the total.

Among both Europeans and Māori, nearly half the deaths were due to cardiovascular disease and quarter of deaths due to cancer (Table 4). Among those dying due to cardiovascular disease, Māori were more likely to have renal comorbidity than Europeans. [13/46 (28%) vs 6 /141 (4%), Chi-squared p value<0.0001].

Table 1. Patient characteristics at start of follow-up by ethnicity

Variables	European	Māori	Total
N	6236	1915	9043
Age at start of follow-up (mean±SD)	61±16	54±13	59±16
Male	3246 (52%)	924 (48%)	4649 (51%)
Type of diabetes*			
Type 2	4948 (80%)	1749 (94%)	7501 (84%)
Type 1	1202 (20%)	115 (6%)	1391 (16%)
Age at diagnosis of diabetes, years* (mean±SD)			
Type 2 (n=7276)	57.7±12.6	46.5±12.4	54.3±13.4
Type 1 (n=1361)	24.8±16.9	27.1±15.1	25.3±16.8
Duration of diabetes at start of follow-up, years			
Type 2	7.8±6.9	8.8±7.9	7.9±7.1
Type 1	16.9±11.7	13.0±9.6	16.4±11.5

* Patients with missing data have been excluded. Data are (mean±SD) or n (%).

Table 2. Crude and age-adjusted death rates for diabetes patients by gender and ethnicity

Variables	European		Māori		Total	
	Male	Female	Male	Female	Male	Female
All diabetes patients (person-years)	3246 (15,409)	2990 (14,376)	924 (4397)	991 (4745)	4649 (22,135)	4394 (21,126)
Observed deaths	380	276	108	98	520	401
Crude mortality per 1000 person years	24.7	19.2	24.6	20.7	23.5	18.9
Age-standardised[‡] to Segi world population, rate/100,000 person-years (95% CI)	551 (496–606)	491 (433–549)	1,012 (821–1203)	808 (648–968)	632 (578–686)	569 (513–625)
Type 2 diabetes patients (person-years)	2608 (7612)	2340 (6910)	843 (2475)	906 (2665)	3880 (11,359)	3621 (10,681)
Observed deaths (Male/ Female)	340	230	94	86	463	340
Age-adjusted* mortality rate/1000 person-years	24.8	16.6	33.5	34.6	27.0	27.0
Age-standardised[‡] to Segi world population, rate/100,000 person-years (95% CI)	458 (409–506)	353 (308–399)	960 (766–1154)	724 (571–877)	570 (518–622)	459 (411–508)
Type 1 diabetes patients (person-years)	596 (1761)	606 (1682)	55 (160)	60 (168)	694 (2049)	697 (2041)
Observed deaths (Male/ Female)	41	29	12	10	41	52
Age-adjusted[†] mortality rate/1000 person-years	10.9	14.3	65.7	66.0	12.2	15.6

*Direct standardisation to Type 2 diabetes population in the study.
[†] Direct standardisation to Type 1 diabetes population in the study.
[‡] Direct standardisation using Segi world population.

Table 3. Age-specific all cause standardised mortality ratios (SMRs) after 5 years of follow-up for European and Māori diabetes patients by gender

Variables		All diabetes patients		Type 2 diabetes patients	
	Age group	Deaths	SMR (95% CI)	Deaths	SMR (95% CI)
European Female	40–49	10	4.32 (2.33–8.03)*	4	2.64 (0.99–7.04)
	50–59	17	1.76 (1.09–2.83)*	16	2.02 (1.24–3.30)*
	60–69	50	1.44 (1.09–1.90)*	41	1.30 (0.96–1.77)
	70–79	94	1.07 (0.88–1.31)	78	0.97 (0.77–1.21)
	80+	102	0.90 (0.74–1.09)	91	0.87 (0.71–1.07)
European Male	40–49	6	1.64 (0.74–3.65)	2	0.82 (0.21–3.28)
	50–59	25	1.55 (1.05–2.29)*	18	1.29 (0.81–2.04)
	60–69	72	1.21 (0.96–1.52)	66	1.20 (0.95–1.53)
	70–79	189	1.29 (1.12–1.49)*	176	1.29 (1.11–1.49)*
	80+	86	0.85 (0.69–1.05)	78	0.84 (0.67–1.05)
Māori Female	40–49	16	9.32 (5.71–15.2)*	14	8.48 (5.02–14.3)*
	50–59	16	3.05 (1.87–4.97)*	14	2.82 (1.67–4.75)*
	60–69	36	3.64 (2.63–5.05)*	31	3.29 (2.31–4.67)*
	70–79	23	1.94 (1.29–2.91)*	21	1.86 (1.21–2.85)*
	80+	5	2.20 (0.91–5.27)	5	2.20 (0.91–5.27)
Māori Male	40–49	12	5.50 (3.12–9.69)*	10	4.96 (2.67–9.21)*
	50–59	29	4.10 (2.85–5.90)*	26	3.92 (2.67–5.75)*
	60–69	41	2.79 (2.05–3.79)*	33	2.39 (1.70–3.36)*
	70–79	21	1.51 (0.98–2.31)	20	1.47 (0.95–2.28)
	80+	4	0.82 (0.31–2.18)	4	0.82 (0.31–2.18)
Māori Female*	40–49	16	3.99 (2.44–6.51)*	14	3.63 (2.15–6.12)*
	50–59	16	1.27 (0.78–2.08)	14	1.18 (0.70–1.99)
	60–69	36	1.55 (1.12–2.15)*	31	1.40 (0.99–1.99)
	70–79	23	0.96 (0.64–1.44)	21	0.92 (0.60–1.41)
	80+	5	1.74 (0.72–4.18)	5	1.74 (0.72–4.18)
Māori Male*	40–49	12	2.30 (1.31–4.05)*	10	2.07 (1.12–3.86)*
	50–59	29	1.60 (1.11–2.31)*	26	1.53 (1.04–2.25)*
	60–69	41	1.32 (0.97–1.79)	33	1.13 (0.80–1.59)
	70–79	21	0.87 (0.57–1.34)	20	0.85 (0.55–1.31)
	80+	4	0.62 (0.23–1.66)	4	0.62 (0.23–1.66)

* In relation to national Māori population rates. SMR is the ratio of observed number of deaths in the diabetic population to the expected number of deaths. Expected deaths were calculated by applying the age (5-year group) and gender specific mortality rates of the general population applied to the number of person-years of follow-up in each group. Numbers for Type 1 diabetes were too small by age group.

Table 4. Primary causes of death and the extent of recognition of diabetes on NZHIS coding

Variables	Causes of deaths			Mention of diabetes		
	European	Māori	Total	European	Māori	Total
Cancer	71 (22.7%)	25 (24.5%)	103 (23.4%)	48%	40%	44%
Renal	9 (2.9%)	14 (13.7%)	25 (5.6%)	100%	100%	100%
Cardiovascular disease	141 (45.1%)	46 (45.1%)	197 (44.7%)	61%	76%	64%
Diabetes/complications	25 (8.0%)	5 (4.9%)	31 (7.0%)	96%	100%	97%
Cerebrovascular	21 (6.7%)	1 (1.0%)	23 (5.2%)	43%	0%	39%
Gastrointestinal	9 (2.9%)	3 (2.9%)	14 (3.1%)	22%	67%	29%
Respiratory	17 (5.4%)	5 (4.9%)	23 (5.2%)	59%	100%	70%
Other	20 (6.4%)	3 (2.9%)	25 (5.7%)	35%	100%	48%
All Cause	313	102	441	58%	73%	61%

Table 5. Cox's proportional hazards ratios (95% CI) for all cause, cardiovascular, cancer related and renal mortality among European and Māori diabetes patients

	All cause mortality by diabetes type		Cause specific mortality†		
	Type 2	Type 1	CVD	Cancer	Renal
No. of deaths	750	92	185	96	23
Age (years)	1.08 (1.07–1.09)*	1.08 (1.06–1.09)*	1.09 (1.07–1.1)*	1.06 (1.04–1.1)*	1.06 (1.03–1.1)*
Māori (vs European)	1.92 (1.61–2.30)*	5.43 (3.31–8.92)*	2.31 (1.6–3.3)*	1.83 (1.1–3)*	11.74 (4.8–29)*
Male (vs Female)	1.44 (1.25–1.68)*	0.83 (0.56–1.25)	1.99 (1.47–2.7)*	1.25 (0.8–1.9)	0.93 (0.4–2.1)
Type 1 (vs Type2)	–	–	2.96 (1.94–4.5)*	0.91 (0.3–2.2)	13.16 (5.3–33)*

European and Māori patients only. Variables are mutually adjusted; * Significant at 5% level;
† Only those events with cause of death information available.

Compared with European diabetes patients, Māori diabetes patients are more likely to die from cardiovascular disease, cancer and renal disease (Table 5). Māori and Type 1 diabetes patients have significantly higher risk of death due to renal disease.

Discussion

Results of the present study indicate that Māori continue to have nearly double the age adjusted mortality rates than Europeans.

Age-specific SMRs decreased with age among all subgroups of ethnicity and gender. Convergence of SMRs with age is expected with the mortality rates in the general population rising exponentially with age. SMRs were higher among females (both European and Māori) compared with males. Gender differences in SMRs were higher in the younger age groups (forties and fifties), especially among Type 2 diabetes patients, but the differences diminished with age. Similar results of excess mortality among women in the younger age groups were observed in the Swedish linkage study, due to significant interaction between index age and gender.[14]

The observed all cause SMRs, especially in the older age groups, were lower than that found in previous New Zealand studies in the 1990s looking at mortality among people with diabetes.[5 6 15] This could be due to a range of factors including increased screening resulting in earlier detection of diabetes before the onset of complications,[16] the introduction of evidence based guidelines in 2003, improvements in the management of risk factors for diabetes complications (example: blood pressure and lipids).[17,18]

Mortality rates have been estimated based on a cohort of diabetes patients registered with the Waikato Regional Diabetes Service. WRDS register is estimated to cover almost 90% of the diabetes patients in the Waikato[10], with the exemption of newly diagnosed diabetes patients who are yet to attend their first retinal screening, those with established eye disease and those who are too frail to attend retinal screening.[19]

Observed mortality rates may be underestimated since deaths among older diabetes patients not needing retinal screening would not be captured. As opposed to the prioritised ethnicity used commonly in New Zealand, a single ethnicity is stored in the WRDS database. But results of the hospital system audit indicate that multiple ethnicities are not commonly recorded and the use of prioritised ethnicity is unlikely to make a huge difference.

Reductions in all-cause mortality among women and men with diabetes mellitus have occurred over time in the U.S,[4 20] but mortality rates among individuals with diabetes mellitus remain 2-fold higher compared with individuals without diabetes. Although overall mortality rates in the New Zealand general population decreased over time,[21] such trends are not available separately for people with and without diabetes.

National estimates of mortality burden due to diabetes (compared with people without diabetes) in New Zealand, derived from multi-state life tables,[22] are constrained by data uncertainties in the estimates of prevalence of diabetes and in the estimates of relative risk of all-cause mortality conditional on diabetes. Previous studies in New Zealand have looked at mortality among diabetes patients in relation to that in the national general population. Māori Type 2 diabetes patients in aged 40–59 in South

Auckland[6] experienced 7 times excess mortality, in relation to the national total population rates.

A record linkage study using hospital discharges, comparing the mortality patterns of patients with diabetes to the general population of the same ethnic group, found that Māori with diabetes have nearly four times excess mortality, while Pacific have slightly over 2 times and non-Māori/non-Pacific have nearly 3 times excess mortality in the 25+ age-group.[5] Studies based on patients with diabetes identified through hospital records report higher SMRs,[14] probably due to of the selective inclusion of more patients in more advanced stages of diabetes and its complications.

With high prevalence of diabetes among middle aged Māori in the general population,[23] SMRs may not be indicative of the true burden due to diabetes. Mortality attributable to diabetes would be better estimated using studies involving people with and without diabetes. Such studies may be feasible using general practice information systems, as in the U.K.[2,3] The choice of population standard affects the magnitude of mortality rates and standardised mortality ratios[24], as evident from the Māori rates standardised using two different populations.

The results suggest that the under-coding of diabetes on death certificates has not improved and continues to be a major limitation for routine mortality analysis solely based on these codes. Māori are more likely to have diabetes reported on death certificates due to higher proportion having renal comorbidities, for which diabetes coding is higher. This would introduce significant bias to mortality analysis using diabetes coding on national mortality data.

Current findings are in agreement with the higher risk of death from nephropathy for Māori with Type 2 diabetes compared with Europeans with Type 2 diabetes observed in South Auckland (adjusted hazard-ratio of 15).[6] Present results indicate that Māori diabetes patients experienced significantly higher mortality due to cardiovascular disease and cancer as well. Excess mortality risk among Type 1 patients may be partly due to the longer duration of diabetes.

Māori in general have high prevalence of cardiovascular disease independent of social deprivation.[25] They are also at increased risk of first cardiovascular event in the presence of Type 2 diabetes.[26] Māori with diabetes experience significant excess mortality compared to the Māori general population.[5 6] Disparities in cancer survival are reported to be partly attributed to late presentation among Māori[27], as well as differences in exposure to risk factors and access to screening and treatment. Ethnic mortality gradients are influenced by socioeconomic factors[28 29] and smoking.[30] Socio economic deprivation, which may be a proximal cause of excess diabetes mortality among Māori, was not available in this study. Māori with diabetes face a range of barriers to self care.[31]

In conclusion, Māori diabetes patients experience significantly higher mortality than Europeans. The data yet again demonstrates the shortcomings of diabetes coding on death certificates. Studies on diabetes related mortality using national mortality database needs to take the increased recognition of diabetes on NZHIS coding for Māori into account. Mortality among diabetes patients in New Zealand would need to be compared with that among people without known diabetes, to estimate the true burden due to diabetes.

Competing interests: None known.

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Exploring physical and psychological wellbeing among adults with Type 2 diabetes in New Zealand: identifying a need to improve the experiences of Pacific peoples

Charlotte A M Paddison

Abstract

Aims This study explores levels of physical and psychological wellbeing among adults with Type 2 diabetes, and aims to identify the clinical, demographic, and psychological factors that are associated with differences in wellbeing.

Methods Research participants (N=615) were randomly selected from a database of primary care records for people with diabetes (N=4857) in New Zealand. Self-report data were collected through a mailed questionnaire and clinical data from electronic records were obtained with participants' permission.

Result Mean HbA_{1c} was 7.5% (*SD*=1.5) although this varied significantly across ethnic groups (*p*=.001) with metabolic control highest among New Zealand Europeans and lowest among Pacific peoples. Results showed that Pacific groups also experienced the highest levels of distress about diabetes, and concern about prescribed medication.

Conclusions Adults with Type 2 diabetes who are young, overweight, have concerns about prescribed medications, and those of Pacific ethnicity, are most likely to experience adverse health outcomes including poor metabolic control and diabetes-related distress. Among Pacific peoples in particular there is a need to address concerns about medication and emotional distress about diabetes, while maintaining a focus on improving metabolic control.

The aims of diabetes treatment are to promote good glycaemic control, and quality of life.[1,2] Research shows that while an HbA_{1c} <7% is recommended,[3,4] many, perhaps even the majority[5] of people with Type 2 diabetes do not achieve this goal. However there is considerable variation between individuals. Previous research has shown that differences in health outcomes among people with Type 2 diabetes are related to clinical and demographic characteristics including ethnicity.[6-8]

Research shows that many people experience diabetes-related distress.[9-12] However there have been very few studies to date exploring individual variation in diabetes-related psychological outcomes. As a consequence we know little about how diabetes distress might vary between individuals. For example, although we know that differences between ethnic groups have been shown for metabolic control, we do not know if the psychological burden of diabetes shows a similar pattern of association with ethnicity. Health services providing psychological support for people with long-term conditions including diabetes are now funded in some regions in New Zealand and so it is very timely that research now identifies the predictors of poor illness-related *psychological* outcomes so that we may learn who is most at risk, and how best to intervene, in order to promote wellbeing among people with diabetes.

In addition to clinical and demographic characteristics, psychological variables are also related to health outcomes. For example the widely-used Common Sense Model of illness self-regulation[13] suggests that psychological variables including beliefs about treatment can play a salient role in determining coping behaviour and subsequent health outcomes.[14] Further, because treatment beliefs are amenable to change in a way that age and ethnicity for example are not, these psychological variables are of particular interest to researchers who may want to identify targets for interventions that aim to improve health outcomes.

Helping people with diabetes to stay well is clearly an important, and challenging, goal. The evidence suggests many people with diabetes experience suboptimal physical and psychological health outcomes but few studies have examined how outcomes – particularly illness-related psychological outcomes—vary across individuals. The aims of this study are to:

1. Explore levels of physical and psychological wellbeing among adults with Type 2 diabetes
2. Identify clinical, demographic, and psychological factors associated with differences in wellbeing
3. Test for between-group differences in physical and psychological wellbeing and treatment beliefs, across ethnicity.

Research design and methods

The research sample (N=1015) was randomly selected from a database of primary care records for people with diabetes (N=4857) held in Wellington, New Zealand. This database showed an under representation of Māori and Pacific groups compared with population-based diabetes estimates.[15] Therefore, Māori and Pacific groups were purposefully over-sampled in the research design so that the ethnicity of participants in this study might closely reflect the demographic profile reported by the Ministry of Health[15] for people with Type 2 diabetes in New Zealand. Inclusion criteria for this study were: diagnosis of Type 2 diabetes by a physician in accordance with national guidelines[4]; and aged ≥18.

Psychological data were collected using a mailed questionnaire survey, with a response rate of 62% providing 629 completed questionnaires. Potential participants were mailed a letter of introduction informing them of the study and inviting their participation; two weeks later they received an information sheet, a consent form, and a copy of the research questionnaire. For consenting participants' (*N*=615, 98%) relevant physician-assessed clinical data including glycosylated haemoglobin (HbA_{1c}) were obtained from electronic medical records and released to the researcher in non-identifiable form. Clinical data and questionnaire data were linked by unique identifier codes; patient anonymity was protected at all times during the research. Ethical approval for this study was obtained from the Massey University Human Ethics Committee, protocol 02/140.

Participants—Table 1 provides a summary of participants' demographic and clinical characteristics. Participants' mean age was 63 years (*SD*=11.6), and forty-seven percent were female. The ethnic composition of participants (57% New Zealand European, 30% Māori, 6.4% Pacific Island) matched closely with proportional

representation of the three main ethnic groups that comprise the New Zealand diabetes population as reported by the New Zealand Ministry of Health.[15] On average, length of diagnosis with diabetes was 8.1 years (*SD*=5.8); mean HbA_{1c} was 7.5% (*SD*=1.5), with a range from 4.5 to 13.5. Nine percent of participants were currently using insulin, almost half (49%) had a treatment regimen that included ACE inhibitors, and 25% had been prescribed statins. Mean body mass index (BMI) was 31 (*SD*=7.1), and 15% were current smokers.

Table 1. Demographic and clinical characteristics of participants (N=615)

Age in years (mean, sd)	63 (11.6)
Gender	
Female (number, %)	292 (47.5)
Male (number, %)	323 (52.5)
Ethnicity	
NZ European (number, %)	352 (57.2)
Māori (number, %)	188 (30.5)
Pacific† (number, %)	39 (6.5)
Other‡ (number, %)	36 (5.8)
Length of diagnosis in years (mean, sd)	8.1 (5.8)
Body Mass Index (mean, sd)	31.9 (7.1)
Haemoglobin A_{1c} (number, %)	
<7%	259 (42.1)
7–8%	196 (31.8)
8.1–9%	80 (13.0)
>9%	80 (13.0)
Current diabetes treatments (number, %)	
Diet and exercise alone	186 (30.2)
Oral treatment: ACE inhibitors	302 (49.1)
Oral treatment: Antihypertensives	221 (36.0)
Oral treatment: Statins	151 (25.0)
Insulin	56 (9.1)

† E.g., Tongan, Samoan, Cook Islands, Fijian, Niuean
‡ E.g., Dutch, German, Indian, Chinese

Measures—Length of diabetes diagnosis, current treatments, BMI, and most recently recorded HbA_{1c} were extracted from electronic medical records. General self-reported mental health and physical health were assessed using the SF-12 MCS subscale, and SF-12 PCS subscale, respectively.[16] Diabetes-related psychological distress was measured using the Problem Areas In Diabetes (PAID) scale.[17] The Beliefs about Medications Questionnaire (BMQ)[18] was used to assess concern about diabetes medicines (six items), and perceived medication necessity (five items), with responses provided on a 5-point Likert scale: (1) strongly disagree; (2) disagree; (3) uncertain;

(4) agree; (5) strongly agree. The average score across items in each BMQ subscale were calculated providing two summary scores (medication concern; medication necessity) with a range from 1 to 5.

Statistical analyses—Mean scores were first calculated for the four primary outcome variables of interest in this study: HbA_{1c}, diabetes-related distress (PAID), self-reported general physical health (SF-12 PCS), and self-reported general mental health (SF-12 MCS), and their relationships with clinical, demographic, and psychological variables were assessed using Pearson's correlation coefficients. Second, ANCOVAs were used to test for between-groups differences in outcome variables across ethnicity. In all ANCOVA tests age, length of diagnosis, BMI, and treatment (insulin use) were entered as co-variates in the model to control for their effects. SPSS/PC version 15.0 was used for all statistical analyses with alpha set at .05.

Results

Levels of physical and psychological wellbeing—Mean HbA_{1c} was 7.5 (SD=1.5) and, as shown in Table 1, 257 participants (42.1%) met the recommended goal for Type 2 diabetes in New Zealand[4] of an HbA_{1c} less than 7%. The average score on the Problem Areas In Diabetes (PAID) scale was 19.6 (*SD*=19.1) and this is in line with average scores reported in previous research.[12] Mean scores on the SF-12 PCS (physical health) and SF-12 MCS (mental health) subscales were 44.0 (*SD*=10.0), and 50.5 (*SD*=9.3), respectively.

Relationships between clinical and demographic characteristics and health outcomes—Table 2 shows the relationships between demographic and clinical characteristics and the four outcome variables of primary interest in this study (HbA_{1c}, diabetes-related psychological distress, self-reported general physical health, and self-reported general mental health). Greater age was associated with lower self-reported physical health, and higher self-reported mental health, as is consistent with previous research.[16] Higher age was also related to lower HbA_{1c}, and fewer worries about diabetes. Longer length of diagnosis was associated with higher HbA_{1c}, and lower self-reported general physical wellbeing; length of diagnosis was unrelated to measures of general (SF-12 MCS) or diabetes-specific (PAID) emotional wellbeing. There was a positive relationship between prescribed insulin treatment, and HbA_{1c}.

Relationships between treatment beliefs and health outcomes—Greater concern about diabetes medication was associated with higher HbA_{1c}, higher scores on the PAID, and lower scores on the SF-12 MCS subscale as shown in Table 2. Perceived need for diabetes medication did not show significant relationships with any of the four outcome measures.

Table 2. Pearson's correlations between health outcomes and clinical, demographic, and psychological variables (N=615)

Variables	Physical and psychological wellbeing			
	HbA_{1c}	PAID	SF-12 PCS	SF-12 MCS
Demographic and clinical variables				
Age	-.25 (*p*=.001)	-.36 (*p*=.001)	-.26 (*p*=.001)	.22 (*p*=.001)
Gender (% female, number)	-.01 (*p*=.587)	-.04 (*p*=.332)	-.12 (*p*=.007)	.02 (*p*=.680)
BMI	.15 (*p*=.001)	.17 (*p*=.001)	-.12 (*p*=.005)	-.13 (*p*=.004)
Length of diabetes diagnosis (years)	.19 (*p*=.001)	-.07 (*p*=.127)	-.12 (*p*=.007)	-.07 (*p*=.133)
Treatment (using insulin)	.13 (*p*=.001)	.07 (*p*=.090)	.01 (*p*=.988)	-.07 (*p*=.110)
Treatment beliefs				
Medication necessity	.06 (*p*=.211)	.02 (*p*=.686)	-.06 (*p*=.232)	-.08 (*p*=.108)
Medication concern	.25 (*p*=.001)	.57 (*p*=.001)	.02 (*p*=.739)	-.34 (*p*=.001)

Differences in diabetes-related outcomes by ethnicity—Figure 1 displays means for HbA_{1c} across four ethnic group categories: New Zealand European; Māori; Pacific; and Other. Figure 2 shows average scores on the Problem Areas in Diabetes scale across these same four ethnic groups. On average HbA_{1c} was lowest (showing better metabolic control) among New Zealand European participants, and highest among Pacific participants. Pacific participants also showed the highest scores on the PAID, as displayed in Figure 2.

Figure 1. Glycaemic control by ethnicity showing means and 95% confidence intervals

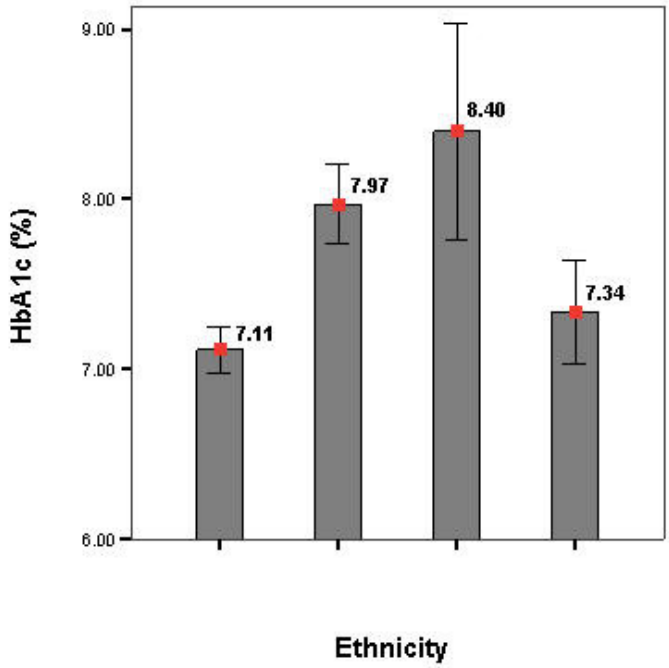
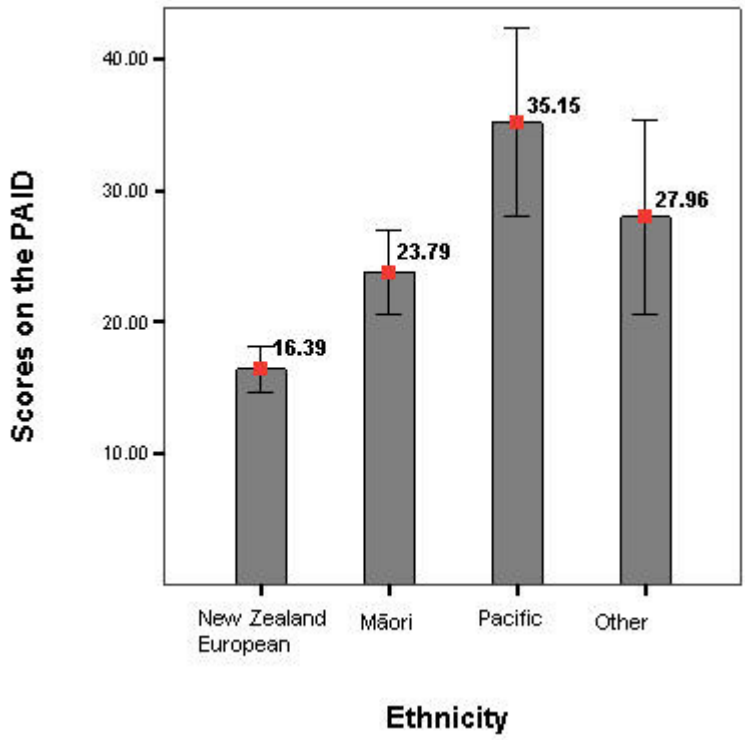


Figure 2. Scores on the Problem Areas in Diabetes (PAID) scale by ethnicity showing means and 95% confidence intervals



Testing between-group differences—Table 3 displays means (SE), and p-values from ANCOVAs testing for between-group differences in physical wellbeing, psychological wellbeing, and medication beliefs across four ethnic groups. Analyses controlling for the effects of age, length of diagnosis, BMI, and insulin use showed that there were significant between-group differences for: HbA_{1c} (p=.001); scores on the PAID (p=.001); SF-12 MCS (p=.030); and the medication concern subscale of the BMQ (p=.001). Results show that, on average, Pacific peoples showed the highest HbA_{1c}, and reported the greatest worry about diabetes; and they also showed the highest levels of concern about prescribed medication. An additional ANCOVA controlling for scores on the SF-12 MCS was run for the PAID and this did not change the reported results. This suggests that the higher levels of diabetes-related distress found among Pacific people were not accounted for by any cultural differences in self-reported general mental health.

Table 3. Means, standard errors, and p-values showing between-groups differences† in physical wellbeing, psychological wellbeing, and treatment beliefs across four ethnic groups

	Ethnicity				
	New Zealand European (*n* = 352)	**Māori** (*n* = 188)	**Pacific** (*n* = 39)	**Other** (*n* = 36)	
	M (*SE*)	*M* (*SE*	*M* (*SE*)	*M* (*SE*)	*p*-value
Physical wellbeing					
Haemoglobin A_{1c}	7.11 (.08)	7.97 (.11)	8.40 (.22)	7.34 (.24)	0.001
SF-12 PCS	43.24 (.56)	45.14 (.78)	44.89 (1.61)	44.23 (1.69)	0.246
Psychological wellbeing					
PAID	16.39 (1.03)	23.79 (1.45)	35.15 (2.99)	27.96 (3.14)	0.001
SF-12 MCS	51.36 (.52)	48.94 (.73)	51.37 (1.50)	48.70 (1.6)	0.030
Treatment beliefs					
Medication concern	2.23 (.05)	2.33 (.07)	3.04 (.13)	2.81 (.14)	0.001
Medication necessity	3.94 (.05)	3.97 (.06)	3.82 (.13)	3.72 (.14)	0.357

† Adjusted for age, length of diagnosis, BMI, and insulin use

Comparison of respondents and non-respondents—T-tests and χ^2 tests examining potential differences between respondents and non-respondents showed no significant difference in gender, or length of diabetes diagnosis, but there was a difference between respondents and non-respondents for HbA_{1c}, $F(1,1015) = 13.67$, $p < .001$, and ethnicity, χ^2 (3, $N = 1275$) = 59.83, $p<.001$. On average, non-respondents had a higher HbA_{1c} ($M = 7.79$, $SD = 1.67$) than respondents ($M = 7.45$, $SD = 1.52$), and those who returned a completed questionnaire were more likely to be of New Zealand European ethnicity.

Discussion

On average, people with Type 2 diabetes in New Zealand appear to show similar levels of self-reported general physical health to peers with diabetes in other countries,[16] and similar levels of general emotional functioning to same-age peers without diabetes.[16,19] In this study the majority (58%) of participants did not meet the recommended goal[4] of an $HbA_{1c} < 7\%$. These results are congruent with international research[5] and show that many people with Type 2 diabetes experience difficulty achieving good metabolic control. However physical and psychological health outcomes do vary across individuals and this variation was found to be associated with clinical, demographic, and psychological factors.

Clinical and demographic factors associated with differences in wellbeing among adults with Type 2 diabetes—Higher BMI, younger age, greater length of diagnosis, and insulin use were associated with higher HbA_{1c}, and this is in keeping with observations that: being overweight exacerbates difficulties with blood glucose regulation; insulin resistance increases over time among people with Type 2 diabetes; and poor metabolic control leads over time to more intensive treatment, including the prescription of insulin.

Results are consistent with research showing that age has a protective effect on general psychological wellbeing,[16] and suggest that this effect may also extend to diabetes-related worries.[10] Older adults may have had more exposure to health problems (as the risk of common chronic illnesses such as coronary heart disease increases with age), and as a result could have developed greater skills for coping successfully with the emotional impact of illness, in comparison with younger adults.[11] Interestingly—and in contrast with some previous research[10]—insulin use was not associated with higher diabetes-related distress.

Relationships between treatment beliefs and physical and psychological wellbeing—Our findings show that people who are more concerned about using prescribed diabetes medication (e.g. worried about harmful side-effects, or possible dependency) are more likely to experience poor metabolic control and higher diabetes-related distress than those who have less concerns about pharmacological treatments. It is possible that this relationship between medication concern and health outcomes is mediated by medication use: this is in line with research showing that greater concern about prescribed medication is associated with lower adherence to the prescribed treatment regimen in other chronic conditions.[20] Perceived need for diabetes medication was not related to differences in any of the four primary outcome variables: this contrasts with results reported by Barnes et al.[7] but is consistent with

the absence of a relationship between medication necessity and self-reported adherence among renal haemodialysis patients.[20]

Differences in diabetes-related health outcomes across ethnicity—In comparison with New Zealand European participants in our study, on average, Māori and Pacific peoples experienced lower glycaemic control. Our findings are in line with research from New Zealand[7,8] and internationally[6,21,22] showing that physical health among people with diabetes varies by ethnicity and that indigenous peoples and recent immigrants are often worst off. There are many possible reasons for this and they include: delays in diagnosis[23] and treatment;[8,24] differences in lifestyle behaviours,[24] rates of obesity,[8,25] and diabetes education;[26] and genetics.[27]

Findings extend our understandings of the way diabetes-related health outcomes vary by showing that *psychological* outcomes such as diabetes distress show a similar pattern of differences across ethnicity to that previously reported for physical health. In our study the observed pattern was very consistent across both diabetes-related physical and psychological wellbeing, with highest metabolic control and lowest diabetes-related distress among New Zealand Europeans, and the reverse (lowest metabolic control and highest diabetes-related distress) reported among Pacific peoples as Figure 1 and Figure 2 illustrate. Our findings are interesting partly because previous international research has not shown a relationship between ethnicity and diabetes-related distress.[10]

While it is useful to better understand how health outcomes—including illness-related psychological outcomes—vary by ethnicity, we emphasise the need to consider how this information can be used to improve population health outcomes. For example: to identify who is most at risk for adverse outcomes; consider how best to intervene; and to provide added impetus for the development of interventions that are acceptable, effective, and culturally appropriate for the New Zealand diabetes population.

Identifying who is most at risk for adverse outcomes—Among adults with Type 2 diabetes in New Zealand overweight individuals; those on insulin; younger adults; and those who have had diabetes for a longer time, are most at risk for adverse physical health outcomes including poor glycaemic control. Younger adults, and those who are overweight, also appear to be most at risk for experiencing diabetes-related psychological distress. Two further characteristics identified as important in showing who is most at risk for adverse health outcomes are: high concerns about diabetes medication, and Pacific ethnicity.

Clinical implications and opportunities for intervention—Patients who have high concerns about diabetes medications could be identified using a brief screening tool (for example, the 6-item medication concern subscale from the BMQ) and invited to talk in detail about their concerns. This gives clinicians an opportunity to clarify any misperceptions (e.g. regarding potential harms), provide reassurance, and explain the benefits of the prescribed treatments. As previous research has shown that lower medication concern is associated with higher self-reported medication adherence[20,28] any success in reducing concern about diabetes medication may benefit psychological wellbeing but could also help to increase medication adherence and therefore metabolic control.

Younger adults and those who are overweight appear to be at greater risk of both diabetes-related psychological distress and poor metabolic control and this suggests another possible target group for clinical intervention. For example, practice records could be used to identify young overweight patients with diabetes and extra support could be provided to help these patients manage the emotional consequences of diabetes and improve self-care behaviour, particularly as Type 2 diabetes in young people is associated with the early onset of significant complications such as nephropathy.[29] Interventions that are engaging to younger adults—for example those that are text or internet-based—may be most useful.

One important clinical implication of our findings is the need to better assist Māori and Pacific people in managing diabetes. Primary prevention programmes that seek to prevent the unnecessary excess burden of morbidity through *early* intervention, include wider family networks, and seek to improve both metabolic control and diabetes-related psychological wellbeing (particularly given the importance placed on physical *and* psychological wellbeing—te taha tinana, and te taha hinengaro—in Māori health frameworks[30]), are strongly encouraged. Evidence suggests that Pacific people with diabetes may be less intensively treated than New Zealand Europeans, for example they are less likely to be prescribed statins,[8] and this may also contribute to poor health outcomes and should be addressed in primary care practice.

We are not aware of any other New Zealand research assessing cultural differences in diabetes-related psychological wellbeing and this is an important original contribution of the present study. Findings show that among Pacific people in particular, there is a pressing need to reduce concerns about diabetes medication and emotional distress about diabetes while continuing to focus on improving glycaemic control. Diabetes nurse educators, diabetes specialist nurses, and health psychologists could play an important role in delivering this type of care in collaboration with primary care and specialist physicians. The recent development of clinics that provide psychological support for people with an ongoing physical illness including diabetes is very timely, and we encourage staff at these clinics to carefully consider the needs of Pacific peoples with diabetes.

Limitations and directions for future research—The cross-sectional design of the current study precludes the interpretation of causal relationships and some caution in the generalisability of results is suggested as there were small numbers of participants in some subgroups (e.g., Pacific peoples). The response rate is modest (62%) and could be improved in future research. Two identifiable strengths of this study are the inclusion of both clinical and psychological data; and the close match between the cultural composition of the research sample and the population of interest (people with Type 2 diabetes in New Zealand).

There are reasons to interpret the results of this study with some caution. First, many people may identify with more than one ethnic group. However in the primary care database used to identify potential participants ethnicity was recorded as a single variable with mutually exclusive categories (e.g., New Zealand European; Māori; Pacific; or Other). Second, differential non-response was associated with ethnicity in this study and may represent a potential compromise to the external validity of the study. The use of an English-language questionnaire may have reduced the response rate among those for whom English is a second language. Further the absolute number

of Pacific Island participants in this study (n = 39) is small. We recommend that further research is undertaken to test the validity of our findings and to determine whether the results that we report are representative of the broader experiences of Pacific peoples with Type 2 diabetes in New Zealand.

Findings from the present study may be useful to guide the direction of future research. Results highlight the poor health outcomes experienced by Pacific people with diabetes; however further research is needed to help us understand how these come about. For example, we need to examine the causal relationships between HbA_{1c} and diabetes-related distress. Poor metabolic control (with the associated increase in complications) may cause an increase in diabetes-related distress: alternatively, high distress and concern about medication could reduce medication adherence, and thus lead to lower metabolic control. Prospective cohort studies will help to disentangle the direction of causality. Future research could also examine variation in health outcomes between Pacific subgroups (e.g. Samoan, Tongan, and Fijian) as this has been shown to be important in studies of cardiovascular disease risk[31] but was not feasible in our study due to small group sizes.

Competing interests: The author has no competing interests to declare.

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Standardisation of reporting haemoglobin A_{1c}: adoption of the New Zealand Society for the Study of Diabetes (NZSSD) position statement

Chris Florkowski, Michael Crooke

The haemoglobin A_{1c} (HbA_{1c}) assay has become the gold-standard measurement of chronic glycaemia, providing an integrated index of glycaemic control over the preceding 2–3 months and with elevated values related to increased risk of microvascular and probably macrovascular complications of diabetes mellitus.[1] The present article describes some of the issues related to HbA_{1c} and how global initiatives have addressed the non-standardisation of this assay, culminating in a major international consensus statement, with implications for the way HbA_{1c} is reported world-wide from clinical laboratories.

At a meeting in Milan on 4 May 2007 a consensus statement on the worldwide standardisation of HbA_{1c} measurement was endorsed by the American Diabetes Association (ADA), European Association for the Study of Diabetes (EASD), International Federation of Clinical Chemistry and Laboratory Medicine (IFCC), and the International Diabetes Federation (IDF). This statement was published in three journals[1–3] with the recommendation for implementation "globally as soon as possible".

The key tenets of the consensus statement, with implications for the way HbA1c is reported from clinical laboratories are:

- HbA_{1c} results are to be reported world-wide in IFCC units (mmol/mol) AND:
- Derived National Glycohaemoglobin Standardisation Program (NGSP) units (%), using the IFCC-NGSP master equation. (*i.e. what is currently reported by clinical laboratories*)
- If the ongoing "average plasma glucose study" fulfils its *a priori* specified criteria, an HbA_{1c} derived average glucose value calculated from the A_{1c} result will also be reported as an interpretation of the A_{1c} results.

After consultation with its membership, NZSSD produced a Position statement (see Appendix 1 at the end of this article) and endorsed recommendations 1 and 2 above, although not 3 for reasons to be discussed. These recommendations have now been implemented in New Zealand clinical laboratories from 03 August 2009. http://www.nzssd.org.nz/position_statements/standardisation.html

The background to these recommendations

HbA_{1c} came to the fore with the publication of the DCCT trial[4] in 1993 and subsequently the UKPDS study (in Type 2 diabetes).[5] Both studies showed that there was a curvi-linear positive relationship between HbA_{1c} values and diabetes complications and enabled the setting of targets for management (≤7% is desirable)[6]

and in some centres, a change of therapy is recommended at levels above 8%. However, it became apparent at the time of the DCCT trial that there were widely differing HbA_{1c} results between laboratories, reflecting widely different analytical principles and also the lack of standardisation between assays.

The DCCT trial employed a high performance liquid chromatography (HPLC) method called the "Biorex-70". In recognition of the need for better harmonisation between assays, the National Glycohaemoglobin Standardisation Program (NGSP) (http://www.ngsp.org) was established in the USA. This organisation developed a network of reference laboratories and produced standards, based on whole blood samples, with HbA_{1c} values traceable to the "Biorex-70" method. This enabled traceability of results to the DCCT method and thus to the patients and clinical outcomes in that landmark trial. Working through both manufacturers and clinical laboratories, the NGSP achieved better standardisation and by the year 2001, there was evidence from Quality Assurance Programmes that HbA_{1c} results from different laboratories were in much tighter agreement.

However, the problem is that what underlies the HbA_{1c} peak on the Biorex-70 chromatogram is not "pure" HbA_{1c}, but rather a mixture of substances. HbA_{1c} refers strictly to Hb glycated at the N-terminal valine residues of the beta chains, whereas the peak contains Hb glycated at other sites, some HbF and the "uraemic-adduct" (Hb with urea attached). "Pure" HbA_{1c} may represent only 60-70% of what underlies the peak on the chromatogram. For this reason, the International Federation of Clinical Chemistry (IFCC) from the mid 1990s moved to develop a reference method with true primary standards.

The IFCC achieved this using the N-terminal hexa-peptide of the haemoglobin beta chain in both glycated and unglycated forms and methods based on mass spectrometry or capillary electrophoresis and also developed an international network of reference laboratories[7]. This is now in place with the methods accepted by the Joint Committee for Traceability in Laboratory Medicine (JCTLM) and the IFCC HbA_{1c} laboratory network providing reference laboratory services[8].

However, the issue is that the HbA_{1c} results that are IFCC aligned are *lower* than those that are NGSP (or DCCT) aligned by an absolute value of 1-2%. For example, 7% by DCCT would be reported as 5.4% by IFCC. Manufacturers are obliged to use calibrators and controls that are traceable to a higher order reference method (IFCC aligned), though currently use "master equations" to convert HbA_{1c} results into values that are NGSP (or DCCT) aligned and which are currently reported.

The main recommendation[1–3] is to use the alternative molar units proposed by the IFCC, namely mmol/mol (haem) for reporting of HbA_{1c}.

See Table 1 for equivalent values.

Table 1. relationships between HbA_{1c} in NGSP % units, HbA_{1c} IFCC mmol/mol units and estimated average glucose (eAG, mmol/L).

HbA_{1c} NGSP (%)	HbA_{1c} IFCC (mmol/mol)	eAG (mmol/L)
5.0	31	5.4
6.0	42	7.0
7.0	53	8.6
8.0	64	10.2

Arguments for change to IFCC Units (mmol/mol)

- Current % unit changes appear small and may be considered unimportant by some patients (e.g. changes of 0.5%).
- The numbers for NGSP units (e.g. 6.8) are similar to those used for blood glucose concentration when measured in mmol/L, which leads to confusion in some patients.
- The IFCC units are scientifically valid and accurately indicate the amount of HbA_{1c} present in the sample. By contrast the NGSP units refer to a non-specific assay which measured other forms of haemoglobin in addition to HbA_{1c}.

Arguments in favour of retaining NGSP % (or DCCT aligned) results.

- Familiar to patients, carers, educators, doctors, labs, manufacturers.
- These units are used in peer-reviewed literature, brochures, treatment guidelines and on analyser readouts.
- The values relate directly to current evidence (e.g. DCCT, UKPDS, others).
- Any change in units is likely to create mishaps. Described by some authors as potentially leading to "great confusion".

The argument, however is not a choice of one unit or the other at this time as the recommendation is for the result to be reported with two values reported with each unit. It has been noted that if both units are reported then users will probably look no further than the unit they are familiar with.

The proposal to report estimated average glucose (eAG)

The expression of HbA_{1c} as an estimated average plasma glucose (eAG) in addition to the HbA_{1c} result is supported in the text of the consensus statement[1-3] as follows: "expressing test results in scientifically correct units along with a clinically relevant interpretation of those results is not an uncommon practice (e.g. creatinine and estimated glomerular filtration rate).

Consequently, clinicians will have the opportunity to convey the concept of chronic glycaemia in terms and units most suitable to the patients under their care."

The proposal originally stems from the observed relationship between HbA_{1c} and average blood glucose in the DCCT trial.[9] However, the relationship shows a wide scatter of average glucose levels for any HbA_{1c}, leading to the suggestion that there is a spectrum from slow to fast "glycators".

More recently, the A_{1c}—derived average glucose study (ADAG)[10] reported the relationship between HbA_{1c} measured at the end of 3 months and the weighted average glucose from at least 2 days of continuous glucose monitoring performed four times, and seven-point daily self-monitoring of blood glucose performed at least 3 days per week. This represented approximately 2700 glucose readings per subject and was undertaken in a total of 507 subjects, including 268 patients with Type 1 diabetes, 159 with Type 2 diabetes and 80 normal subjects.

Participants were aged 18-70 and patients with diabetes had stable glycaemic control (HbA_{1c} values within 1% over a 6 month period), with a range of HbA_{1c} values up to approximately 12%. The study was undertaken in 11 centres in the USA, Europe and Africa. The derived regression equation showed a lower eAG compared with DCCT and with less scatter, thus fulfilling the *a priori* quality criterion that 90% of estimates fell within ±15% of the regression line.

There were no differences in the relationship according to diabetes type or ethnic group, although there was a trend to lower eAG in African Americans. Asian ethnic groups were under represented in the study and children were excluded. Those with haemoglobinopathies, likely to confound interpretation of HbA_{1c} were also excluded from the study.[10]

The accompanying editorial[11] and others[12] have advocated introduction of eAG into reporting of results.

Arguments in favour of routinely reporting the eAG

- Reporting HbA_{1c} with an estimated average blood glucose should assist with patient's understanding of the results.
- A number of clinicians in Australasia have already expressed a preference for this type of additional reporting.
- If patients can gain an improved understand of the meaning of the HbA_{1c}, improved outcomes may be achieved.
- The test name "HbA_{1c}" is confusing as haemoglobin usually refers to the red cells.

Arguments against routinely reporting the eAG

- The nature of the relationship between HbA_{1c} and average blood glucose remains poorly understood.
- There is considerable scatter around the line used to convert the HbA_{1c} results to eAG in the DCCT, ADAG and other studies.
- The term "Average Blood Glucose" has different meanings depending on the method used to determine it. For example average glucose can be obtained

from many home blood glucose meters with limited testing, more detailed meter testing (e.g. 7 times per day) or continuous monitoring.

- There is the possibility of confusion over the response to eAG (decisions about long term management changes) and single blood glucose measurement (decision about immediate changes).
- The range of values reported in patients using calculated eAG is much narrower than from random glucoses. While the eAG values are clearly significant in terms of HbA_{1c} values, they may be seen as being not significant if they were fingerprick glucose readings—e.g. random glucose readings of 7.0 mmol/L and 10.2 mmol/L are close, however the HbA_{1c} results that generate these values as eAG are markedly different (6% and 8%).
- This method of reporting may have little benefit in understanding for patients with Type 2 diabetes who are not performing home blood glucose monitoring.
- At this time we are unaware of any evidence that reporting in this manner will improve outcomes.

Manufacturers of Point of Care testing devices will also need to make adjustments in order to allow for two (HbA_{1c} in % and mmol/mol) or possibly three (also eAG) results to be displayed on a screen or printout. Until these adjustments are made, they will lag behind in the changeover process.

A corollary to all of the above is the additional recommendation,[1-3] namely that "glycaemic goals appearing in clinical guidelines should be expressed in IFCC units, derived NGSP units, and as estimated average glucose". This recommendation is a vital adjunct to the main recommendations. If reporting of laboratory results is changed, then the documentation available to doctors, diabetes educators and patients must also be expressed in the relevant units.

The process of making change

A recent multidisciplinary meeting in the UK with wide representation has considered these issues.[13] The use of IFCC molar units is supported but with recognition of the major educational requirements and lengthy period of dual reporting. The reporting of eAG was not supported at this time although further research was recommended[13]. Other editorials also have not been supportive of reporting of eAG.[14] NZSSD has consulted its Membership, having presented all the information above and reviewed the feedback in the formulation of its Position Statement, the key points of which were implemented in New Zealand on 3 August 2009.

In Australasia there ideally needs to be broad consensus between the clinical and laboratory organisations. Stakeholders for the clinical side include the Australian Diabetes Association (ADA), the New Zealand Society for the Study of Diabetes (NZSSD), the Australia Diabetes Educators Association (ADEA), the Royal Australasian College of Physicians (RACP) and the Royal Australian College of General Practitioners (RACGP).

Patient representative groups through Diabetes Australia and Diabetes New Zealand (DNZ) should also be included in the process. The Royal College of Pathologists of Australasia (RCPA) and the Australasian Association of Clinical Biochemists

(AACB) are the laboratory professional bodies involved with this issue. In Australia, the issues are still being deliberated with no firm plan of action set.

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Appendix 1. NZSSD Position Statement on standardisation of reporting units for HbA_{1c} and application of estimated average glucose (eAG)

New Zealand (NZ) clinical laboratories should implement dual reporting of HbA_{1c} in both molar units (mmol/mol) and currently reported DCCT-aligned units (%), as recommended in a consensus statement from ADA, EASD, IFCC and IDF, published in 2007. After a period of two years from the time of implementation it is envisaged that only molar units will be reported.

Although explicit times have been set in the United Kingdom (1 June 2009 for initiation of dual reporting and 1 June 2011 for reporting only molar units), it is most important that implementation is coordinated across NZ laboratories, ideally in synchrony with Australia. The NZ clinical laboratory community should cooperate to achieve dual reporting in a standardised format.

There is some evidence in support of also reporting estimated average glucose (eAG), although this has not received universal endorsement. It is recommended that eAG may be used at the discretion of individual practitioners as an educational tool at the point of delivery of care to patients with diabetes. It is not recommended that eAG should appear on laboratory reports at the present time, although there should be flexibility to adopt this if a strong Australasian commitment emerges.

The above recommendations should be supported by educational tools and resources, which should be adapted to meet local requirements.

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How well does routine hospitalisation data capture information on comorbidity in New Zealand?

Diana Sarfati, Sarah Hill, Gordon Purdie, Elizabeth Dennett, Tony Blakely

Abstract

Aims This study aims to assess the quality of routinely collected comorbidity data in New Zealand which are increasingly used in health service planning and research.

Methods Detailed medical notes-based comorbidity data from a cohort study of New Zealanders diagnosed with colon cancer in 1996–2003, were compared with routine hospital discharge data collected from the same patients using 1-year and 8-year lookback periods. We compared agreement between data sources for individual conditions, Charlson comorbidity index scores and total comorbidity counts using McNemar's p-test and the kappa statistic. We also assessed the association of comorbidity with all-cause survival using Cox proportional hazard models using data ascertained from the two sources.

Results Among these 569 patients, we found generally higher comorbidity was measured from notes than administrative data, with better comparability with an 8-year lookback period. Regardless of source of data, all measures of comorbidity significantly improved the ability of multivariable models to explain all-cause survival, but using both data sources combined resulted in better risk adjustment than either source separately.

Conclusion While differences in medical notes and administrative comorbidity data exist, the latter provides a reasonably useful source of accessible information on comorbidity for risk adjustment particularly in multivariable models.

Comorbidities are diseases or disorders that coexist with a disease of interest.[1] The importance of comorbidity has long been recognised in the clinical management of patients, but there is now increasing recognition of its importance in health related research and policy. Comorbidity can affect quality of life, increase mortality, influence treatment decisions, prolong hospitalisation and confound analysis.[1-5] As the population ages, these issues will become increasingly common and pressing.

To date there has been very little work published on comorbidity in New Zealand. Davis et al published a study in 2002[2] investigating the burden of comorbid disease in major Auckland hospitals. They found that over a third of patients admitted had at least one comorbid condition, and that comorbidity was associated with length of stay, mortality and the occurrence of adverse events. Similarly Stevens et al found that comorbidity was very common among a cohort of lung cancer patients, and that it was adversely associated with survival.[6]

Currently it is unclear how common comorbidity is in New Zealand more generally, or how well routine hospitalisation data captures information on important comorbid conditions. This latter point is important as the majority of health policy, service

planning and research projects requiring information on comorbidity will rely on secondary data. This paper aims to assess how well data on comorbidity are captured in routine databases in New Zealand by comparing detailed comorbidity data extracted by a physician from hospital records of patients with routinely collected hospitalisation data from these patients.

Methods

Data for this study come from two sources, firstly from a cohort study which investigated factors affecting colon cancer survival; and secondly from routine hospitalisation data obtained from New Zealand Health Information Service (NZHIS).

Cohort study—Details of this study are available elsewhere.[7] In brief, the cohort was made up of patients with first primary colon cancer diagnosed between 1996 and 2003, and notified to the New Zealand Cancer Registry (ICD-10-AM site codes C18-C19 excluding 18.1). Patients were ineligible if they were less than 25 years at diagnosis, or were diagnosed after death. All Māori patients meeting the above criteria were included along with an approximately equal number of randomly-sampled non-Māori patients. This was to allow an assessment of survival disparities between Māori and non-Māori patients with colon cancer.[7]

Clinical data were abstracted directly from patients' hospital medical notes during 2006-07. These were recorded on a standardised form by a physician (SH) and double-entered into an electronic database. Data were collected on all major comorbid conditions present at the time of diagnosis and all conditions included in the Charlson comorbidity index.

The Charlson index was developed in 1987 using data from a cohort of 607 medical patients, and validated with a population of breast cancer patients. Nineteen conditions are allocated a weight of 1 to 6 depending on the adjusted relative risk of 1-year mortality, and summed to give an overall score. [8]

In addition to the conditions included in the Charlson Index, data were collected on the following conditions: angina, essential hypertension, cardiac arrhythmias, previous pulmonary embolism, cardiac valvular disease, inflammatory bowel disease, other neurological conditions (including multiple sclerosis, Parkinson's disease, other abnormal movement disorders, epilepsy, spinocerebellar disease, anterior horn disease, other disease of the spinal cord, other demyelinating diseases of the CNS, cerebral palsy, myoneural disorders and muscular dystrophies) and major psychiatric conditions (including schizophrenia, bipolar disease, and depressive psychosis).

Comorbidities were classed in three different ways:

- The total number of comorbid conditions ('comorbidity count') was summed for each patient and categorised into four groups 0, 1, 2 or 3+ conditions;
- Charlson comorbidity scores were categorised into 0, 1, 2 or 3+; and
- Specific comorbid conditions were individually categorised. For our calculations of cross-source agreement, we used uncategorised comorbidity count and Charlson scores.

Administrative data—Routine hospital discharge data coded to ICD-9-CM-A were obtained from NZHIS in 2005 on the cohort specified above. These data are coded routinely from patient discharge records by coders based at District Health Boards and sent electronically in agreed format to NZHIS. We treated the admission for surgical resection of colon cancer as the index admission. Where a patient did not receive surgical resection, we treated the first hospital admission with colon cancer as primary diagnosis as the index admission. Those without such an admission were excluded from the study.

One of the problems with using administrative data to assess comorbidity is deciding on an optimal comorbidity ascertainment lookback period. Shorter periods may be more likely to identify currently active health issues, while longer periods may be more likely to identify all important comorbidity.[9] In this study we assessed two lookback periods; 1 and 8 years, 8 years being the longest available time for the earliest cancer registrations.

We used both the principal and secondary diagnoses fields to identify comorbid conditions from the administrative dataset. We used the Deyo et al[10] system which provides a method of translating the Charlson index for use on administrative data using ICD coding. The algorithm was modified to take account of the fact that we collected data on additional conditions to those included in the Charlson Index. These are listed in Table 1. Because it can be difficult to differentiate between pre-existing

conditions and complications of treatment, some conditions are only included in the definition of comorbidity if they are listed prior to the index admission.

We followed the approach used by Deyo et al[10], except that we included non-colorectal malignancies in our definition of comorbidity if they were listed in index or prior hospital discharges.[11]

Table 1. Diagnostic codes used for mapping

Diagnostic category	ICD-9 codes
Myocardial infarction	410.x, 412*
Congestive heart failure	428.x
Peripheral vascular disease	441.x*, 443.9*, 785.4*, V43.4*, procedure 38.48
Cerebrovascular disease	430-437.x, 438*
Dementia	290.x*
Chronic pulmonary disease	490-496*, 500-505*, 506.4*
Connective tissue disease	710.0-710.1*, 710.4*, 714.0-714.2*, 714.81*, 725*
GI ulcer disease	531.x-534.9*
Mild liver disease	571.2*, 571.4*, 571.5*, 571.6x*
Diabetes (mild to moderate)	250.0x-250.3x*, 250.7x*
Hemiplegia or paraplegia	342.x*, 344.1*
Moderate or severe renal disease	582.x*, 583.0-583.7*, 585*, 586*, 588.x*
Diabetes with end organ damage	250.4x-250.6x*
Any malignancy (except colon or rectal) including lymphoma or leukaemia	140.x-152.x*, 155.x-172.0*, 174.x-195.8*, 200.x-208.x*
Moderate or severe liver disease	572.2-572.8*, 456.0-456.21*
Metastatic solid tumour	196.x-199.1
AIDS	042.x-044.x
Angina‡	411.1*, 413.0*, 413.1*, 413.9*
Essential hypertension‡	401.x
Cardiac arrhythmias‡	426.x-427.x
Previous pulmonary embolism‡	415.1
Cardiac valve disease‡	394.x-397.0*, 424.0-424.3*
Inflammatory bowel disease‡	555.x*, 556.x*
Other neurological condition‡ [a]	332.x-336.x*, 340.x*, 341.x*, 343.x*, 345.x*, 358.x*, 359.x*
Major psychiatric conditions‡ [b] (with psychosis)	295.x*, 296.x*, 298.0*

* Included in definition of a comorbidity if they are listed either in the index or prior hospital discharge; other codes only included if they are recorded prior to index admission

‡ Not included as part of Charlson Comorbidity Index

[a] Includes multiple sclerosis, Parkinson's disease, other abnormal movement disorders, epilepsy, spinocerebellar disease, anterior horn disease, other diseases of spinal cord, other demyelinating diseases of CNS, cerebral palsy, myoneural disorders, muscular dystrophies.

[b] Includes schizophrenia, bipolar disease and depressive psychosis

Analysis—To calculate the maximum comorbidity we could identify from all data we had available, we first calculated the total number and proportion of patients who were recorded with each condition either in the medical notes review, or in the administrative data combined (separately for 1 and 8 year lookback). We then compared the proportion of these who had been identified in the notes, the administrative data or both, and calculated p-values using McNemar's test to test whether the number of people with the condition differed significantly between the medical notes and administrative data.

We calculated the distribution of Charlson score and comorbidity count using medical notes, and administrative data with 1 and 8 year lookback. We then measured cross-source agreement for each condition as well as for the Charlson score and comorbidity count (uncategorised) using the weighted kappa statistic with quadratic (Fleiss-Cohen) weights.[12]

This statistic approximates the intraclass correlation coefficient and provides a measure of reliability that adjusts for agreement that occurs by chance. We considered scores of <0.40 to suggest poor agreement, 0.40 to 0.74 to suggest moderate agreement and 0.75 or higher to suggest very good agreement.

We assessed the association of comorbidity and all-cause survival among this cohort with colon cancer using Cox proportional hazards regression models. We fitted a baseline model that included sex, age, and ethnicity, year of registration, stage, grade and site of disease. The fit of the baseline model was compared to various models that included comorbidity using the likelihood ratio test. For these models comorbidity was measured using Charlson categories or individual conditions.

The conditions were selected on the basis that they had been previously shown to be related to survival from colon cancer in this cohort[4], and that there were a minimum of 10 cases within the cohort (these conditions were previous myocardial infarction, congestive heart failure, diabetes, chronic respiratory disease, renal disease, cardiac arrhythmias, non-cerebrovascular neurological conditions and peripheral vascular disease). We compared results from models that included comorbidity measured using data from medial notes to those using administrative data.

Often comorbidity will not be an exposure of interest, but a potential confounding factor in another putative association. Researchers therefore have an interest in knowing how much of the 'true' confounding by comorbidity might be captured when adjusting for a misclassified measure such as that from routine administrative data. We explored this for the putative association of ethnicity with survival, and how much of the association might be due to confounding/ mediation by comorbidity (we know that Māori experience poorer survival from colon cancer than non-Māori, and that some of this association is due to Māori carrying a higher burden of comorbidity than non-Māori[7]).

We measured the hazard ratio for all-cause mortality of Māori compared with non-Māori having adjusted for sex, age, year of registration, stage, grade and site. We then added to the model comorbidity measured using the individual conditions specified above identified either in the notes, or in the administrative data to assess the extent to which each changed the underlying hazard ratio.

Approval for this study was granted by the New Zealand Multi-Region Ethics Committee.

Results

A total of 685 patients met the eligibility criteria for the cohort study, and full data were obtained for 92% of eligible patients to give an initial study sample of 642 (308 Māori and 334 non-Māori). When these cases were matched to the routine hospitalisation data, 73 were excluded because they did not have an admission that met the criteria for the index admission giving a final cohort for this study of 569 patients, 515 having an admission for surgical resection of colon cancer.

Tables 2 and 3 show the comparison of medical notes data with administrative data with 1- and 8-year lookback respectively. They show that there were considerable differences in the comorbidity data obtained from these two data sources. For most conditions, higher numbers of patients were identified with notes review data than administrative data, and this effect was more marked with 1-year than 8-year lookback.

This pattern was reversed for diabetes and renal disease for both lookback periods, as well as non-colorectal malignancy, cardiac valve disease and hemiplegia with the longer lookback period. There was very good agreement (kappa=0.77 and 0.75 for 1- and 8-year lookback respectively) between the sources of data for only one condition (mild to moderate diabetes).

For the 1-year lookback, 11 conditions showed moderate agreement (kappa 0.40 to 0.74), and the remaining five showed poor agreement (kappa<0.40). Agreement between the two data sources improved with the longer lookback period with 14 conditions showing moderate and two showing poor agreement.

Table 2. Comparison of ascertainment of comorbidity using data from medical notes or administrative data from index admission and 1 year prior

Condition	Total number (%) with condition recorded in notes or admin data		Total no (%*) in notes		Total no (%*) in admin data		Total no (%*) in both		p-value**	Kappa coefficient	95% confidence Intervals for kappa
Myocardial infarction	53	(9.3)	49	(92.5)	21	(39.6)	17	(32.1)	<0.001	0.46	0.31-0.60
Congestive heart failure	74	(13.0)	64	(86.5)	30	(40.5)	20	(27.0)	<0.001	0.38	0.25-0.51
Peripheral vascular disease	27	(4.7)	24	(88.9)	13	(48.1)	10	(37.0)	0.013	0.53	0.33-0.72
Cerebrovascular disease	46	(8.1)	39	(84.8)	15	(32.6)	8	(17.4)	0.001	0.27	0.11-0.43
Dementia	14	(2.5)	13	(92.9)	5	(35.7)	4	(28.6)	0.021	0.44	0.15-0.72
Chronic pulmonary disease	141	(24.8)	128	(90.8)	74	(52.5)	61	(43.3)	<0.001	0.53	0.44-0.61
GI ulcer disease	26	(4.6)	22	(84.6)	9	(34.6)	5	(19.2)	0.007	0.31	0.09-0.52
Diabetes (mild to moderate)	94	(16.5)	73	(77.7)	84	(89.4)	63	(67.0)	<0.001	0.77	0.69-0.85
Diabetes with end organ damage	28	(4.9)	21	(75.0)	18	(64.3)	11	(39.3)	0.630	0.55	0.36-0.74
Hemiplegia or paraplegia	15	(2.6)	9	(60.0)	12	(80.0)	6	(40.0)	0.511	0.56	0.31-0.82
Moderate or severe renal disease	24	(4.2)	7	(29.2)	22	(91.7)	5	(20.8)	<0.001	0.33	0.11-0.55
Any malignancy (except colon or rectal) including lymphoma or leukaemia	39	(6.9)	25	(64.1)	27	(69.2)	13	(33.3)	0.857	0.48	0.30-0.65
Angina‡	74	(13.0)	69	(93.2)	22	(29.7)	17	(23.0)	<0.001	0.33	0.21-0.46
Essential hypertension‡	239	(42.0)	216	(90.4)	152	(63.6)	129	(54.0)	<0.001	0.56	0.49-0.63
Cardiac arrhythmias‡	82	(14.4)	78	(95.1)	30	(36.6)	26	(31.7)	<0.001	0.44	0.32-0.56
CV valve disease‡	22	(3.9)	13	(59.1)	15	(68.2)	6	(27.3)	0.801	0.41	0.18-0.65
Other neurological condition‡ [a]	17	(3.0)	13	(76.5)	9	(52.9)	5	(29.4)	0.391	0.44	0.18-0.71

*As a percentage of Column 1 (Total number with condition recorded in notes or admin data); **Testing whether the proportion of patients with condition is significantly different between the medical notes and administrative data; ‡Condition not included in Charlson Comorbidity Index.

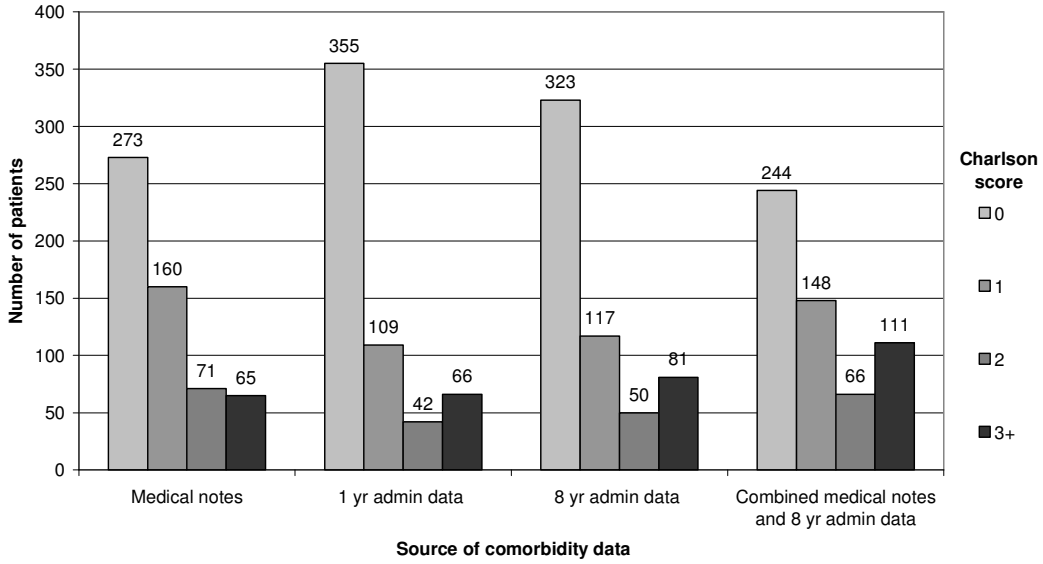
Table 3. Comparison of ascertainment of comorbidity using data from medical notes or administrative data from index admission and 8 years prior

Condition	Total number (%) with condition recorded in notes or admin data		Total no (%*) in notes		Total no (%*) in admin data		Total no (%*) in both		p-value**	Kappa coefficient	95% confidence Intervals for kappa
Myocardial infarction	55	(9.7)	49	(89.1)	35	(63.6)	29	(52.7)	0.009	0.67	0.55-0.79
Congestive heart failure	80	(14.1)	64	(80.0)	49	(61.3)	33	(41.3)	0.040	0.54	0.42-0.66
Peripheral vascular disease	29	(5.1)	24	(82.8)	19	(65.5)	14	(48.3)	0.302	0.64	0.47-0.81
Cerebrovascular disease	49	(8.6)	39	(79.6)	27	(55.1)	17	(34.7)	0.050	0.49	0.33-0.64
Dementia	14	(2.5)	13	(92.9)	6	(42.9)	5	(35.7)	0.039	0.52	0.25-0.79
Chronic pulmonary disease	147	(25.8)	128	(87.1)	91	(61.9)	72	(49.0)	<0.001	0.58	0.49-0.66
GI ulcer disease	28	(4.9)	22	(78.6)	13	(46.4)	7	(25.0)	0.078	0.38	0.17-0.59
Diabetes (mild to moderate)	99	(17.4)	73	(73.7)	90	(90.9)	64	(64.6)	0.006	0.75	0.67-0.83
Diabetes with end organ damage	29	(5.1)	9	(72.4)	8	(69.0)	12	(41.4)	1.00	0.57	0.39-0.75
Hemiplegia or paraplegia	18	(3.2)	9	(50.0)	16	(88.9)	7	(38.9)	0.065	0.55	0.31-0.79
Moderate or severe renal disease	25	(4.4)	7	(28.0)	23	(92.0)	5	(20.0)	<0.001	0.32	0.10-0.54
Any malignancy (except colon or rectal) including lymphoma or leukaemia	42	(7.4)	25	(59.5)	33	(78.6)	16	(38.1)	0.170	0.53	0.37-0.69
Angina‡	76	(13.4)	69	(90.8)	39	(51.3)	32	(42.1)	<0.001	0.55	0.44-0.67
Essential hypertension‡	247	(43.4)	216	(87.4)	175	(70.9)	144	(58.3)	<0.001	0.60	0.53-0.67
Cardiac arrhythmias‡	92	(16.2)	78	(84.8)	54	(58.7)	40	(43.5)	0.001	0.56	0.45-0.66
CV valve disease‡	26	(4.6)	13	(50.0)	21	(80.8)	8	(30.8)	0.096	0.46	0.24-0.67
Other neurological condition‡ [a]	18	(3.2)	13	(72.2)	12	(66.7)	7	(38.9)	1.00	0.55	0.31-0.79

*As a percentage of Column 1 (Total number with condition recorded in notes or admin data); **Testing whether the proportion of patients with condition is significantly different between the medical notes and administrative data; ‡Condition not included in Charlson Comorbidity Index.

As expected, both Charlson scores and comorbidity counts tended to be higher when calculated from data extracted from medical notes than from administrative data with 1 or 8 year lookback, and the highest scores were obtained by combining both data sources (Figures 1 and 2). For the Charlson index, agreement between the medical notes data and the administrative data was somewhat better for the longer lookback period (kappa=0.66; 95% CI: 0.57-0.75) than the shorter one (kappa =0.61; 95% CI: 0.51-0.70).

Figure 1. Comparison of Charlson comorbidity scores calculated using medical notes or administrative data



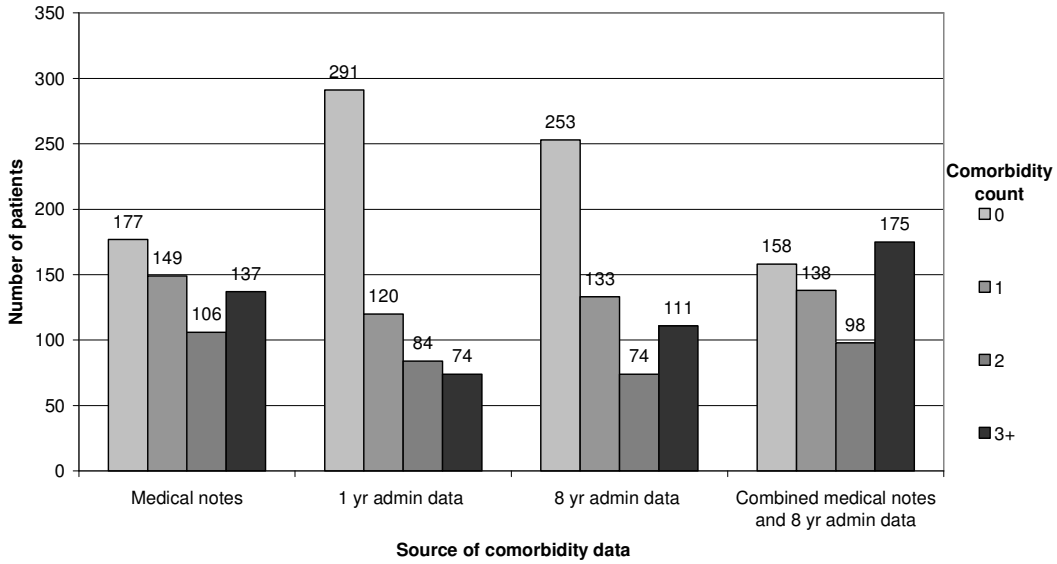
A similar pattern was seen for comorbidity count, although because more conditions were included in this count, scores were generally higher (Figure 2). The agreement between notes and administrative data was also better with kappa coefficients of 0.66 (95% CI 0.60-0.73) and 0.77 (95% CI 0.72-0.81) for administrative data with 1 and 8 year lookback respectively.

We found that comorbidity measures added significantly to the ability of the base model to explain all-cause survival regardless of whether comorbidity was measured using the Charlson score or individual conditions, or whether data was collected from medical notes, administrative data or both (in all cases likelihood ratio test $p<0.0001$ for model including comorbidity measured compared with base model).

In this cohort, we found that after adjusting for sex, age, year of registration, stage, grade and site, the baseline hazard ratio of all-cause mortality for Māori compared with non-Māori was 1.34 (95% CI 1.03-1.74). When we adjusted for comorbidity using data from both sources combined, the excess hazard ratio decreased to 1.17

(0.89-1.53). Adjusting for comorbidity using either notes or administrative-based data alone resulted in somewhat less reduction in the hazard ratio to 1.23 (95% CI 0.94-1.60), and 1.26 (95% CI 0.96-1.64) respectively.

Figure 2. Comparison of comorbidity counts calculated using medical notes or administrative data



Discussion

We found that there were considerable differences in the comorbidity data held in the routine administrative hospitalisation database in New Zealand compared with that collected by a physician from medical records. In general, more comorbidity was identified from medical records, however some conditions were more frequently identified from administrative data notably diabetes and renal failure. Agreement between the two data sources improved with a longer lookback period for the administrative data. Despite these differences, any of the measures of comorbidity that we used, regardless of the source of the data, improved the ability of multivariable model to predict all-cause survival in this cohort of colon cancer patients.

This is the first study in New Zealand to assess the quality of routinely collected comorbidity data, which are being increasingly used for health service funding and planning, and research. This is reasonable because although medical notes review data is generally considered superior to administrative data, it is not gold standard. While there may be concern about the accuracy of diagnoses recorded in administrative data, medical notes are also not entirely complete, standardised or error free.[13]

Furthermore, the results here and elsewhere clearly show that administrative comorbidity data are not a subset of medical notes data, and it is likely that combining datasets provides less misclassification of comorbidity than either source alone.[13-15] This is, of course, rarely possible.

Given that both sources result in misclassification of the (immeasurable) underlying construct of 'true' comorbidity, it is also possible, or even likely, that each of the sources of data correlates more strongly with this third measure than they do with each other, assuming that the misclassification errors in administrative and notes review data are independent of each other. That is, the kappa comparing the administrative and note-based comorbidity indices probably underestimate the correlation of each with a 'true' measure of comorbidity (unless errors in administrative and notes-based measures are moderately to highly correlated).

Furthermore, routinely collected data are considerably more accessible for large population groups than notes review, and a number of approaches to dealing with administratively collected comorbidity data are possible depending on the purpose of the data, and the outcome being assessed.[1, 16-20]

Our finding that medical notes review results in higher ascertainment of comorbidity is consistent with other studies. [13-15, 21, 22] The extent of this difference depends on a number of factors including the measure or condition that is being compared, the mapping algorithm used and the lookback period used for administrative data. There is considerable variability between conditions in terms of their ascertainment in administrative compared with medical notes data.

For the administrative data with 8-year lookback, this varied from kappa coefficients of 0.32 to 0.75. This variation is likely to depend in part on the seriousness of the condition, and coding practices relating to administrative data. As a general rule in New Zealand, comorbidities are only coded in administrative data if they co-exist or arise during a given episode of care *and* that they affect patient management in a way which might extend length of hospital stay. This approach is likely to result in an emphasis on the most active and clinically important conditions, and will explain some of the difference between notes and administrative comorbidity data.

It is not entirely clear how one should map conditions from clinical notes to ICD codes, and there has been dissent expressed on this in the literature.[10,11,21,23,24] We employed a commonly used approach, but one that has also been criticised by some authors.[11,21] For example, we found that for six of the nine mismatches for diabetes with end organ damage, had been coded as diabetes without mention of complication.

Currently no gold standard mapping approach has been developed. The length of the lookback period also makes a difference, but the ideal lookback period seems to depend on the outcome for which the data is being collected.[9,25] For example Preen et al (2006)[9] found that a one-year lookback provided better comorbidity data to predict mortality while five-year lookback was better for readmission rates. In our study the longer lookback period seemed to give more comparable data to the notes review.

Both the kappa coefficients for the individual conditions and those for the Charlson and comorbidity count (0.66 and 0.77 respectively) compare favourably with similar comparisons carried out elsewhere.[15,22] For example, Kieszak et al[22] compared comorbidity derived from medical notes with administrative data in the United States

and found that only three of 16 individual conditions had kappa coefficients greater than 0.4 (compared with 15/17 for our data with 8 year lookback), and that the correlation between the notes Charlson index and the administrative index was only 0.47.

Regardless of the source of data used, we found that any measure of comorbidity improved multivariable model fit compared with using none. We also found in this study that using data from both sources combined resulted in somewhat better risk adjustment than either source separately. However, both the notes and administrative-based comorbidity measures substantially reduced the excess mortality hazard for a model comparing Māori with non-Māori for colon cancer survival, although more so for the use of notes-based comorbidity index consistent with an *a priori* expectation that it is a superior estimate of comorbidity.

Furthermore, given that including both the notes and administrative-based measures of comorbidity resulted in greater reduction again in the hazard ratio, it seems reasonable to conclude that neither measure alone (notes or administrative-based) fully captures the confounding or mediating effects of comorbidity.

Of note, is that this study focused solely on patients with colon cancer. Patients with other primary conditions may have different patterns of comorbidity, but it seems unlikely that this will affect the quality of the recording of their comorbidity data. In that respect, it seems reasonable to be able to generalise the findings of this study to hospital-based comorbidity data in New Zealand.

In conclusion, measuring comorbidity is potentially important for risk adjustment in health service policy, funding and planning, and health-related research. Data from clinical notes review are often considered superior but are rarely available. The correlation between clinical notes and administrative data in New Zealand is moderate and varies considerably between individual conditions. However, administrative data provides a source of relatively accessible comorbidity data which we have found allows for reasonable risk adjustment in the cohort presented here, although not quite as good as for a notes-based or combined comorbidity measure.

Competing interests: None known.

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A population-based approach to the estimation of diabetes prevalence and health resource utilisation

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Abstract

Aim This study estimated diabetes prevalence and utilisation of healthcare services in Counties Manukau using routinely collected administrative data and compared estimates with findings for three other district health boards (DHBs) in close geographic proximity.

Method Records of subsidy claims for pharmaceuticals and laboratory investigations were linked to records in a national hospital admissions database to 'reconstruct' populations of four DHBs—Counties Manukau, Northland, Waitemata and Auckland. Individuals were included in reconstructed populations if they had health events recorded between January 2006 and December 2007. Diabetes cases were identified using an algorithm based on claims for monitoring tests and pharmaceuticals, as well as clinical codes for diabetes in hospital admissions.

Results Reconstructed populations were only 6% lower than census population counts indicating that the vast majority of the population use health services in a two year period. The age- and sex-standardised prevalence of diabetes was 7.1% in Counties Manukau and 5.2% in the other three DHBs combined. Prevalence of diabetes was highest amongst Māori (10.6% in women and 12.2% in men) and Pacific peoples (15.0% for women and 13.5% for men). Māori diabetes cases had the highest hospital discharge rate of any ethnic group. Community pharmaceutical prescribing patterns and laboratory test frequency were similar between diabetes cases by ethnicity and deprivation.

Conclusion Estimates of diabetes prevalence using linkage of routinely collected administrative data were consistent with epidemiological surveys, suggesting that linkage of pharmaceutical and laboratory subsidy databases with hospital admissions data can be used as an alternative to traditional surveys for estimating the prevalence of some long-term conditions. This study demonstrated substantial differences in the prevalence of diabetes and in hospitalisation rates by ethnicity, but measures of community diabetes care were similar by ethnicity and deprivation.

Counties Manukau District Health Board (CMDHB) is responsible for the funding and provision of health services for the people of Counties Manukau, serving around 480,000 people in 2009. It has the fastest-growing population of any district health board (DHB), the highest number of young people (aged ≤14 years), the highest numbers of Māori and Pacific peoples and the highest number of people living in the most deprived two deciles of the New Zealand deprivation index.[1]

Diabetes is a leading cause of morbidity and mortality in New Zealand[2,3] and is particularly common in Counties Manukau. Māori and Pacific peoples, who together

make up around 40% of the Counties Manukau population, have a disproportionate burden of diabetes compared with other groups.[4–6] The 2006/07 New Zealand Health Survey (NZHS) estimated that Counties Manukau had the highest prevalence of self-reported diabetes in those aged ≥15 years of any DHB area.[7] Furthermore, in Counties Manukau around one-third of adult respondents to the NZHS were found to be obese, with body mass indices ≥30 kg/m^2.[7]

Initiatives to reduce the growing burden of diabetes in Counties Manukau are underway. *Let's Beat Diabetes* (LBD) is a long-term, intersectoral strategy which draws on wide-ranging activities such as community-based programmes, social marketing, support for primary care and collaboration with the food industry, to prevent or delay the onset of Type 2 diabetes, limit disease progression and improve quality of life for those with diabetes in CMDHB.[8] The *Chronic Care Management* (CCM) programme, a community- and primary care-focused programme, has a diabetes module aimed at structured management of individuals with complicated diabetes in the community.

In order to plan effectively for future community needs related to diabetes and understand the impact of initiatives such as LBD and CCM, timely and readily updateable estimates of the prevalence of diabetes in Counties Manukau are necessary. To take a 'whole of community' approach to planning, it is also important to gain insight into community care of diabetes, by examining factors such as community pharmaceutical prescribing patterns and laboratory monitoring.

This study estimated diabetes prevalence and utilisation of healthcare services in Counties Manukau using routinely collected administrative data and compared estimates with findings for three other DHBs in close geographic proximity.

Method

Study population—We linked three routinely collected administrative health databases in four DHBs: Counties Manukau, Northland, Waitemata and Auckland. Data from the National Minimum Data Set (NMDS—a national database containing details of all public and private hospital discharges from 1990 onwards) were linked with 30 months of community laboratory and pharmacy subsidy claims data for the period July 2005 to December 2007. Laboratory and pharmacy claims data containing details of reimbursement claims for subsidised community laboratory tests and pharmaceuticals dispensed on the New Zealand Pharmaceutical Schedule[9] were obtained from the Ministry of Health.

The three databases were linked using unique National Health Index (NHI) numbers for each case record and all NHI numbers were encrypted by the Ministry of Health prior to analysis, to avoid identification of individuals. Individuals were included in the aggregated data set if they had any health service recorded in one or more of the three data sets in the 2 years between January 2006 and December 2007 (inclusive). All deceased individuals were removed from the reconstructed group using encrypted data from the Ministry of Health Mortality Collection.

Data were not available for those who did not have hospital events recorded in NMDS, or did not have claims made for subsidised pharmaceuticals or laboratory tests (with NHI numbers documented) during the study period. These aggregated data created a 'reconstructed' population of around 1.4 million people.

Identification of diabetes cases—In this study, 'diabetes' refers to all forms of diabetes mellitus. An individual from the study population defined above was identified as a 'case' if he or she met any of the following criteria: a hospital event with a principal or secondary International Classification of Diseases, 10th edition, Australian Modification (ICD-10-AM) diagnosis code E10-E14 'Impaired glucose regulation and diabetes mellitus', or the codes O24.0 to O24.3 which cover pre-existing diabetes in pregnancy since 1990, three or more HbA1c test claims within 2 years (2006/07), or two or

more community pharmaceutical dispensing claims for New Zealand Pharmaceutical Schedule therapeutic group (TG) level 2 categories 'Diabetes' and 'Diabetes management' in 2006/07.

Ethnicity—Prioritised ethnicity was used to classify ethnicity, so that individuals were categorised into only one ethnic group, according to a prioritised schedule, if more than one ethnicity was recorded in different databases. Consistent with the standard prioritisation protocol recommended by the Ministry of Health, ethnicity was prioritised in the following order: Māori, Pacific, Asian, NZ European/Other.[10]

Deprivation—Average NZDep2006 scores for the census area unit (CAU) in which a case lived were applied to each individual as a measure of socioeconomic status. NZDep2006 is a multi-dimensional index of deprivation used for small areas.[11] It includes nine dimensions covering income, home ownership, household occupancy, education, employment and access to transport and to a telephone.

Statistical analyses—Statistical analyses used Microsoft Excel™, SPSS® (version 13.0) and SAS® software applications. Prevalence estimates have been calculated using both the reconstructed population and 2006 national census estimates as denominators. Point estimates are reported for descriptive statistics and 95% confidence intervals are included where appropriate (95% CI). For direct standardisation, prevalence proportions were divided into the following age groups: 0-4 years, 10-year age intervals until age 84 years, 85+ years. Standardisation for age and sex used Statistics New Zealand national population estimates for 2006/07. Further detail on statistical methods is available in a background report.[12]

Ethical considerations—Ethical review was not required, as all unit record data were de-identified by the New Zealand Ministry of Health and no contact was made with participants. Only aggregated results have been reported.

Results

Study population—The combined data sets identified about 1.4 million people in all four DHBs, compared with 1.5 million in the March 2006 census estimate. The reconstructed population of CMDHB in 2006/07 contained 427,350 people, 6% fewer than the 454,790 identified in CMDHB in the March 2006 national census estimate. Social and demographic characteristics of the CMDHB reconstructed population are compared with 2006 national census estimates for CMDHB in Table 1.

Diabetes prevalence—Almost 27,000 diabetes cases were identified as resident in Counties Manukau in 2006/07, while 51,000 diabetes cases were identified in the remaining three northern region DHBs. This translated to a crude prevalence of 6.3% (95%CI 6.2%-6.4%) for Counties Manukau and 5.3% (95%CI 5.2%-5.3%) for the remaining three DHBs, using the reconstructed denominator. Counties Manukau had the highest age- and sex-standardised prevalence of diabetes of any of the four DHBs.

Using the reconstructed denominator, the age- and sex-standardised prevalence of diabetes in Counties Manukau was 7.1% (95%CI 7.0%-7.2%), compared with 5.2% (95%CI 5.1%-5.2%) for the remaining three DHBs combined. While a difference of 0.4% was found between the two crude Counties Manukau prevalence estimates using reconstructed and census denominators, this difference narrowed considerably with standardisation (Table 2).

Age-standardised prevalence of diabetes by ethnicity is presented in Table 3. Pacific women had the highest prevalence of any group, with an age-standardised prevalence (using reconstructed denominator) of 15.0%. Women of NZ European/Other ethnicity had the lowest diabetes prevalence of any group, at 4.0%.

Table 1. Social and demographic characteristics of reconstructed CMDHB population and estimated population in 2006 national census

	Category	Proportion of reconstructed population	Proportion of 2006 census estimate	Reconstructed count as a proportion of 2006 census count
Age group (years)	0-14	27%	25%	99%
	15-24	14%	15%	87%
	25-34	12%	13%	88%
	35-44	14%	15%	86%
	45-54	12%	13%	93%
	55-64	10%	9%	100%
	65-74	6%	5%	108%
	≥75	4%	4%	108%
Sex	Male	46%	49%	88%
	Female	54%	51%	99%
Ethnicity	Māori	15%	17%	86%
	Pacific	21%	21%	97%
	Asian	12%	16%	71%
	NZ European/ Other	52%	46%	104%
Deprivation quintile (average NZDep2006 for CAU)	Unknown	<1%	-	
	1	17%	23%	69%
	2	13%	8%	143%
	3	10%	16%	59%
	4	15%	9%	149%
	5	45%	44%	98%

Table 2. Crude and age- and sex-standardised diabetes prevalence estimates in CMDHB, 2006/07, using reconstructed and 2006 census denominators

		Prevalence (%)	95% CI for prevalence (%)	Adult (15+ years) prevalence (%)	95% CI for adult prevalence (%)
Reconstructed denominator	Crude	6.3	6.2-6.4	8.6	8.5-8.7
	Age- and sex-standardised	7.1	7.0-7.2	9.0	8.9-9.1
Census denominator	Crude	5.9	5.8-5.9	8.3	8.2-8.4
	Age- and sex-standardised	7.0	7.0-7.1	8.9	8.8-9.0

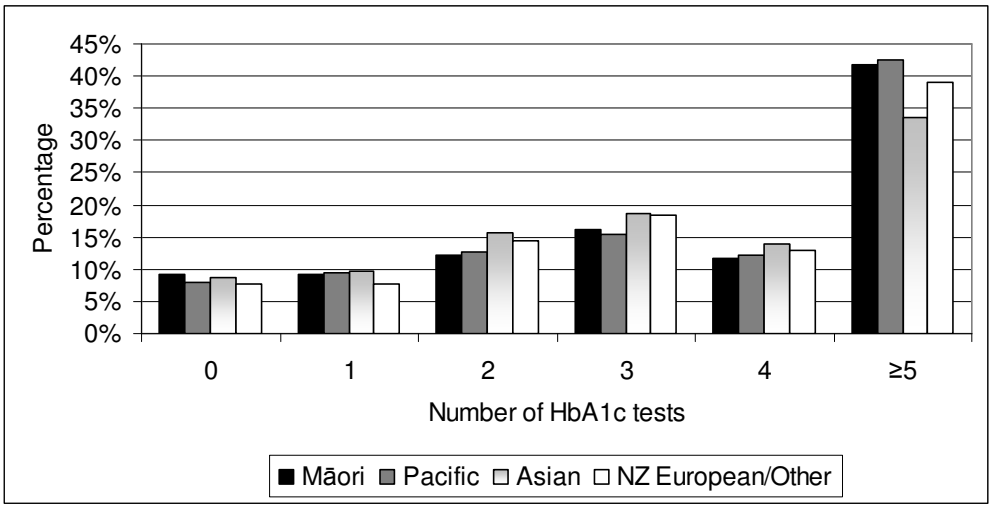
Table 3. Age-standardised prevalence of diabetes in Counties Manukau, by ethnicity, 2006/07*

	Female		Male	
	Standardised prevalence (%)	95% CI for prevalence (%)	Standardised prevalence (%)	95% CI for prevalence (%)
Māori	10.6	10.1 - 11.1	12.2	11.6 - 12.7
Pacific	15.0	14.6 - 15.4	13.9	13.5 - 14.3
Asian	9.1	8.7 - 9.4	11.3	10.9 - 11.8
NZ European/Other	4.0	3.9 - 4.1	5.0	4.9 - 5.2
Total	6.8	6.7 - 6.9	7.4	7.3 - 7.5

* Using the 'reconstructed' population as the denominator

Community care of diabetes—In Counties Manukau, 83% of diabetes cases had at least two glycated haemoglobin (HbA1c) community test claims in 2006/07, compared with 82% in the other DHBs. Just over half (52%) of Counties Manukau diabetes cases had four or more HbA1c test claims in 2006/07, versus 44% of cases in the other DHBs. Almost 40% of Counties Manukau diabetes cases had five or more HbA1c tests in 2006/07. Frequency of HbA1c test monitoring appeared consistent both by ethnicity and by socioeconomic status. Figure 1 shows the similarity between distributions of HbA1c test frequency by ethnicity in Counties Manukau, while Figure 2 compares HbA1c test frequency for those living in the most deprived areas with the remainder of diabetes cases in Counties Manukau.

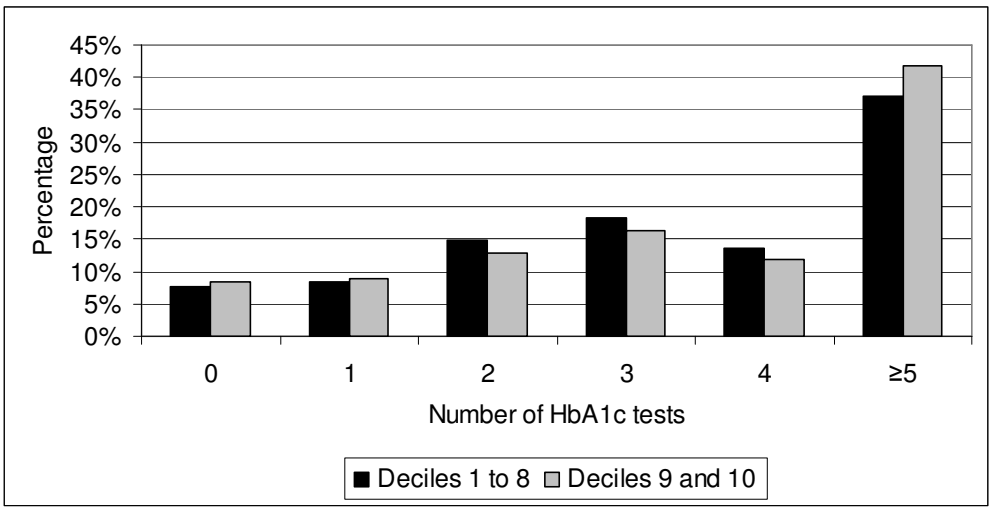
Figure 1. Frequency of HbA1c testing by ethnicity in Counties Manukau in 2006/07



Ninety-two percent of diabetes cases in Counties Manukau had fasting lipid studies performed at least once in 2006/07 (90% in other three DHBs) and 81% of Counties Manukau cases had two or more fasting lipid tests in this two-year period (75% in

other DHBs). Around one third of Counties Manukau cases had five or more claims for lipid tests in 2006/07. As with HbA1c, no important differences in serum lipid test frequency were noted by ethnicity or by socioeconomic status.

Figure 2: Frequency of HbA1c testing by NZDep2006 socioeconomic status in Counties Manukau, 2006/07



Use of a range of different medications by diabetes cases in Counties Manukau was examined. Of particular value in our understanding of quality of care was use of medications to treat secondary complications of diabetes, such as lipid lowering agents and agents affecting the renin-angiotensin system (like ACE inhibitors).

It was not possible from the data available to identify which diabetes cases 'should' have had subsidy claims for lipid lowering agents, although current guidelines suggest a high proportion of diabetes cases may benefit from them. In CMDHB, 64% of diabetes cases had at least two pharmaceutical claims for lipid lowering agents, the majority of which were HMG CoA reductase inhibitors (statins). Minor differences were identified in the proportion of diabetes cases accessing these medications by ethnicity and by deprivation (Figures 3 and 4).

Seventy percent of diabetes cases had at least two pharmaceutical claims for blood pressure-lowering medications. As expected from guidelines, claims for agents affecting the renin-angiotensin system were particularly common, with 61% of diabetes cases recording regular claims for these medications. The distribution of subsidy claims for agents affecting the renin-angiotensin system showed a relatively even spread across all ten deprivation deciles (Figures 3 and 4). Asian diabetes cases were found to have slightly lower utilisation of these medications than the other three groups.

Figure 3. Proportion of diabetes cases with regular claims for agents affecting the renin-angiotensin system and statins, by ethnicity in Counties Manukau, 2006/07

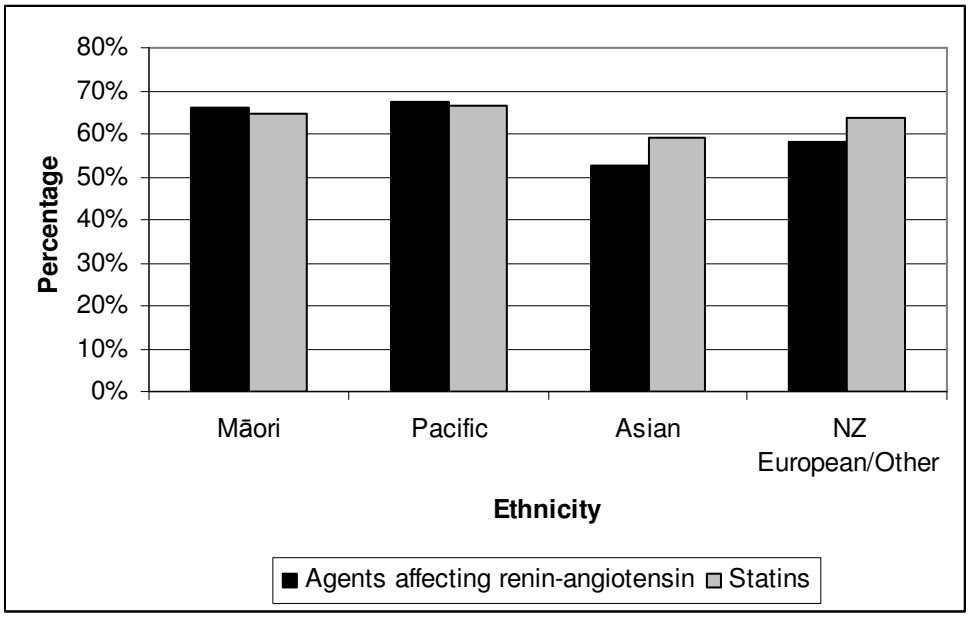
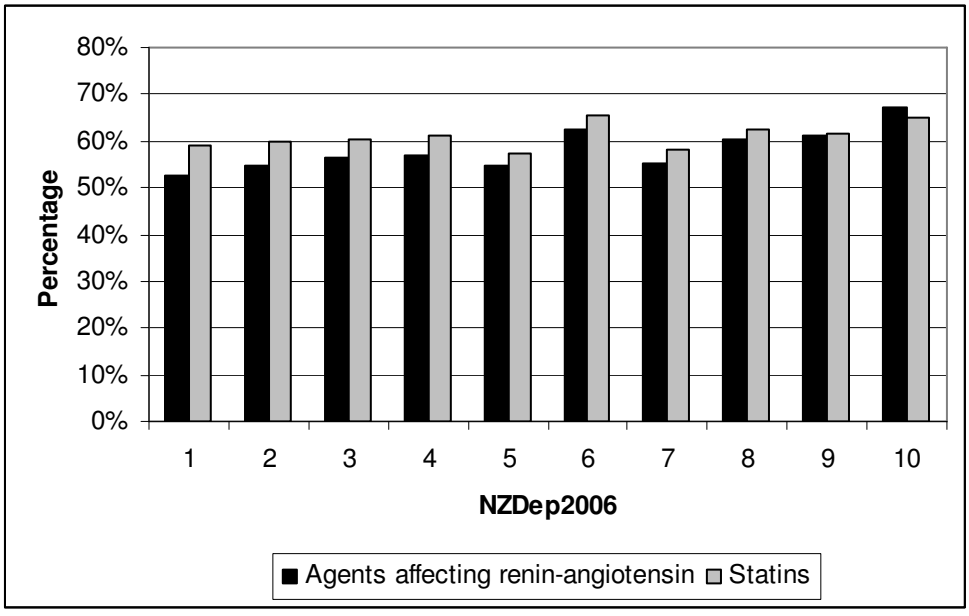


Figure 4. Proportion of diabetes cases with regular claims for agents affecting the renin-angiotensin system and statins, by socio-economic status in Counties Manukau, 2006/07



Hospital service utilisation—In 2007, 11,800 CMDHB medical and surgical hospital discharges were recorded in NMDS among diabetes cases, 17% of all hospital

discharges in that year. The average length of stay for diabetes cases was 3.6 days, compared with 2.4 days for those without diabetes.

Twenty-three percent of diabetes cases had one or more medical or surgical hospital admissions in 2007, compared with only 11% of those without diabetes in the reconstructed population. When these figures were age- and sex-standardised, 29% of diabetes cases were found to have had one or more admissions in 2007, compared with 11% of those without diabetes. Considerably more Māori diabetes cases had one or more hospital admissions in 2007 than diabetes cases of other ethnicities (Table 4).

Table 4. Proportion of diabetes cases in CMDHB with medical/surgical hospital admissions in 2007, by ethnicity

	Crude proportion of cases admitted ≥ 1 times in 2007 (%)	Age-standardised proportion admitted ≥ 1 times in 2007 (%)
Māori	31	37
Pacific	25	25
Asian	18	21
NZ European/Other	22	27

Discussion

This study used routinely collected administrative data from community laboratory and pharmaceutical subsidy claims, together with data on hospital discharges recorded in NMDS, to create a reconstructed population for four northern region DHBs. It took a 'whole of community' approach to explore both diabetes prevalence and utilisation of monitoring tests, medications and hospital services. Record linkage of administrative databases has previously been used to examine the epidemiology of diabetes in Denmark, Scotland, Canada and Sweden.[13-17] Administrative data has also been used to estimate prevalence and to review quality of care of conditions like cardiovascular disease and asthma.[18, 19] To our knowledge, this is the first study to estimate the prevalence of diabetes and resource utilisation in a DHB setting in New Zealand, using record linkage of administrative data sources.

We found diabetes to be common in Counties Manukau, with almost 27,000 people identified as diabetes cases. The age- and sex-standardised prevalence of diabetes was seven percent. This was the highest prevalence of any of the four DHBs studied and almost two percent higher than the average prevalence in the other three DHBs. Standardised prevalence estimates were similar using reconstructed and census denominators. Māori and Pacific people, in particular Māori men and Pacific women, had the highest prevalence of diabetes.

Our prevalence estimates are consistent with other estimates of diabetes in Counties Manukau. The 2006/07 New Zealand Health Survey estimated the total number of adults aged 15+ years with self-reported diabetes to be around 26,400, only around 600 less than our estimate.[7] The crude adult prevalence of diabetes in this survey was 8.2%, similar to findings in our study. The LBD 2006/07 benchmark survey of 2,520

people in Counties Manukau found an age-standardised, self-reported prevalence of 7.0% in those aged 16+ years, a finding consistent with our estimates.[5] Our findings were also comparable to diabetes prevalence estimates by ethnicity for adults aged 35-74 years in the Diabetes Heart and Health Survey (DHAH) undertaken in Auckland in 2002/03, where 9.3% of respondents were found to have Type 2 diabetes (previously- or newly-diagnosed).[4] While there were important differences between the study populations in DHAH and our study, analysis of the same age group for the three Auckland DHBs using the reconstructed population gave an estimated diabetes prevalence of 9.5%.

Key strengths in this study were currency of the data, low additional cost to the DHB of data collection, extensive detail in the three contributing databases and the ability to link numerators with denominators from the same dataset. Laboratory and pharmaceutical data came from the HealthPAC General Transaction Processing System for reimbursement of community laboratories and retail pharmacies, meaning that data became available only a few months after claims for late 2007 were processed. All data had already been collected for administrative purposes and there was no requirement for the DHB to undertake any further data collection.

Substantial data were available for analysis in the final data set. The pharmaceutical data alone contained 42 separate variables. All numerators and denominators in the analyses came from the same reconstructed population and social and demographic variables in the data analyses were directly linked, thereby avoiding numerator-denominator bias in the analysis of ethnicity[20] (although ethnicity data in hospital records can still differ substantially from self-identified ethnicity[21]).

The validity of the decision rules has not yet been formally established. Only subsidy claims for HbA1c were recorded in the data and not laboratory values. Also, only claims for community laboratory and pharmaceutical tests were available. Such data were unavailable for hospital admissions. Three approaches were taken to assess the suitability of the rules. A literature review substantiated the scope of HbA1c use and use of diabetes medications for conditions other than diabetes. Both HbA1c and diabetes medications were predominantly used in diabetes, although medications like metformin are sometimes used for other purposes.[22, 23] Individuals with local expertise in diabetes care, primary care, epidemiology and clinical coding were consulted about suitability of the rules and sensitivity analyses were undertaken using a range of test frequency and pharmaceutical dispensing claim thresholds. Further work is underway, validating the rules using capture-recapture methods. In the interim, we believe our findings are a reasonable reflection of diabetes in Counties Manukau. Not only are our estimates consistent with those of other studies, but work validating similar rules has demonstrated high levels of sensitivity and positive predictive value.[24] Some individuals at high risk of developing diabetes may have been misclassified as having diabetes. Such misclassification is acceptable for planning purposes, as these people share many of the risks experienced by those with diabetes.[25-29]

Only individuals with records of hospital events or subsidy claims during 2006/07 were included in the reconstructed population, meaning exclusion of the remainder from the reconstructed group. 'Residential churn' over the period of data collection also meant obsolescence of some addresses, despite most recent health encounter being used to allocate address. However, our estimated population for CMDHB was

only six percent less than that estimated by the 2006 national census and only small differences were seen between the socio-demographic profiles of the two groups. Use of the smaller reconstructed population as denominator resulted in over-estimation of crude diabetes prevalence, although the effect of over-estimation was lost when prevalence was age- and sex-standardised, as the bulk of diabetes cases occurred in those aged 35+ years and this group had the best coverage in the reconstructed population compared with census data.

Analysis of ethnicity using high-level categories such as 'Pacific' and 'Asian' is not without limitation and assumes ethnicities aggregated within these groups have similar health characteristics. This is not necessarily the case, as was highlighted in a report on Asian health needs which found considerable heterogeneity in health indicators between different Asian groups (such as Indian and Chinese).[30]

No data were available on appropriateness of prescribing and laboratory monitoring for individual clinical cases. For example, similarities in community care by ethnicity did not account for differences in diabetes severity. Results of the descriptive analysis were checked using logistic regression and findings for resource utilisation were consistent with those described. However, pharmaceutical and laboratory utilisation for Māori and Pacific was similar to other groups, despite substantial differences in hospital admission frequency. Further work is needed to understand whether resource use and quality of care are appropriate to disease severity and are equitable across these groups.

This study used a set of timely, comprehensive and inexpensive data to examine diabetes prevalence and resource utilisation in Counties Manukau. The analysis will be repeated regularly to inform planning for the diabetes epidemic. The methods may also be used to examine other long-term conditions and other aspects of diabetes care, such as the additional health care cost to the DHB of diabetes.[6] Disparities in the prevalence of diabetes by ethnicity were demonstrated, highlighting again the need for community-based primary prevention programmes such as LBD, which aim to address this inequity.

Competing interests: None known.

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Capillary glucose meter accuracy and sources of error in the ambulatory setting

Helen Lunt, Christopher Florkowski, Michael Bignall, Christopher Budgen

Abstract

Hand-held glucose meters are used throughout the health system by both patients with diabetes and also by health care practitioners. Glucose meter technology is constantly evolving. The current generation of meters and strips are quick to use and require a very small volume of blood. This review aims to describe meters currently available in New Zealand, for use in the ambulatory setting. It also aims to discuss the limits of meter performance and provide technical information that is relevant to the clinician, using locally available data. Commoner causes and consequences of end-user (patient and health professional) error are illustrated using clinical case examples. No meter offers definite advantages over other meters in all clinical situations, rather meters should be chosen because they fit the needs of individual patients and because the provider is able to offer appropriate educational and quality assurance backup to the meter user. A broad understanding of the advantages and disadvantages of the subsidised meter systems available in New Zealand will help the health practitioner decide when it is in the best interests of their patients to change or update meter technology.

Glucose results derived from hand-held meters are used by patients and their health care team to make therapeutic decisions such as insulin dosing. Incorrect glucose values may result in both acute and also long-term therapeutic consequences. It is therefore essential that results are as accurate and precise as possible.

Meter technology has shown incremental improvements since the introduction of the first commercially available hand-held meters in 1970s, including improvements in ease of use, technical performance and affordability.[1-3] Capillary glucose testing is an international multi-billion dollar industry.[2] In New Zealand reimbursement of test strips for the 12 months to June 2009 was $19 million, accounting for 40% of PHARMAC's entire diabetes 'spend'. The number of meters available has expanded, both in New Zealand as well as internationally.[1,2]

Currently in New Zealand, six different meters are available for use with PHARMAC funded strips (see Table 1). It is therefore timely to describe current meter technology from a clinical perspective, highlighting some of the limits of meter performance. This review focuses on technical issues that impact on clinical interpretation of meter results in the ambulatory setting. It does not aim to be a comprehensive technical discussion. Although there are additional meter systems available in New Zealand with unsubsidised strips such as the Glucocard, which is used in many hospital inpatient settings, the focus of this review is meters with subsidised strips.

Table 1. Meters with subsidised test strips

System	Accu-Chek Performa	CareSens II	CareSens POP	FreeStyle Lite	On Call Advanced	Optium Xceed
						
Manufacturer	Roche Diagnostics	i-Sens Corp	i-Sens Corp	Abbott Diabetes Care	Acon Laboratories	Abbott Diabetes Care
Test Strip	Accu-Chek Performa	CareSens (includes lancets)	CareSens (includes lancets)	FreeStyle Lite	On Call Advanced (includes lancets)	Optium
Coding	Automatic via code chip	Manual input	Manual input	Not needed	Automatic via code chip	Automatic via code chip
Test Time	5 seconds	5 seconds	5 seconds	5 seconds	5 seconds	5 seconds
Sample volume	0.6 µL	0.5 µL	0.5 µL	0.3 µL	0.8 µL	0.6 µL
Operation Temperature	6º–44ºC	10º–40ºC	10º–40ºC	4º–40ºC	5º–45ºC	10º–50ºC

The clinical impact of recent improvements in hand held blood glucose meter systems

Recent developments in meter technology have improved this testing system's ease of use and analytical robustness.[1-3] Test strips now require 8µL or less of blood (see Table 1).

Using a low blood volume system has the following advantages: It allows most patients to get a successful sample each time they undertake lancing. It allows a shallower finger lancing depth, thus patients should experience less pain.[2] In addition, the need to squeeze fingers for blood letting, a practice that may lead to a change in the effective composition of the blood test sample and a false glucose value, is reduced. Strip technology utilises a capillary filling system with a fill indicator or fill time detector to ensure that the assay does not start until sufficient blood sample is provided to the strip. This minimises the risk of obtaining a 'false low' result caused by insufficient sample volume.[3]

A development that has been appreciated by patients residing in colder areas of New Zealand, is the wider functional temperature range of meter and strip systems.[3] Historically, low winter temperatures and cold houses made it difficult for patients to obtain accurate results. A temperature sensor is now present either in the meter or in the strips. This allows correction of the glucose value for ambient temperature across a wide temperature range. Inadvertent patient use of time expired meter test strips, which often contain 'spoiled' analytical reagents, was a common source of error with older systems. This error has been minimised but not eliminated in some meter and strip systems. One example of how this is achieved, is by determining the expiry date of the strip batch from the calibration chip and pre-setting the meter software to 'disallow' strip use after the batch's expiry date.

One meter system, the Optium Xceed, can be dual calibrated to measure both glucose and also capillary ketones (beta hydroxybutryrate), allowing patients to treat mild ketoacidosis at home.

Understanding how the difference between venous and capillary samples impacts on meter performance

Finger stick test results are derived by converting an electrochemically generated signal to a glucose value by means of an algorithm. In New Zealand and most other countries, the current expectation from clinicians is that the algorithm is programmed so that a capillary whole blood glucose sample (i.e. finger stick result) will read as a laboratory venous plasma sample (i.e. a venesection result). Thus if a patient went to get a laboratory plasma venous glucose check and did a simultaneous capillary test with their meter, the expectation is that the two glucose results should read approximately the same. The comparison between a capillary finger stick test and a laboratory plasma venous glucose is not however straightforward, in part because two different types of samples are being used, which have some shared but also have some distinct physiological characteristics.[3-5]

Whole blood (e.g. a capillary sample) is composed predominantly of plasma and cells. In the laboratory, glucose is measured on a plasma sample i.e. a whole blood sample

is centrifuged, followed by removal of the cellular component of blood. Red cells have a lower water and glucose content than plasma. As a result of this, the glucose concentration of whole blood is about 11% less than the glucose concentration of plasma.[4] Historically, some meters available in New Zealand gave capillary results as a whole blood glucose equivalent. When these meter systems were updated, the algorithm was also updated to display results as venous plasma equivalent, rather than whole blood equivalent. For example, a whole blood glucose of 5.0mmol/L from an 'old' meter would equate to a plasma glucose of around 5.6mmol/L, using a 'new' meter. This change had the potential to cause confusion.

The current international recommendation, aimed to provide harmonised reporting and reduce confusion, is to report glucose results as plasma equivalent.[4] All meters currently available in New Zealand do this.

A second major difference between capillary and laboratory venous results, relates to the fact that the glucose value of a capillary sample is higher than for a corresponding venous sample, because glucose uptake by tissues as blood flows from the capillaries to the veins partially depletes the venous sample of glucose. Tissue uptake of glucose increases after food.[6] The glucose gradient between capillary and venous samples therefore shows a postprandial increase which may be as high as 20% total glucose concentration.[5,6]

In summary, because capillary and plasma glucose samples have several physiologically distinct characteristics, comparison between these two samples is not expected to correlate as closely as a comparison that uses the same type of blood sample, for example comparing the same venous sample using two different measurement techniques. Clinicians should anticipate a slight variation in glucose values between capillary and venous samples but at least 95% of capillary results should show an analytical variance of <20%, when compared to a laboratory result.[7,8]

Figures 1a and 1b illustrate this point, using results from the On Call Advanced meter (methodology is based on a previous study).[8] This meter and test strip system can measure venous as well as capillary whole blood i.e. a venous sample from the antecubital fossa can be applied directly to the test strip. Figure 1a compares a laboratory venous plasma glucose sample with a simultaneously collected capillary whole blood sample i.e. two distinct samples are used. Figure 1b compares the same venous sample, obtained from the antecubital fossa, using two different methods. Not surprisingly, visual inspection of results in Figure 1b (the same venous sample measured using two different methods) shows a closer correlation than those of 1a (separate venous and capillary samples, obtained from different anatomical sites).

Figure 1a. Comparison of venous plasma with capillary glucose (On Call Advanced)

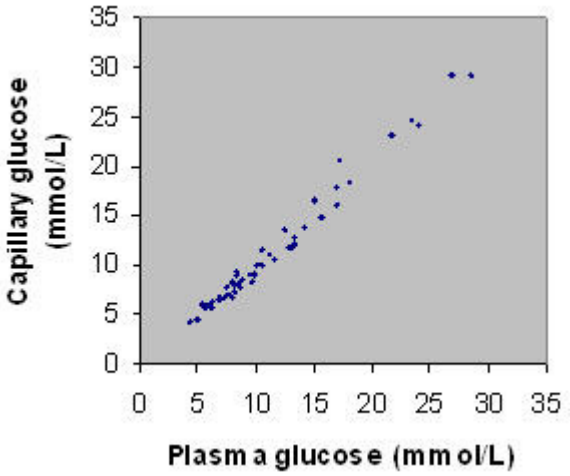
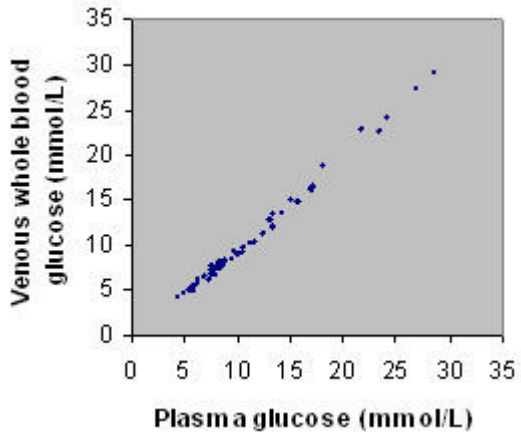


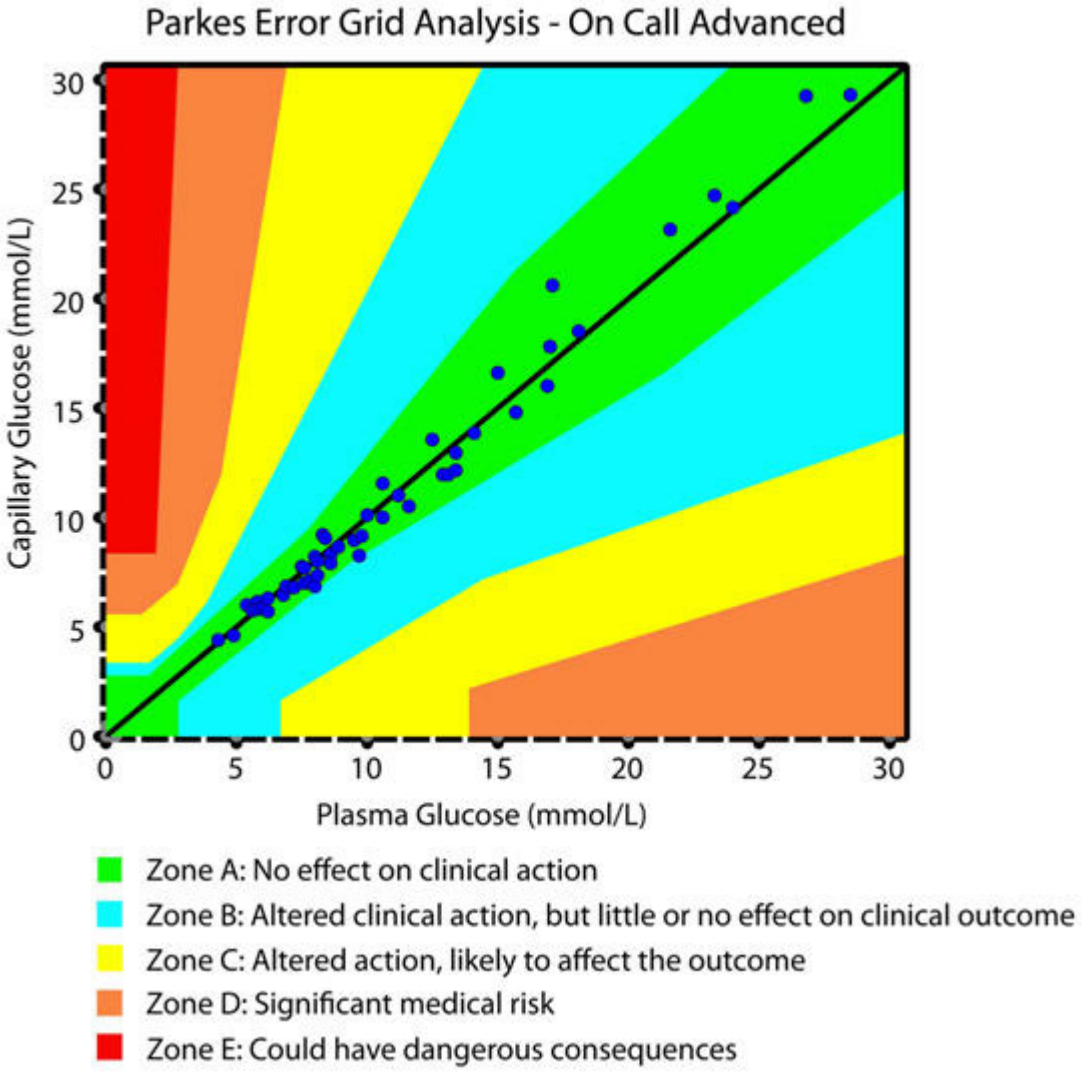
Figure 1b. Comparison of venous plasma with venous whole blood glucose (On Call Advanced)



Error grid analysis

Parkes error grid analysis is used to detect clinically significant errors in glucose measurement, when comparing capillary with laboratory plasma venous glucose results.[9] Using this visual method of analysis, method comparisons are intuitively easy to understand. The information in Figure 1a has been redrawn in Figure 2 as a Parkes error grid with a key showing how to interpret the zones contained within the grid. Error grid analysis of the different meters currently available in New Zealand is available on www.pharmac.govt.nz/usingmedicine and shows that all locally available meters perform to a satisfactory standard.

Figure 2. Comparison of simultaneously collected venous plasma with capillary glucose results



Clinical implications of limitations in meter accuracy and precision, in other settings

In other settings, for example in the ICU, factors related to patient pathology such as hypoxia, hypoperfusion and extremes of haematocrit may lead to additional sources of error.[5] Also, increased viscosity, which is common in severely dehydrated patients, may impair capillary strip filling and give an erroneous result with some older systems.[10] It is therefore of passing interest to note that one reason why very tight glycaemic control in some ICU settings may result in adverse clinical outcomes,[11] relates to the difficulty of safely achieving tight control with intravenous insulin, when insulin dose is determined using capillary meter glucose values.[12]

Another situation where meter inaccuracy and imprecision can amplify errors in glucose measurement is with CGM (continuous glucose monitoring) systems,[13] as the CGM biosensor requires ongoing calibration using finger stick readings from a conventional meter.

Minimising sources of error in the real world setting

The above discussion on meter performance is based predominantly on tests undertaken under controlled conditions by trained technicians. In the real world setting,[14] end-user (patient or health practitioner) error, including problems with interpretation of glucose results, may have negative clinical consequences. In theory, errors can be minimised by reviewing patients' meter technique on a regular basis,[1] and also by undertaking quality control checks using commercial control solutions supplied by meter manufacturers for use with their specific meter. In practice, a combination of cost of these control solutions and their short shelf life after opening, limits their use to selected service providers such as hospital based point of care co-ordinators.

End-user error is minimised by the multiple safety features embedded into currently available meter systems but many potential sources of error remain. The clinical cases below describe extreme examples of real world problems that can occur with current meter systems. We hope that these illustrations will help improve clinicians' awareness of potential problems, as well as help them with troubleshooting. Although major errors in glucose measurement are rare, minor errors are not uncommon. Further descriptions of common potential sources of error are given in Table 2.

Case studies

Case 1—Meter not coded for current batch of test strips

A patient was diagnosed with Type 1 diabetes 2 years previously and undertook frequent glucose testing. He had a year-long discrepancy between his home glucose results, which were consistently <10mmol/L and his laboratory glucose results which were >10mmol/L. During the same year his HbA1c increased from 8.5% to 11%. Thus there was a discrepancy between his finger stick and laboratory values. On reviewing meter technique, he was found not to have recoded (recalibrated) his meter since the time of diagnosis. The combination of an old strip batch code and currently available test strips produced meter glucose results that were much lower than their real value, leading to significant under-dosing of insulin. The patient was given structured meter education at the time of diagnosis but does not recall receiving instructions about calibration and did not recall any update on meter use at any subsequent appointment.

Comment: This problem is common.[15,16] Another common problem is inadvertent use of time expired strips,[2] which is less likely to occur in systems that use a strip calibration code which also signals that the strip batch is past its expiry date . Regular review of meter technique is recommended, but it may be difficult to achieve in our resource constrained environment.

Table 2. Commoner potential sources of error in glucose meter measurement in the ambulatory setting

Description of source of error	Comment
Simultaneously collected capillary and venous plasma samples show slightly discrepant results	Capillary meter results should read within ± 20% of a laboratory plasma venous sample, 95% of the time. A higher discrepancy than this may point to an additional source of error (see below)
Meter not calibrated (using the calibration chip) to read the current batch of test strips	A common potential source of error – up to 25% of patients may be using an incorrectly calibrated meter/strip system
Contamination of test finger surface	Glucose rich foodstuffs on fingers may elevate the measured glucose value. Inadequate hand drying after washing may produce a dilutional error
Time expired and/or 'spoiled' strips	Strip performance is likely to be suboptimal, once past the strip expiry date. Performance may also be affected by prolonged exposure to adverse environmental conditions e.g. heat, humidity. Some (but not all) strip/meter systems will signal an error in these situations
Pathophysiological factors present in the patient, which may affect glucose meter / strip accuracy	More common in the inpatient (e.g. ICU) rather than ambulatory setting, but occasional ambulatory patients may experience extremes of haematocrit (e.g. anaemia) hypoxia (including acrocyanosis) and hyperviscosity syndromes, sufficient to affect the accuracy of glucose readings
Interfering substances present in the patient's blood	Icodextrin (found in dialysis fluid) can produce marked pseudohyperglycaemia. Many other compounds may produce interference, including aspirin, high dose vitamin C and paracetamol. These substances have a much smaller effect on glucose values compared to icodextrin. Interference is not always predictable and is in part dependent on the interaction between the interfering substance and the specific meter and strip system used for testing
Use of meter/strip systems in extreme environmental conditions	Meters and strips tend to perform suboptimally under 'extreme' conditions. This includes the high atmospheric pressure used in hyperbaric chambers

Case 2—Dilutional error

An adolescent on insulin had a history of recurrent diabetic ketoacidosis. She had a 'contract' with her parents to show them her latest glucose value recorded on her meter. Her parents reported observing satisfactory glucose results. Computer download of her meter's memory demonstrated clusters of tests undertaken over several minutes. A typical series of glucose results was: 21mmol/L at initial testing followed by 18, 10 and 6mmol/L over the next 5 minutes.

Comment: It is physiologically impossible to drop glucose levels by this magnitude over 5 minutes. It was assumed she was manipulating results by undertaking self-

dilution of samples, so that the glucose on the meter display read 6mmol/L, rather than 21mmol/L. Downloading glucose results from memory meters and comparing this with self recorded glucose results often highlights discrepancies in self reported results.[17] The concentration of salivary glucose is much lower than that of blood,[18] thus 'licking fingers clean' prior to testing may also cause a dilutional error, as can hand washing followed by incomplete hand drying. These errors are usually unintentional, but can on occasions be intentional.

Case 3—Change of meter from one calibrated to whole blood glucose, to a system calibrated to plasma glucose

A patient with Type 1 diabetes had tight glucose control (HbA1c 6.4%), frequent minor hypoglycaemia and hypoglycaemic unawareness. He updated his meter system but was unaware that his old meter was calibrated to read as whole blood yet his new meter read as venous plasma equivalent. Glucose values from the new meter therefore 'read higher' than those from the old meter. The patient concluded that glycaemic control had deteriorated and increased his insulin. He then had a hypoglycaemic fall and sustained a fracture.

Comment: All subsidised meters in New Zealand read glucose as plasma and this calibration related scenario is therefore now uncommon. However there are some countries that still use meters calibrated to whole blood,[2] thus patients with diabetes who move to New Zealand may need additional education when changing meters. This case also highlights the fact that patients become familiar over time with how their own meter functions and reads and they incorporate meter performance characteristics into their everyday self care.

Case 4—Interfering substances

A 53-year-old with insulin treated diabetes was commenced on peritoneal dialysis. He experienced unexplained severe hypoglycaemic symptoms despite apparently normal or elevated glucose readings using the Accuchek Perfoma meter. His high mean glucose value from the Performa meter contrasted with his normal HbA1c value of 5.4%. Paired glucose meter tests were then done using both the Performa meter (strips use a glucose dehydrogenase system) and Glucocard meter (strips use a glucose oxidase system).

A Performa glucose reading of around 8.0mmol/L corresponded to 2.0mmol/L using the Glucocard. The attending clinical team was advised by the local laboratory that systemic absorption of 7.5% icodextrin from the peritoneal dialysis fluid was a source of interference for glucose dehydrogenase based strip systems (which include the Performa, Freestyle Lite, On Call Advanced and Xceed systems, but not the CareSens system). Thus interference from icodextrin resulted in artefactually high glucose levels.[5,13] This was rectified by changing the patient to a glucose oxidase based meter/strip system.

Comment: Many other substances, including high dose ascorbic acid and aspirin,[5,13] may also interfere with glucose measurement (see Table 2) but, in contrast to icodextrin, they usually produce only a small change in measured glucose value.

Case 5—Meter reading glucose values as mg/dL

A teenager with Type 1 diabetes switched meters and inadvertently set his new meter to the mg/dL setting (i.e. to the glucose units used in the USA and several other countries) rather than to mmol/L. The conversion factor between the two units is 18:1. The patient was unclear how to interpret results, but worked on the assumption that 100mg/L equated to 10mmol/L.

He therefore titrated his insulin dose to achieve results between 40mg/L and 100mg/L (i.e. 2.2 to 5.6mmol/L), assuming erroneously that this was equivalent to 4 to 10mmol/L. Over the next four months, the patient's HbA1c dropped from 8.7% to 5.9%. He experienced frequent hypoglycaemia and excessive weight gain and developed hypoglycaemic unawareness. Fortunately all these negative clinical developments reversed when the error was identified and corrected.

Comment: Whilst this patient's persistent misinterpretation of results was unusual, we have witnessed patients making similar errors for short periods of time. The Care Sens meters are able to be set to read glucose units as either mg/dL or mmol/L. This may be advantageous for occasional patients who move between countries and health systems that utilise different units.

Conclusions

Meter analytical performance and ease of use has improved markedly over recent years. Safety features in the meter and strip systems may result in potentially erroneous values being 'disallowed', for example by giving an error message. Also, there are now far fewer potential sources of errors in measurement, but errors in measurement and in interpretation of results can nevertheless occur. An understanding of glucose physiology and meter performance should help minimise meter related errors and help with trouble shooting.

Most published data about meter performance is based on assessments undertaken in controlled environments. The error contribution made by end users (i.e. patients and health care practitioners), in real world settings is acknowledged to be large. There are however few systematic studies of the reasons for and magnitude of this source of error. Patients and their health practitioners therefore need to remain vigilant about the possibility of meter error. Undertaking occasional comparisons between simultaneous laboratory and finger prick samples measured on the patient's own meter system and undertaking regular reviews of meter technique remain important tools for minimising errors.

Clinicians want their meter derived glucose results to show close agreement with a plasma laboratory value. There are however challenges in achieving this, which relate in part to intrinsic physiological differences between these two specimens. Although current meter systems are accurate, they lack precision and only 95% of results might fall within 20% of the reference plasma laboratory value. Clinicians need to be aware of this fact, especially in situations such as diabetes in pregnancy and insulin pump therapy, where the patients and their health care team are aiming for tight glucose control. In practice, patients who use the same, familiar meter system over a prolonged period seem to be the least troubled by issues related to meter accuracy and imprecision. This may in part be because regular use of the same meter system yields

consistent readings in similar situations. From the health practitioner's perspective, an understanding of the differences between currently available funded meter systems should enable practitioners to select meters that best fulfil their patients' and their practice's needs. Encouraging staff and patients within your practice to become very familiar with one or two meter systems allows for an in depth understanding of the behaviour of that particular meter system and its related software for downloading of meter results, in the real world setting.

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Prasugrel, Maori, and personalised medicine in New Zealand

Patrick Gladding, Harvey White, Mark Webster

The response to thienopyridine antiplatelet therapy is heterogeneous and is in part explained by clinical and genetic factors. A recent meta-analysis has demonstrated the clinical significance of a genetic polymorphism in the cytochrome P450 2C19 gene. Carriers of this polymorphism have a higher incidence of stent thrombosis and cardiovascular death, whilst on the thienipyridine clopidogrel. The polymorphism and rarer variants display higher carrier frequencies in ethnic groups with disproportionate cardiovascular mortality, such as Māori. Knowledge of an individual's genetic status may assist in optimising antiplatelet therapy, thereby reducing the cost of adverse events, expenditure on new medicines, and the ethnic disparities seen in healthcare outcomes. A demonstration of the cost-effectiveness of genetic testing, on a population basis, and a proven alternative, personalised strategy is required before the adoption of this technology can be advocated.

Pharmacogenetics has long held the promise of individualising pharmacological therapy using genetic biomarkers. Within the last year pharmacogenetic tests predicting adverse reactions to the antiepileptic drug carbamazepine and HIV medication abacavir have entered routine clinical practice.[1,2] With ever growing healthcare costs, new pharmaceuticals providing only a modest incremental benefit over current therapies, and the world-wide economic downturn there has never been a greater need to match treatment to those who have the most to gain from it.[3]

Cardiovascular medicine is well positioned to benefit from rapid advances in this field. Potential genomic biomarkers for drugs in clinical use include a genetic test for warfarin to predict the treatment maintenance dose,[4] for simvastatin to predict the likelihood of myopathy/myositis,[5] for bucindolol to predict potential efficacy in heart failure[6] and for clopidogrel to predict increased recurrent thrombotic events, including stent thrombosis.[7–9]

Elucidation of the genetic markers predicting response to clopidogrel, the second most-prescribed drug in the world, is of particular importance as a reduced antiplatelet effect with clopidogrel is associated with adverse clinical outcomes including cardiovascular death, myocardial infarction, stroke and additional healthcare costs.

Clopidogrel is attractive for pharmacogenetic study as it is a pro-drug that requires conversion to an active derivative, catalyzed by cytochrome P450 (CYPs). The functional polymorphisms within the CYP genes have been relatively well characterised, with those of interest including *3A4* and *3A5,* 2*C19*, 2*C9*, 2B6 and *1A2* enzymes.[10–12]

Several studies have shown that the loss of function allele *CYP2C19*2* is associated with adverse vascular outcomes in those taking clopidogrel. While other rarer variants such as the *CYP2C19 *3* and **4* alleles are also associated with reduced function of

the enzyme, the *CYP2C19*17* variant is associated with ultrarapid enzyme activity.[13] In contrast the third generation thienopyridine prasugrel is not as dependent on the CYP2C19 and CYP2C9 enzymes for biotransformation into its active metabolite.[11]

Genotypes that code for a phenotypic poor response to clopidogrel are more frequently found in some ethnic groups than others. The *CYP2C19*2* loss of function variant occurs in 13% of Caucasians, 18% of African Americans and 29% of East Asians. It also occurs in higher frequency in Māori (24%) than NZ Europeans (15%).[14]

*CYP2C19*3* is four to five times more frequent in Polynesians and Māori (1.8%) than Europeans (0.4%).[14,15] This variant codes for a truncated protein and, together with the *2 allele, accounts for 99% of poor metabolisers in Asian populations.[16]

These ethnic disparities have two potential important clinical consequences. Firstly, these differences should be considered when interpreting trial data. For example, the largest trial evaluating clopidogrel and its effect on mortality was undertaken in 46,000 Chinese patients presenting with ST elevation myocardial infarction (COMMIT-CCS trial).[17] While the response to clopidogrel found in this study might reasonably be extrapolated to a Māori and Pacific Island population, the magnitude of benefit observed may have been greater in other ethnic groups with a lower prevalence of CYP2C19*2.

Secondly, using pharmacogenetics to individually tailor treatment may improve outcomes to a greater extent in some ethnic groups than others. Taking this hypothesis one step further, it is possible that therapy guided by genomics may help reduce the disparity in treatment outcome in populations such as Māori where cardiovascular disease is highly prevalent and clinical outcomes on treatment are poor.

Pre-determining poor-responders to clopidogrel may aid in optimising antiplatelet therapy in these patients by either giving a higher dose of clopidogrel or using alternative therapy such as prasugrel. While prospective clinical trials are necessary to assess this theoretically-attractive approach, pharmacogenetic data from TRITON (Trial to Assess Improvement in Therapeutic Outcomes by Optimizing Platelet Inhibition with Prasugrel–Thrombolysis in Myocardial Infarction), a comparison of prasugrel with clopidogrel in patients undergoing percutaneous coronary intervention, give some insights.

Patients with the reduced-response allele CYP2C19*2 on clopidogrel treatment had a higher incidence of vascular events, stent thrombosis and death, whereas those with the same variant on prasugrel had no increased events and, interestingly, no increase in bleeding.[18,19] Further large population outcome studies have confirmed the association between the *2 allele and adverse outcomes in those taking clopidogrel.[7,9,20,21]

Although this individualised genetic approach to therapeutics may improve the patient's responsive to treatment, it does not address the lifestyle changes that need to be implemented to prevent disease, issues such as reduced access to healthcare resources, and socio-economic, educational or cultural influences on treatment choices. Further understanding of the molecular basis of disease may well bring us effective tailored preventative therapies targeted at currently unmodifiable risk

factors. We can hope that these are affordable to the healthcare system, and accessible to disadvantaged ethnic groups.

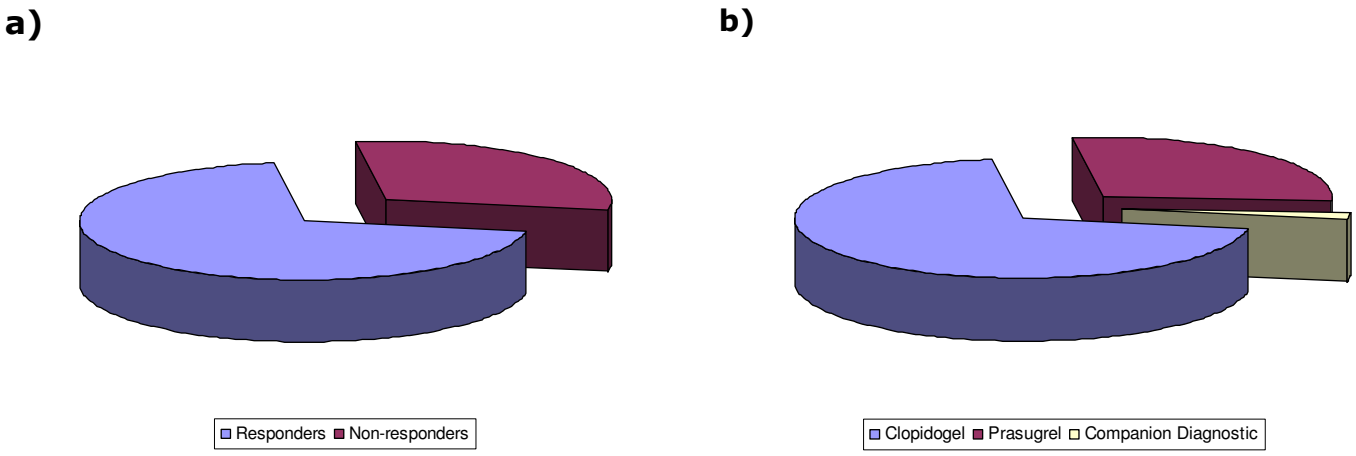
Genotyping prior to drug administration may be of particular importance for drugs like clopidogrel, which is often started in the acute setting with the need for a clinical benefit from a rapid and effective antiplatelet effect. It may also help predict the clinical importance of drug interactions, such as with omeprazole[22,23] and other CYP2C19 inhibitors. Although phenotyping, using platelet function testing, provides a more integrated assessment of drug response, testing can only be performed after a drug is administered. There is some evidence that combining genotyping and phenotyping may be more effective in predicting clinical outcomes than either alone.[24]

The US Food and Drug Administration (FDA) has recently updated the package insert for clopidogrel, to include information on pharmacogenetic testing. Testing is not officially advocated and the cost utility of testing, in terms of preventing adverse events, has not yet been proven. Recent analysis has shown that a simple three SNP test for warfarin is not cost effective under current average test prices.[25] However the costs of genotyping are reducing exponentially and the era of the $1000 genome is not far away.

A shift from treating everyone with a particular condition to individualizing treatment based on genomic or proteomic biomarkers promises to improve safety, efficacy and allocate expensive treatments to those who have the most to gain. The concept of rationing treatment in the current economic climate appears appealing but reduced expenditure will only be achieved if the incremental cost of the diagnostic test can be recouped (Figure 1).[26]

With appropriate safeguards in place, a once in a lifetime genetic test could soon be part of every patient's medical record. Busy physicians may need to integrate this "companion diagnostic" information into their day-to-day clinical decision-making, when they use the information from clinical trials, patient comorbidities and potential drug interactions to apply evidence-based practice in the individual patient.

Figure 1. Microeconomics of Personalised Medicine



a) Displays current expenditure on a pharmaceutical agent, with substantial portion of spending wasted on treating non-responders. **b)** Future expenditure based on personalised approach where therapeutic diagnostic ('theranostic') constitutes a fraction of total expenditure. The objective of the targeted approach is to maximise benefit of next generation pharmaceutical and minimise potential harm. A cost-effectiveness analysis is required prior to adoption of the new model, taking into account savings from prevented events.[26] (Adapted from Personalized Medicine: The Emerging Pharmacogenomics Revolution. A 2005 monograph by Price Waterhouse Coopers.)

Competing interests: PG has founded a company offering genetic tests to consumers/doctors.

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THE NEW ZEALAND MEDICAL JOURNAL
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Podiatry services for patients with arthritis: an unmet need

Keith Rome, Jonathan Chapman, Anita E Williams, Peter Gow, Nicola Dalbeth

Abstract

Foot problems are extremely common in patients with rheumatoid arthritis (RA). There is ample evidence that foot pain, either alone or as a comorbidity, contributes significantly to disability. Despite the high prevalence of foot disease in RA, this problem is often trivialised or underappreciated. The inequity in foot health provision for patients with rheumatic disorders in New Zealand has recently been highlighted. Expertise in dealing with foot problems is often limited among healthcare professionals, and it has been argued that better integration of podiatric services into rheumatology services would be beneficial. The aim of this paper is to highlight the major issues related to foot care for patients with arthritis and provide key recommendations that should implemented to improve access to podiatric services in New Zealand.

A publication in the *Journal of Foot and Ankle Research* has highlighted the need for improved access to podiatry care for rheumatology patients in New Zealand.[1] In summary, the clinical audit demonstrates that there is an unmet need for professional and specialist foot care and concurs with findings from an audit in the UK that also identified the same unmet need.[2] Although these two studies were carried out in one locality, albeit different countries, it is perceived generally that the input of specialist podiatry into rheumatology services is, at best patchy.

The rheumatoid foot

Rheumatoid arthritis (RA) is the most common inflammatory arthritis. It is a chronic, immune-mediated inflammatory disease which can lead to significant joint damage and functional impairment.[3] Up to 90% of people with RA have foot involvement and the prevalence and impact of foot problems is strongly associated with disease severity and duration.[3] Medical management focuses mainly on controlling disease activity, providing symptom relief and maximising quality of life.

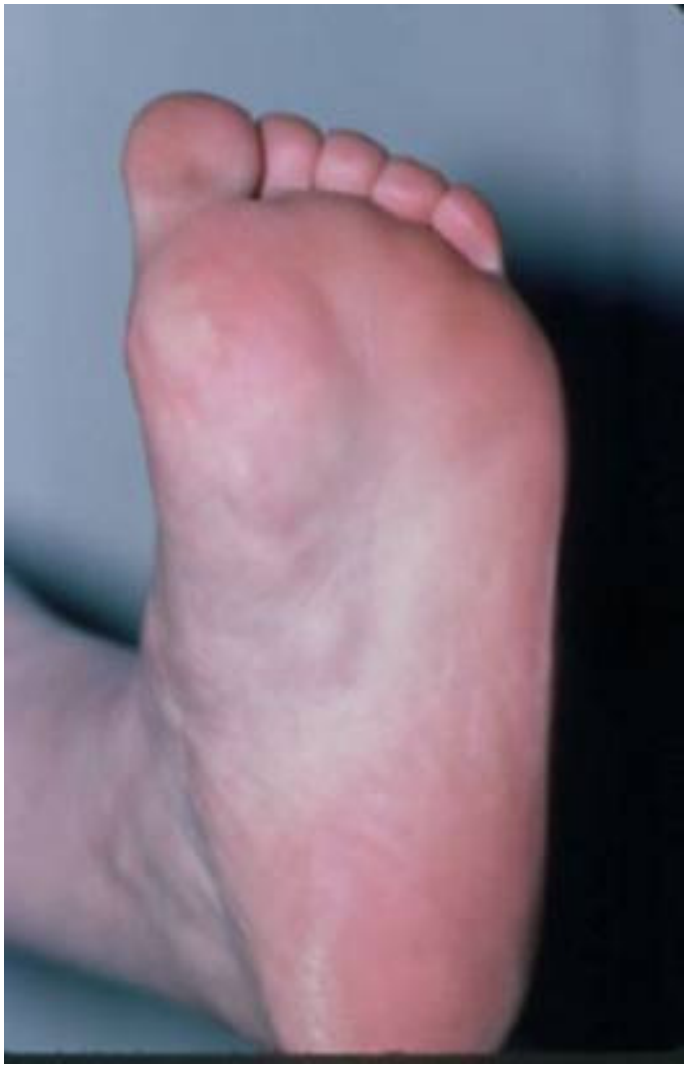
Although modern treatments have improved systemic disease control substantially in recent years, complete remission is still unusual. However, despite the medical management patients often continue to suffer the effects of joint damage with the foot being affected in the majority of cases.

People with RA often present at their consultation with complex needs, and it is easy for foot problems to be overlooked. Assessment of the feet is not as straightforward as more accessible parts of the body. Foot examination may be considered awkward by some practitioners as the footwear has to be removed. Where a foot assessment is performed, the management of foot problems is sometimes not well understood. The end result of this uncertainty and inconsistency is that foot problems are often neglected, and lack of integration of podiatry into the rheumatology team maintains it

as a *Cinderella service* and people with RA continue to suffer foot pain, limited ability and poor quality of life.[4]

It is of concern that foot problems are overlooked as we know that the majority of patients have foot involvement. Patients with inflammatory arthritis have an increased need for a range of basic foot care services. Long-standing inflammation leads to structural deformity and soft tissue lesions which in turn generate areas of pressure that result in callus and corn formation (Figure 1). There is evidence that early intervention for existing or potential foot problems can improve long term outcomes.[5] Baseline foot examinations can identify people with existing or imminent needs and provide a comparator for assessment. Regular assessments that document the rate of structural change can aid treatment decisions and improves outcomes.[4]

Figure 1. Rheumatoid foot with a severe bunion, lesser toe deformities and bursitis over the second and third metatarsal heads



Patients with RA who experience a sudden 'flare' in disease activity should have direct access to specialist advice, and be offered the option of an early review with appropriate multidisciplinary team members, including podiatrists. Similar reviews of needs should be undertaken during periods of disease remission. Podiatrists have a role to play in supporting patients with RA in managing aspects of their condition themselves, as well as in providing timely and relevant foot health specific advice and education.

Patients with RA can experience variations in disease activity (exacerbations and remissions) and may have acute needs (e.g. infection) superimposed on the overall disease process.[2] The foot contributes to difficulty with walking in about 75% of people with RA, and is the main or only cause of walking difficulty in 25%. In the foot, joint pain and stiffness is the most common initial presentation, but a range of other features, including tenosynovitis, nodule formation and tarsal tunnel syndrome may also present, reflecting widespread soft-tissue involvement. [3]

Podiatrists have a prominent role to play in symptom relief and improving quality of life because involvement of the feet, even to a mild degree, is a significant marker for impaired mobility, functional incapacity and negative psychosocial impact.[4] In the UK, NICE have published the guidelines on the treatment of people with RA.[6] These guidelines provide a clear information and direction to commissioners and providers on what is expected by NICE in terms of funding and service provision. NICE recommended that all people with RA and foot problems should have access to a podiatrist for assessment and periodic review of their foot health needs, and that foot orthoses and therapeutic footwear should be available for all people with RA if indicated.

Current podiatric services and rheumatoid arthritis

A scoping exercise led by the key author exploring regionalised access to podiatry services was carried out through the professional body, Podiatry New Zealand. The findings overall were a lack of consistency and integration with rheumatology services with aspects of podiatry being provided by a range of disciplines including consultants, medical trainees, general practitioners, nurses, orthotists, physiotherapists, and occupational therapists in addition to podiatrists when they are accessible. In comparison with diabetic foot care there is inequality.

The New Zealand Guidelines Group published a minimum standard of guidelines for the assessment and monitoring of the diabetic foot in New Zealand.[7] People with rheumatic diseases often present with complex needs, and it is easy for podiatric problems to be overlooked such as pain, functional activities and disability.

However, there is published evidence of unmet podiatric care needs for patients with RA;[8] evidence that single interventions such as orthotics and footwear are clinically effective;[9,10] and evidence of UK-wide under provision of foot care either in primary or secondary care settings.[11] Phase I and II data and the methodological considerations for a definitive phase III trial of podiatry-led care have recently been published.[12] Recent review papers report moderate-to-good evidence for the use of foot orthoses in patients with rheumatoid arthritis.[9,10,13]

Current evidence from a New Zealand perspective has recently been reported.[1]The goal of the study was to identify the nature of foot problems experienced by patients with RA attending the rheumatology outpatient clinics at Counties Manukau DHB and to ascertain the availability of a podiatry services for these patients. Foot and ankle assessment were based upon the recommendations from the Standards of Care for People with Musculoskeletal Foot Health Problems.[4]

100 patients (n=100) who fulfilled the American College of Rheumatology criteria for diagnosis of RA were recruited into the study.[14] Patients were excluded if they did not fulfil American College of Rheumatology criteria for RA and non-residents/visitors with only brief contact with Counties Manukau DHB (< 3 months) or who lived outside Counties Manukau DHB.

The results demonstrated over 85% of RA patients suffered from foot lesions, ranging from callus, corns and nail problems. 86% of patients had deformities of their lesser toes. The majority of foot lesions (64%) were observed on the forefoot around the metatarsal heads. Bilateral hallux valgus (bunions) was observed in 64% of patients.

The current study highlighted that patients with RA at Counties Manukau DHB have an increased need for a range of podiatric interventions and preventions. The results also highlighted high number of patients with foot pain and disability associated with foot problems that includes callus, corns and lesser toe deformities with RA. Recommendations of the study included that baseline foot examination can identify people with existing or imminent needs and provide a comparator for assessment. Regular assessments that document the rate of structural change can aid treatment decisions and improves outcomes. An annual musculoskeletal, vascular and neurological assessment, which includes an assessment of the lower limbs and feet, will help identify problems early.

Recommendations to improve access to podiatry services for New Zealand with rheumatoid arthritis

Expertise in dealing with foot problems is often limited among rheumatologists and primary care practitioners, and it has been argued that better integration of podiatric services into rheumatology care would be beneficial. Last year, a foot and ankle symposium was held last year prior to the New Zealand Rheumatology Association conference which speakers from New Zealand and the UK presented the problems associated with the musculoskeletal foot and ankle. The key speakers included orthopaedic surgeons, rheumatologists, physiotherapists, podiatrists and specialist nurses. The conference was very well attended by a range of health care professionals and the need to develop and implement a rheumatology focussed foot and ankle interest group was agreed by all delegates.

The recent work by our group[1] further emphasises that this is an unmet need for patients with arthritis in New Zealand, and that incorporation into the rheumatology multidisciplinary team is required to improve clinical outcome of these patients. It was also clear from the discussions that ensued that what is needed is an integrated approach to the management of foot problems with podiatrists being the key practitioner in co-ordinating assessment and management of the foot and its related problems.

Future directions should include education and training should be provided to primary care staff and foot health care providers to enable them to understand the systemic consequences of musculoskeletal disorders on the feet. Training should begin with undergraduate education and extend to post-registration education and continuing professional development.[4,15]

Clear guidelines, protocols and referral pathways should be developed locally that include agreed criteria for suitability for self-management, eligibility for access to foot health services from both primary and secondary care referrals, and also for self-referral.

Referral pathways in to podiatric services should make clear:

- Who has access to their services (e.g. geographic location, referring agencies and organisations);
- The signs and symptoms that indicate referral;
- Red flags that indicate priority referral.[4] Examples would include: joint pain and synovitis—within the first 18 months from onset of symptoms. Restricted mobility and activity limitation associated with the above or established foot disease. Inability to care for own feet; Risk factors for ulceration—deformity, vasculitis poor footwear, medication affecting tissue viability. Signs of infection (bacterial, fungal or viral) or ulceration (particularly those patients on biologics).

Podiatrists should be fully integrated as a member of a multidisciplinary team. While some musculoskeletal foot problems can be managed in isolation, complex or systemic conditions such as RA require a multidisciplinary approach to management.[16,17]

Foot disorders can affect many aspects of a person's life, especially when associated with systemic disease, and care may need to include input from many different professionals from health and social care. Surgery may also be considered when severe symptoms persist and do not respond to conservative treatment. People with progressive foot problems may require specialist surgical opinion with the facility for immediate surgical referrals e.g. those with nerve compression or tendon ruptures.[4]

In summary, we hope the recommendations suggested in this view-point will act as a catalyst for all stakeholders—service users, providers, commissioners and policymakers—to work together to implement access to their local podiatric services and crucially, to strive for integration of specialist podiatrists into the multidisciplinary team.

Competing interests: None known.

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Fatty infiltration of the liver in a case of hypobetalipoproteinaemia with a novel mutation in the *APOB* gene

Chris Florkowski, John Hedley, Vivienne Bickley, Amanda J Hooper, John R Burnett, Peter George

Case report

A 63-year-old man was referred with abnormal liver function tests. ALT elevation had been documented for approximately 10 years and was 103 U/L with GGT of 103 U/L at the time of review. General health was good with no background of operations, transfusions, acupuncture or tattoos. He was a storeman by trade, a non smoker and alcohol consumption was modest. There was no family history of liver disease, although he was known to have low plasma cholesterol with his lowest documented level being 1.8 mmol/L with triglycerides 0.9 mmol/L, HDL-cholesterol 0.8 mmol/L, and a calculated LDL-cholesterol of 0.7 mmol/L.

Examination showed an overweight man, with body mass index of 28.3 kg/m^2 and blood pressure of 115/60 mmHg. There was no hepatosplenomegaly nor ascites and no stigmata of chronic liver disease. The rest of the examination was essentially normal.

Iron studies were normal apart from raised ferritin at 996 µg/L (reference interval 20-500), though *HFE* genotyping showed no mutations. He was negative for hepatitis A, B and C serologies and also smooth muscle antibodies. Plasma vitamin E was low at 16 µmol/L (reference interval 23-70). Abdominal ultrasonography confirmed fatty infiltration of the liver.

Western blotting (Figure 1) showed an abnormal apoB variant, approximately 80% of full-length apoB-100. The region of the *APOB* gene predicted to harbour the mutation was sequenced. This confirmed that the patient was heterozygous for a novel *APOB* mutation c.10312delA (p.Met3438X), predicted to cause a truncated apoB consisting of the amino-terminal 75.2% (apoB-75.2, 3410 amino acids out of 4536 in mature apoB).

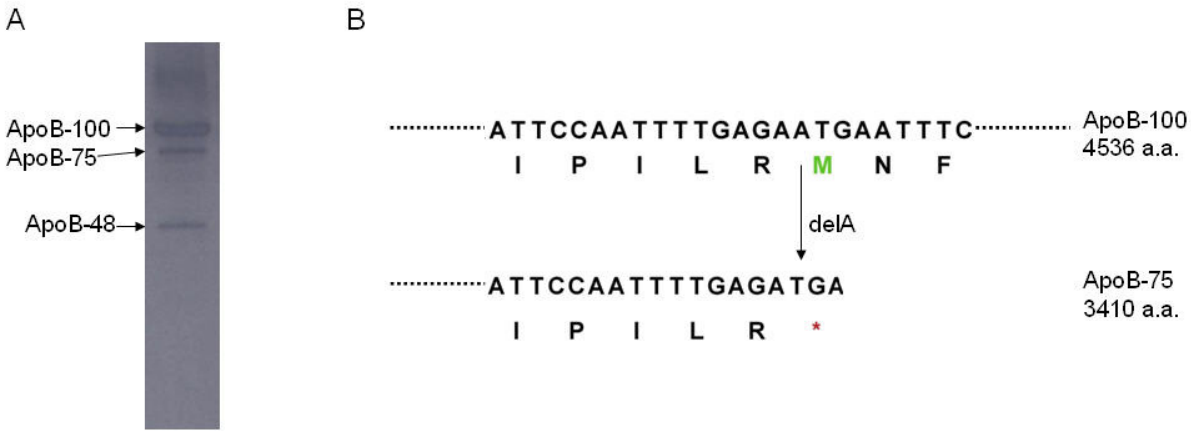
The patient thus had heterozygous familial hypobetalipoproteinaemia (FHBL), which is known to be associated with fatty infiltration of the liver.[1] He was placed on a fat restricted diet and given vitamin E supplementation.

Discussion

Non-alcoholic fatty liver disease (NAFLD) is one of the most common liver diseases encountered in the developed world and refers to a spectrum of hepatic pathology that resembles alcoholic liver disease, but appears in individuals who have low or negligible alcohol consumption. It was initially recognized in morbidly obese females

with Type 2 diabetes, in whom the hepatic histology was consistent with alcoholic hepatitis, but there was no history of alcohol use.[2]

Figure 1



Key: Panel A shows separation of apoB-100, apoB-48 and the truncated apoB-75 variant in the index patient. Plasma proteins were separated by electrophoresis for 1.5 h at 150 V using a NuPAGE 3-8% Tris-acetate gel (Invitrogen). Proteins were transferred to nitrocellulose, and, after blocking, incubated with a monoclonal anti-apoB antibody (1D1, a kind gift from Dr Ross Milne, University of Ottawa Heart Institute) followed by sheep anti-mouse HRP-conjugated antibody (Millipore), and chemiluminescent detection (GE Healthcare). The region of the *APOB* gene predicted to harbour the mutation was sequenced. Panel B shows a representation of the wild-type and mutant alleles present in this patient, with deletion of A from native sequence (top line) resulting in the creation of TGA (bottom line), a premature stop codon.

More recently it has become apparent that NAFLD is a spectrum of disease and is probably the commonest cause of abnormal liver function tests in general practice.[3] Obesity, with insulin resistance is usually associated with the development of NAFLD and patients commonly exhibit hypertriglyceridaemia with low HDL cholesterol.[4] Fat accumulates in the liver when the rate of delivery of fatty acids to hepatocytes exceeds the metabolic capacity to process them.[3] Fatty acids are delivered to the liver from peripheral adipose tissue, and also from local synthesis in the liver as a result of either protein or carbohydrate excess.[3] Fatty acid disposal occurs through either mitochondrial beta-oxidation to ATP and ketone bodies, or secretion into the blood as triglycerides in very low-density lipoprotein (VLDL).

Disturbances in these processes can be inherited or acquired, resulting in the accumulation of triglycerides in the liver.[3] FHBL is a Mendelian co-dominant condition[5], usually caused by truncation-producing mutations in the *APOB* gene. Truncations shorter than apoB-27 are not expressed in lipoproteins while those less than apoB-75 show little expression in LDL. The postulated mechanism of fat accumulation in the liver is decreased VLDL-triglyceride export, resulting from the defective assembly of lipoproteins containing truncated apoB[1,7].

It has been shown that the smaller the apoB, the greater the decrease in hepatic secretion.[8] Other series have shown that approximately 50% of patients affected with this disorder have elevated transaminases, suggestive of fatty infiltration of the liver.[6] Low vitamin E was found in this case, as expected in one with very low LDL cholesterol and supplementation was given, although there are some data to suggest that tissue levels are not necessarily deficient.[9]

The association of NAFLD with very low cholesterol should prompt investigation for underlying FHBL, a recognised cause of NAFLD, as in our case, in which a novel mutation of the *APOB* gene was discovered.

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Testicular seminoma metastasis to pancreas: a rare cause of obstructive jaundice

Mohit Girotra, Niraj Jani, Javaid M Khan, Viplove Senadhi, Sudhir K Dutta

Testicular cancer represents the most common malignancy in men from age 15–35 years. The North American standard classification[1] divides testicular cancers into Germ cell tumours (GCT) and Non-Germ cell tumours. The lymphatic spread[2] of GCT usually involves the retroperitoneal lymph nodes.[3] However, this spread to retroperitoneum rarely involves the upper gastrointestinal tract.

Case report:

A 43-year-old African-American man presents to the ER with right upper quadrant abdominal pain and pruritus of 2-week duration. He also had dark stools and increasing constipation. On exam, notable findings included icteric sclera, right upper quadrant and epigastric tenderness. Laboratory studies revealed a total bilrubin of 6.3mg/dl, AST/ALT of 52/143 units/L, and ALP of 408 units/L. CT abdomen showed a large soft tissue mass at the 2nd and 3rd portions of the duodenum with intra and extra-hepatic biliary dilatation

At endoscopy, the entire second portion of the duodenum was found to be ulcerated, friable, and the major papilla could not be identified (Figure 1). EUS revealed a 5-cm hypoechoic lesion extending from the pancreatic head to the duodenum with a grossly dilated bile duct upstream (Figure 2). ERCP was attempted for biliary decompression followed by EUS-FNA for staging and diagnosis. The patient had a percutaneous transhepatic catheter placed for biliary decompression. Endoscopic biopsy and cytology specimens of pancreatic mass revealed a poorly differentiated neoplasm with features resembling testicular seminoma (Figure 3). Immunostains for hPLAP (human Placental Alkaline Phosphatase) positivity confirmed metastatic seminoma.

As his bilirubin decreased, he began to feel better and was then started on chemotherapy. He was readmitted 2 months later for massive upper GI bleed and EGD showed a bleeding duodenal mass near the ampulla. Haemostasis was achieved with a heat probe application and an epinephrine injection. Tagged RBC scan showed no specific bleeding site to embolise. He underwent a right hepatic artery ligation and a pylorus sparing Whipple procedure (R0 resection). The patient was discharged home after 3 weeks and continued on adjuvant therapy with chemo and radiation.

The patient had initially presented 1 year back with right lower quadrant abdominal pain radiating to his groin with a swollen and tender right testicle (6cm). He underwent a right inguinal orchiectomy and was found to have T3N0M0 testicular seminoma. Chemotherapy and radiation were recommended but the patient did not pursue.

Figure 1. Endoscopic appearance of pancreatic tumour in the 2nd and 3rd part of duodenum, loss of duodenal papilla

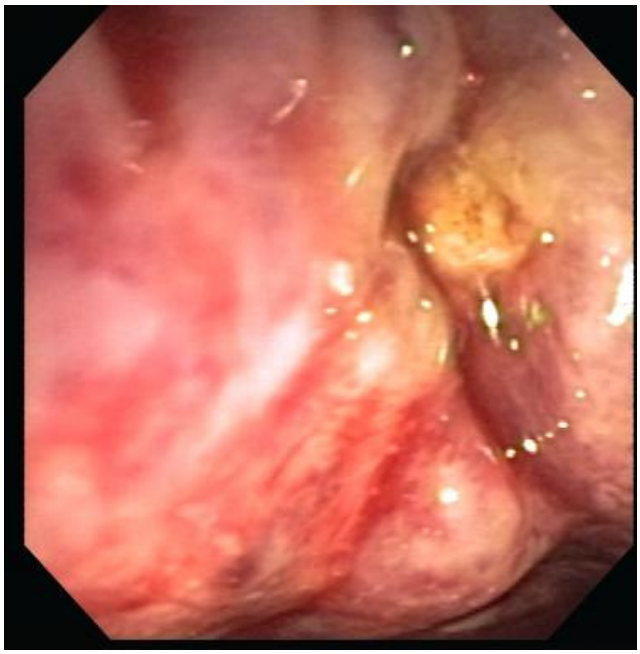


Figure 2. Endoscopic ultrasound appearance of pancreatic tumour

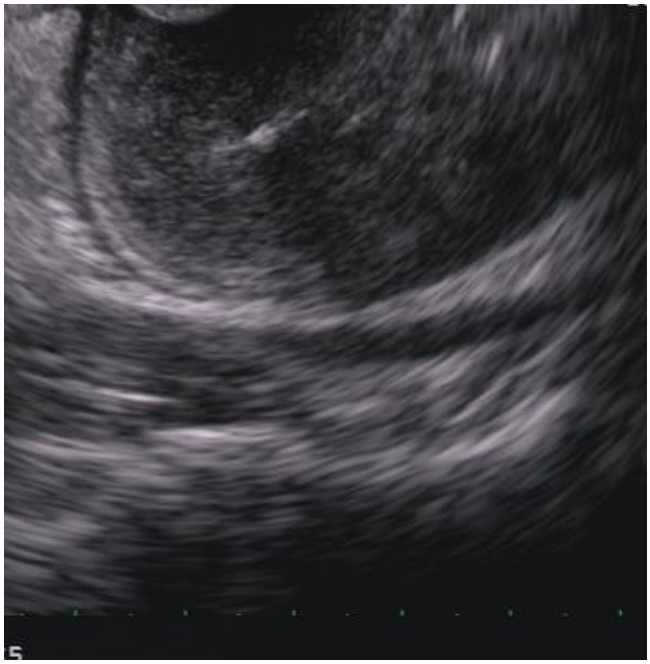
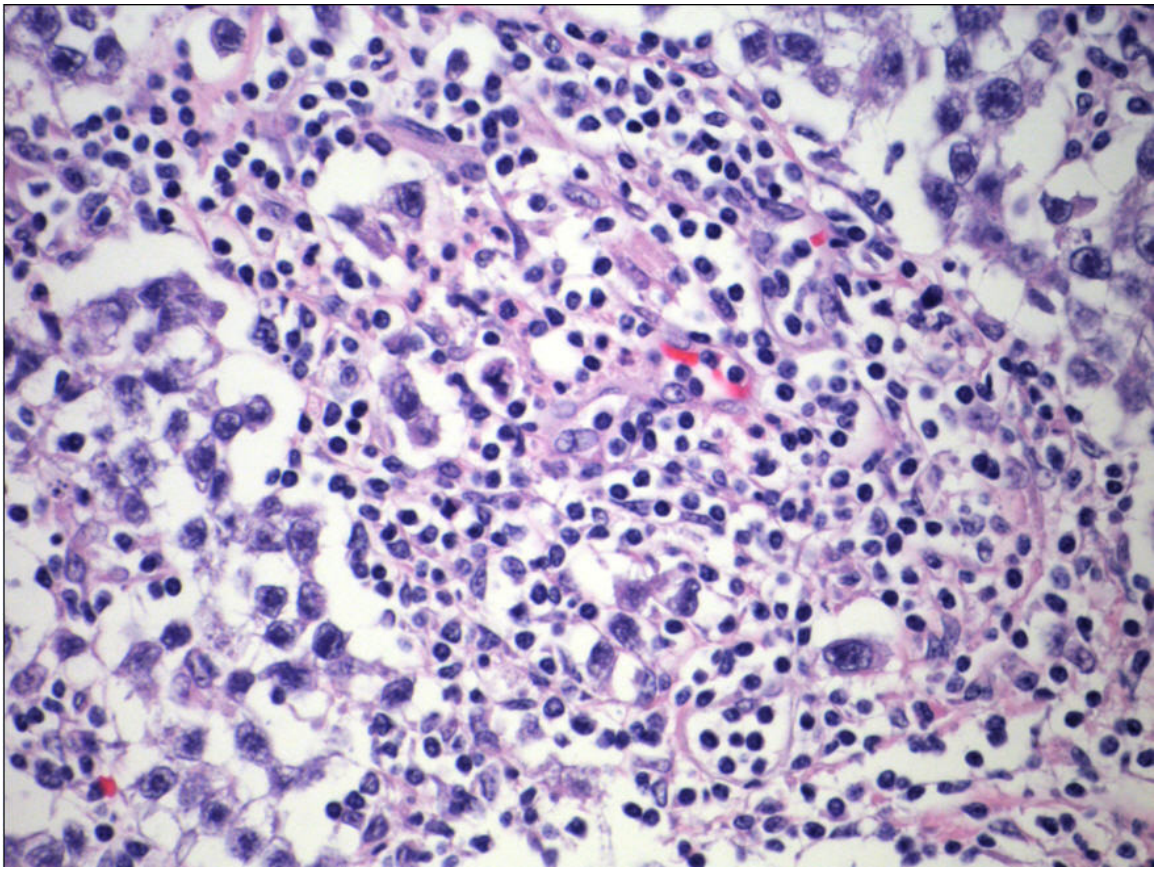


Figure 3. Pathology slide of pancreatic mass showing the features of testicular seminoma



Discussion

GCT metastasis to the upper gastrointestinal (GI) tract is uncommon (<5%). Non-seminomatous GCT is much more likely to spread to the GI tract than seminomas.[8] A case series from Memorial Kettering described 16 cases of metastatic pancreatic cancers, but none of them were testicular in origin.[9] Another postmortem case series did not document any purely seminomatous tumours metastasising to the upper GI tract[7].

Our patient was initially diagnosed with testicular cancer and underwent surgical orchiectomy, but refused any post-operative chemotherapy and radiation. The tumour was T3N0M0 and no evidence of metastases was noted at that time. He later presented with obstructive jaundice, and was found to have a large pancreatic head mass pressing into the duodenum. The pathology and immunostaining of the mass was consistent with patient's earlier diagnosis of testicular seminoma. He was started on chemotherapy with BEP (bleomycin, etoposide, and cisplatin) and responded well, with decrease in size of the mass on serial CT scans and also symptomatic relief from his jaundice. He later had GI bleed, which is a common complication of GI tumours, and underwent Right hepatic artery ligation followed by R0 Whipple's resection.

Later adjuvant therapy with chemo and radiation were continued and patient responded well.

Seminoma is a highly chemosensitive tumour and modern chemotherapeutic regimens (BEP) have shown high success rates.[10] Most cases of metastases have been documented in patients not receiving chemotherapy. Our case is unique in being the first to report pancreatic metastasis of testicular seminoma, and emphasises the importance of adjuvant therapy (chemo/radiation therapy) following the surgical resection of large testicular tumours to prevent future complications and metastases.

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THE NEW ZEALAND MEDICAL JOURNAL
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Calcified mass in right upper abdomen

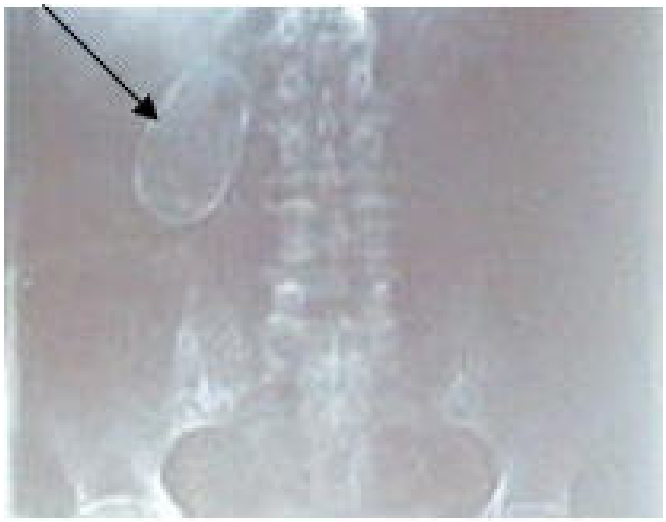
Cemil Kavalci, Atakan Sezer, Eylem Sezenler, Mütasım Sungun

Clinical

A 80-year-old she patient presented with nausea, vomiting and abdominal pain. Her past medical history was diabetes mellitus and hypertension.

On abdominal examination, Murphy's sign was positive. An abdominal radiograph was performed (Figure 1). The patient declined surgery, which is the usual treatment, and was treated with antibiotics.

Figure 1



What is the diagnosis?

Answer

The abdominal radiograph shows a *porcelain gallbladder* arrowed (Figure 1).

Discussion

Porcelain gallbladder (calcification of the gallbladder wall) is a rare condition with prevalence in cholecystectomy specimens ranging from 0.06 to 0.8%.[1] It is most commonly found in the sixth decade of life, with a higher prevalence in woman.[2] The aetiology of porcelain gallbladder is still unclear.[3]

In recent reviews the association between porcelain gallbladder and carcinoma is less than that previously suggested (5–7%).[4] However, a cholecystectomy may be performed on diagnosis because the risk of cancer is still increased.[4]

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THE NEW ZEALAND MEDICAL JOURNAL
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Anaemia and skeletal deformities

Kundan Kumar, Alakendu Ghosh

Clinical

A 22-year-old female presented to us with anaemia and gross skeletal deformities of all four limbs. Physical examination revealed pallor, icterus and hepatosplenomegaly, short stature and stunted growth. Plain radiographs of the chest and limbs were performed (Figures 1, 2, and 3).

Figure 1. Chest radiograph (PA view)

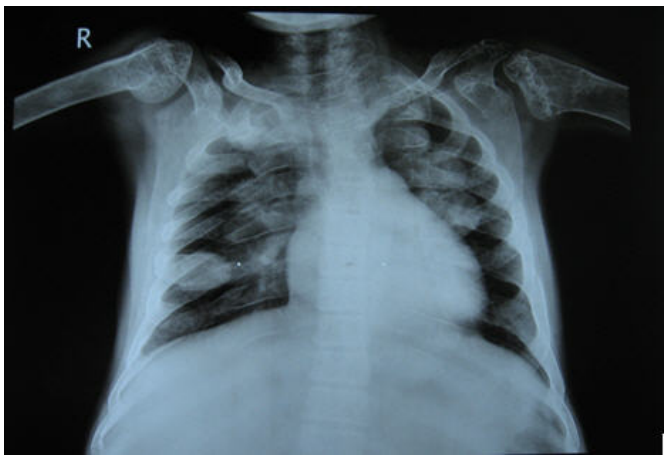


Figure 2. Radiograph of left forearm and hand

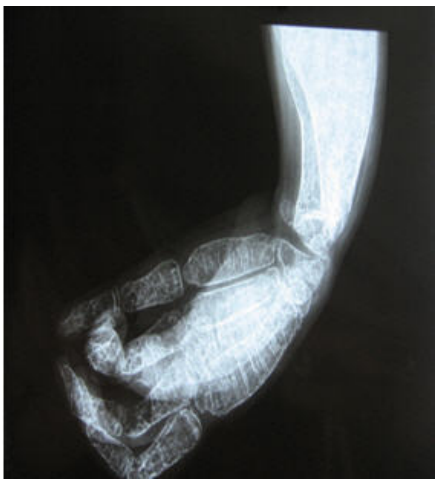
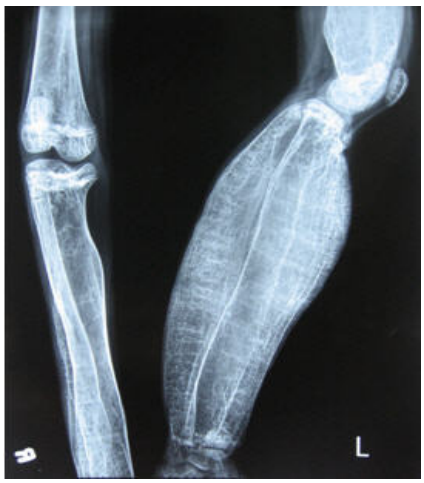


Figure 3. Radiograph of bilateral lower limbs



What is the diagnosis?

Answer

The chest radiograph (Figure 1) shows grossly expanded anterior ends of the ribs. The limb radiographs (Figures 2 and 3) show cortical thinning and lacy trabeculation of the limb bones. The patient was diagnosed as having *Beta-thalassaemia major* by haemoglobin electrophoresis.

The radiologic bone changes are due to marrow hyperplasia and marrow space expansion, resulting from prolonged and excessive haematopoiesis. These changes lead to growth disturbances, modelling deformities, and premature closure of the growth plate. These changes are associated with a characteristic radiologic appearance of the skull, long bones, ribs and hands. Growth is stunted. Gross skeletal deformities can occur as in this case.

These changes are not usually seen in today's practice due to wide and easy availability of safe transfusion therapy. An inadequately transfused child develops these typical radiological features.

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Are doctors team players, and do they need to be?

Jennifer Weller, John Thwaites, Harsh Bhoopatkar, Wayne Hazell

Abstract

Evidence suggests that teamwork failures contribute to poor outcomes in hospitals and that changes in healthcare delivery have at times worked against the development of effective healthcare teams. Doctors' engagement with the concept of healthcare teams, although variable, has generally been supportive and there have been several successful initiatives. However, lack of evidence on the critical components that improve the performance of healthcare teams impedes growth in our understanding and development of effective teams. In an endeavour to improve the function of healthcare teams through education and systems change, the psychology literature remains a useful framework for studying the critical components of team processes.

The training of healthcare professionals has traditionally focused on the knowledge and skills of individual clinical practitioners. This focus is gradually changing however with modern health care increasingly being delivered by teams of health professionals with the expectation that this will lead to improved healthcare delivery processes, better outcomes for patients and lower costs compared to non team approaches.[1]

Adverse events are common in Australasian hospitals with up to 16% of all hospital admissions associated with an adverse event, resulting in disability or longer hospital stay.[2,3] Notably, failures in teamwork and communication have been found to make a substantial contribution to such adverse events and suboptimal care.[4–9] Lingard,[10] observing communication between members of operating room teams, found over a quarter of all communications failed due to poor timing, inaccurate or missing content, or failure to resolve issues. Many of these failures had observable deleterious effects, including inefficiency, tension between team members, wasted resources, delays or procedural errors.

The development of effective clinical teams however is complex and requires more than simply the grouping or clustering of health professionals in a clinical area with the expectation that they will work effectively as a team. Different professional groups have different approaches and attitudes towards teamwork,[11] which may impede the development of a well-functioning team. Changes in the educational and clinical environment can impact on the development of team structures. Furthermore, current studies provide little insight into what are the critical components that improve the performance of patient care teams.[1]

To explore the concept of team work further and whether doctors are team players it is important to firstly define what a team is.

What is a team?

A general consensus in the literature defines a team as consisting of two or more individuals who have specific roles, perform interdependent tasks, are adaptable, and share a common goal.[12] A doctor's role within the team could include; creating a vision; managing change, coordinating tasks, maintaining or supporting team function, or active followership.

Doctors often think of teams in terms of their traditional medical team, but the wider healthcare team can be usefully considered as multidisciplinary, interdisciplinary and transdisciplinary depending on the degree of interaction between members and the degree of shared responsibility for patient care.[13, 14] Members of a multidisciplinary team work in parallel, with minimal interaction except through the doctor, who traditionally, is in charge. In transdisciplinary teams, roles are blurred as professional functions overlap, team members share knowledge, skills and responsibilities, and trust is an essential component for successful group dynamics.[13] The interdisciplinary (or interprofessional) team sits somewhere in between, where the team members work together around common tasks [14] and collaborative communication and decision-making are key elements.[13]

The clinical setting may dictate the appropriate structure for the team and an interdisciplinary team will be required where complex and diverse patient needs require input from a range of health professionals.[15]

Changing healthcare environment affecting the development of team structures

The past 25 years has seen considerable change in the environment for healthcare delivery due to changing demographics with ageing populations, increasing complexity of healthcare, rising costs of health-related technology and increasing consumer expectations.[16] This has occurred against a background of macro health economic changes in New Zealand with experimentation with a competitive model of healthcare delivery in the 1990s, a clash of cultures between doctors and management,[17] and increasingly constrained health funding and resources in the current decade. This has challenged health professionals and medical staff in particular, to work together more effectively to reduce admissions, decrease length of stay, rationalise expensive interventions, while still endeavouring to provide high quality care.

With the increasing complexity of healthcare, doctors meanwhile have become more specialised in response to the continuing growth in scientific knowledge and technological advances. The time and energy required with subspecialisation and the maintenance of working relations with other branches of the medical profession has at times, been to the detriment of relations with other healthcare professions.[18] This medical focus has subsequently been challenged however by the changing expectations of other healthcare professions with their respective subspecialisation and the emergence of interprofessionalism.[18] Traditional medical roles and ward hierarchies have not only been questioned but changed with greater responsibility for many aspects of patient care being assumed by other health professions.

Intraprofessional employment changes have also had an impact on the environment for healthcare delivery. Stricter limits on working hours for resident medical staff as a result of the M10 working hours determination in New Zealand has seen a major change in the composition and structure of traditional medical teams with a decrease in the ratio of senior medical staff to resident medical staff.

Increased shift work rosters have emerged affecting traditional team structures. The continuity of medical care for patients has become more difficult in this environment. The introduction of the European Working Time Directive, which placed comparable restrictions on hours worked by resident medical officers, has also raised concerns about the effect on team structures and the continuity of patient care.[19-21]

The increasing reliance on locum medical and nursing staff in New Zealand hospitals, in conjunction with the changing work patterns of resident medical staff, may also negatively impact on the development of collaborative inter professional relationships. Higher staff turnover provides fewer opportunities to understand and appreciate respective team member's roles and capabilities and insufficient time to develop the respect and trust required for a well functioning team. The high proportion (40%)[22] of international medical graduates in the New Zealand environment may create additional challenges for effective team functioning as attitudes of doctors towards the roles of nurses, and attitudes to speaking up and challenging authority can vary across cultural groups.[23]

By contrast, changing expectations of both consumers and providers in recent years has impacted on the clinical environment with demand for greater accountability of health practitioners and with the expectation that health providers will co-operate between each other thus improving healthcare. Policy documents in countries such as the USA and United Kingdom continue to reinforce the importance of team work in the delivery of health care.[1]

Are doctors team players?

Against this background of change, how have doctors reacted to demands to learn and work in different ways, work more collaboratively and become team players? Often doctors have not been seen as team players unless it was their team and they were the leader. Team work is complex and specific aspects of teams require compromise. Teamwork requires team members to sacrifice some of their individual autonomy, in the interest of collective decision making.[1]

The evidence on doctors as team players is mixed. In the educational environment selection processes for medical school and the competition for training posts have tended to favour individualist behaviours rather than the attributes of team players. Horsburgh et al[24] found medical, nursing and pharmacy students differed in how they believed clinical work should be organised even before they started their training. Medical students believed that clinical work should be the responsibility of individuals. In contrast, nursing students had a collective view and believed that work should be systemised, whereas pharmacy students were at a mid-point in this continuum. On the other hand, medical curriculum activities are increasingly in cooperative small groups the medical course itself may to some extent diminish competitive behaviours[25].

The interprofessional education movement was conceived as a means to improve teamwork amongst health and social care professions. Suggestions that doctors and medical students have been reluctant participants in interprofessional education have been challenged. Two surveys in the United Kingdom found that doctors were well represented in the interprofessional movement relative to their overall numbers.[18, 26] The Royal College of General Practitioners in the United Kingdom was noteworthy for the lead it gave, as it joined in conference with the other professions, in the publication of interprofessional reports.[27, 28]

In clinical practice doctors have often become team players of a sort through clinical necessity. Specific tasks in patient care have in many instances become too complex to be performed by individual practitioners and therefore teamwork is needed. Teamwork has also been seen as a way of overcoming the fragmentation of care by specialisation[1] with recognition that patients who receive care from a team of caregivers may benefit from the insights of different bodies of knowledge.[29]

The concept of "teamwork" is gradually becoming part of mainstream health care[12] as a greater understanding of the importance of teams develops. Patient care teams with doctors playing a team role have been successfully developed around patient populations such as the elderly,[30] or grouped according to disease processes such as diabetes[31] and stroke care[32] with improved clinical outcomes. There is a large body of evidence showing the effectiveness of using a team as part of disease management, especially for chronic disease (e.g., heart failure, diabetes, and hypertension)[28].

Advantages of team

With skilled leadership and a well-functioning team, the many different skill sets of individuals can be utilised to provide more efficient and effective clinical care. Whilst some may consider decisions by consensus prone to problems, teamwork can facilitate clinical decision making. If information is shared among team members, more input can be provided into problem solving and decision making.

A good team leader will listen to the team inviting suggestions or options for diagnosis or management with evidence to suggest that discouraging team input into decision making or "flying solo" may increase the risk of error. Tasks can be allocated more equitably between team members to ensure individuals are not overloaded, with team members supporting each other in reaching shared goals in patient care. A recent review of the literature on leadership and healthcare teams provides good evidence that effective teams can improve patient safety, and leadership is vital for teams to function effectively.[33]

Meta-analyses of randomised controlled trials show that in patients with heart failure, use of multidisciplinary teams reduce the rates of re-hospitalisation and mortality as compared with usual care.[34] Cost-effectiveness studies also show a benefit to a team approach.[35] The evidence on the use of a team approach to disease management is robust and has translated to recommendations in evidence-based guidelines.[36]

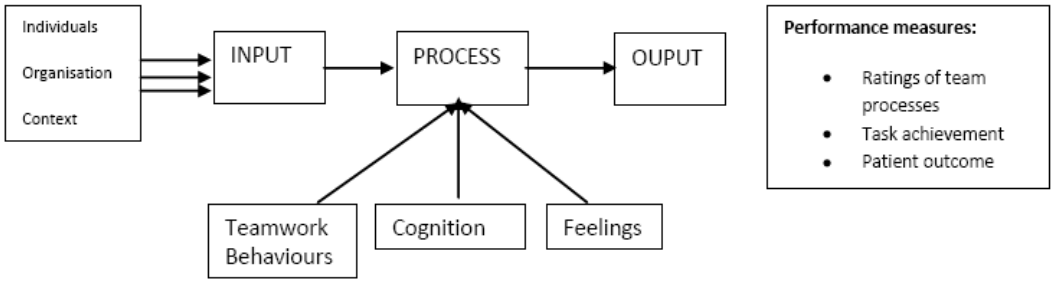
What behaviours and skills are needed to make a team work?

Creating an effective healthcare team is an active process. It requires specific actions and skills. Review of the literature on teamwork suggests a common set of

requirements for an effective team; mutual respect and trust; shared mental models; an open environment for communication; team co-ordination.[37]

Rousseau describes a systematic framework for the study of teams, where team function is considered in terms of input (individuals, organisation and context), team processes (teamwork behaviours, cognition, feelings) and team outputs (patient and team) (Figure 1).

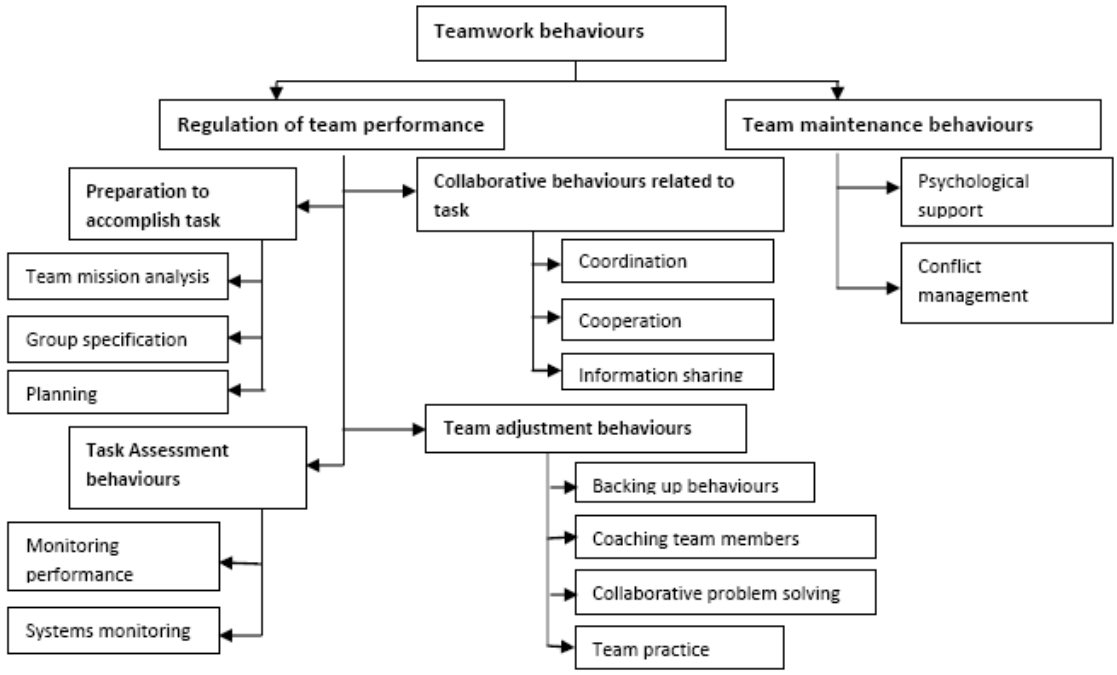
Figure 1. A framework for studying team function (from Rousseau[38])



"Teamwork Behaviours" can be further considered in terms of behaviours required for maintaining a team, behaviours required to accomplish a task, and behaviours required to ensure collaboration between team members (Figure 2). Several factors will affect the requirement for and type of teamwork behaviours. These are related to the nature of the task (task complexity, interdependence of tasks allocated to different team members).

A complex task may require diverse teamwork behaviours and collaborative behaviours in order to accomplish the task; an unstructured task with ambiguous outputs requires high levels of preparation to accomplish the task (i.e. working out what needs to be done) and "task assessment behaviours" (i.e. monitoring how the situation is progressing in response to actions). For example, to save the life of a rapidly deteriorating patient, the team may need to specify roles and coordinate tasks to ensure timely treatment; a team member may need to challenge an authority figure[23, 39] to ensure collaborative problem solving and avert inappropriate management decisions. In highly structured tasks where each team member knows exactly what is to be done there is less need for these behaviours.

Figure 2. Analysis of teamwork behaviours (adapted from Rousseau[38])



Initiatives in creating healthcare teams

One approach to improving teamwork in healthcare has been interdisciplinary education. Hall and Weaver[14] conducted a comprehensive review of the literature from the 1970s on interdisciplinary education of the healthcare team. There were two main themes identified in the literature: system issues and content issues. System issues include availability of an interdisciplinary education curriculum, timing of the intervention (although there is no clear consensus), non-traditional nature of teaching methods, need for faculty development to address motivation to participate, institutional support, and participants' characteristics.

Content-related issues include learning about the roles of other health professionals (maintaining professional role demarcation) rather than learning how to do each other's jobs (role blurring) and the need to learn skills in group work, communication, conflict resolution and leadership. Interdisciplinary initiatives frequently only address the component of learning about the capabilities of people from other disciplines and can fail if they do not actually address the entire process involved in teamwork.

Simulated learning environments may be a way forward for the future. They provide an opportunity for multidisciplinary teams to work together on relevant clinical tasks to develop and practise a range of skills including communication, task co-ordination, sharing information, collaborative problem solving.[40-43]

Recent initiatives in Australia relating to interprofessional education include the "Learning and Teaching for Interprofessional Practice in Australia"[44] which made

recommendations on the integration of interprofessional education into health professional training.

Where to from here?

The New Zealand Health and Disability Commissioner places obligations on health providers with regards to team work and communication. Right 4(5) of *The Code of Health and Disability Services Consumers' Rights* states that, "Every consumer has the right to co-operation among providers to ensure quality and continuity of services". We propose that doctors should be equipped, with the knowledge, skills and attitudes required to work effectively in healthcare teams as leaders and participants. With current evidence, a curriculum for leadership and teamwork should be integrated into the curriculum for undergraduate and postgraduate medical education.

Evidence suggests that teamwork failures contribute to poor outcomes in hospitals and that changes in healthcare delivery have at times worked against the development of effective healthcare teams. Further systems research to better define organisational structures which facilitate or work against the development of healthcare teams, and research into innovations to foster the formation of effective teams is required.

Doctors' engagement with the concept of healthcare teams although variable, has generally been supportive, with several successful initiatives; however, lack of evidence on the critical components that improve the performance of healthcare teams impedes growth in the understanding and development of effective teams. The psychology literature remains a useful framework for studying the critical components of team structure and function, and further research could identify these critical components in an endeavour to improve the performance of healthcare teams.

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Graves' disease: causes and associations

Excerpt from article "On Goitre" by Dr Colquhoun published in NZMJ 1910 Feb;8(33):17–71.

Fifteen years ago I attended a lady for severe Graves' disease with glycosuria, and this year I had her younger sister under my care for the same combination. Many such instances are recorded. One of my correspondents has been so much impressed by this fact that he says when he has attended one member of a family for Graves' disease he expects sooner or later to be called in to see another with the same trouble.

There is also some evidence of what may be called a "Thyroid Constitution." That is, some member of a family may suffer from Active, some from Passive, Goitre. For instance, there is reported the case of one family in which two sisters and a brother were attacked by passive goitre, and one sister developed the active form. I attended a patient some years ago who had a severe attack of the active form; a few years afterwards a younger sister developed a similar attack, and at the same time a brother showed signs of Thyroid inadequacy in the bleaching of parts of the hair, falling out of hair, leucoderma, etc.

Exciting Causes.—In many cases no exciting causes can be traced, but in the great majority there is some history of sudden acute shock or of long continued passive strain. I may cite the case of a patient whom I attended, a woman of middle age, not neurotic or in ill-health; she was living alone in a large house, and in the middle of the night she was roused out of her sleep by the fall of a skylight window down a well staircase to the bottom floor; a few days afterwards when I saw her she had marked tachycardia, and later she developed goitre and exophthalmos. A good many cases are cited by my correspondents, showing that grief, injury, worry, fright and sudden emotions may induce the disease.

Pregnancy.—Lawful and unlawful is a very frequent antecedent, and in some cases has evidently a causal connection with the attack.

Endemic Influences.—I have already expressed he opinion that the active form of goitre differs from he passive in not being endemic, but the very remarkable group of cases noted by Dr. Fleming while he was practising in Balclutha seems to show that occasionally there may be some local conditions, at present of a nature unknown to us, which may predispose to or excite the disease.

Race.—Dr. Buck says that active goitre is practically unknown among the Maoris. Dr. Wilson, of Palmerston North, however, has informed me that he has seen at least one case. Among the European elements in New Zealand I do not know of any evidence that any one race suffers more than another.

Toxaemia.—There is some evidence that Graves' disease may follow septic absorption from caried teeth, etc. This is a point worthy of further consideration.

With Tuberculosis and other Diseases.—The cases cited by Dr. Wohlman and Dr. Fleming that Phthisis and Graves' disease may co-exist. Considering the frequency of phthisis in the whole population this is only what might be expected, but Dr Leonard Williams has pointed out that although generally the Thyroid secretion is antitoxic, patients with any tubercular trouble bear Thyroid extract very badly. It seems to aggravate their condition. I would be interested to know if Graves' disease in any way predisposes to Tubercular disease. My own experience is negative on the point.

Dr. Stevens, of Kurow, suggests an analog between Diabetes Mellitus and Graves' disease. Glycosuria is of course common enough, but is usually only temporary. It may be noted that in the New Zealand Statistics, Otago, which has the highest proportion of deaths from active goitre of any of provinces, has also the highest death-rate for Diabetes Mellitus.

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Can Ginkgo prevent cognitive decline in older adults?

Ginkgo biloba is marketed widely in the USA and elsewhere and used with the hope of improving, preventing, or delaying cognitive impairment associated with aging and neurodegenerative disorders such as Alzheimer disease.

Previous controlled trials have found Ginkgo to be ineffective in the prevention of, or deterioration of, cognitive decline in subjects with Alzheimer disease. This study randomised 3069 elderly (72–96 years) subjects with normal cognition to 120mg of Ginkgo twice daily or placebo. After a median follow-up of 6.1 years it was concluded that Ginkgo did not result in less cognitive decline.

JAMA 2009;302(24):2663–70.

Total health care expenditure as percentage of GDP (gross domestic product) in the Czech Republic—a comparison with the rest of us

This commentary discusses a paradox that exists—Czech health statistics are comparable with other developed countries but their GDP% spent on health is the lowest—6.8% in 2006. In the same year, other European countries had figures ranging from 8.5% for the UK to 11% for France. You will not be surprised to find that the comparable figure for the USA was about 15.5%. So the Czech system, which features fees per service (doctor visit, prescription, and day fees for hospitalisation) may, or may not, be worth considering. The 6.8% of GDP spent on health in the Czech Republic is low, but not as low as the NZ figure—recently reported as 6.3% (public expenditure).

Lancet 2010;375:179–81.

Postoperative risk of venous thromboembolism in middle-aged women (50–64 years)

This study involved 947,454 middle-aged women in the UK who were involved in the Million Women Study between 1996 and 2001. During follow-up, 239,614 were admitted to hospital for inpatient or day case surgery; as well, 5689 women were admitted to hospital or died from venous thromboembolism. The findings were that those having surgery were 70 times more likely to suffer thromboembolism. The risk peaked at 3 weeks postoperatively but was still substantial at 12 weeks. Somewhat as expected but the 12-week observation is disconcerting. The risks varied substantially by type of surgery, being greatest after operations for cancer and for hip or knee replacement. Unfortunately data on postoperative thromboembolic prophylaxis was not available. This would have been very useful. Anyway, the message would appear to be that in high-risk cases prophylaxis should be given for 12 weeks? We assume that middle-aged men would be similarly affected?

BMJ 2010;340:32.

A nurse-led anterior circulation TIA clinic

This report from St Mary's Hospital in London points out that an estimated 23% of ischaemic strokes are preceded by a transient ischaemic attack (TIA), with the cumulative risk of stroke after TIA from the Oxford series being 8% at 7 days, 11.5% at 30 days, and 17.3% at 90 days. As stroke prevention is the aim, early evaluation of such patients is essential. The numbers are overwhelming the ability of the UK neurology services to see such patients urgently. Hence this initiative—specially trained neurovascular specialist nurses run what is called the FAST (face,arm,speech test) TIA clinic at St Mary's.

The acronym is in recognition of the clinical presentation of most anterior circulation TIAs. After 3 years experience (282 patients) they report a high pick-up rate of 86% of neurovascular events (national average about 55%). The median time from referral to clinic was 3 days and one-third were seen within 24 hours. Sounds good.

Postgrad Med J 2009;85:637–42.

Angiotensin-converting enzyme inhibitors (ACEI) or angiotensin II-receptor blockers (ARB), or both, for ischaemic heart disease

Both classes of drugs are known to have established benefit in patients with heart failure and those who have had a myocardial infarction with ventricular dysfunction. Their use, however, in patients with preserved ventricular function is less certain.

This paper attempts to resolve this by a systematic review of 41 relevant studies. The conclusion was that adding ACE inhibitor to standard medical therapy improves outcomes, including reduced risk for mortality and myocardial infarctions (relative risks 0.87 and 0.83 respectively) in patients with ischaemic heart disease with preserved ventricular function. Less evidence supports a benefit of ARB therapy, and combination therapy seems no better than ACE inhibitor therapy alone and increases harms. In particular, treatment withdrawal from combined therapy because of hypotension and syncope.

Ann Intern Med 2009;151:861–71.

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Reducing delay for myocardial infarction

Dr Swanson and colleagues report the important finding that hospital delay in delivering primary percutaneous intervention (PPCI) for acute ST elevation myocardial infarction can be reduced by about 15 minutes by shortening the chain of command after the patient arrives at hospital.[1] But as, the authors point out, this has no effect on the much longer delay (average 2–3 hours in most series) between the onset of symptoms and the patient's arrival. Most of this is due to delay by the patient in calling for help, and, as the authors also say, only community education is likely to improve the situation.

Important—as is recanalisation of the infarct-related artery by thrombolytic treatment or PPCI—timely resuscitation from ventricular fibrillation (most likely to be successful during the first few hours of onset) has the potential to save many more lives.[2] So to call 111 for an ambulance staffed by a paramedic with a defibrillator is the first imperative for a patient with developing infarction.

The "Heart Attack Action!" message to the community has been promulgated in New Zealand by the National Heart Foundation in the past, and in other countries mainly by media campaigns. But results have been disappointing, both here and overseas. A new initiative is required.

One logical initiative would be to couple a brief description of cardiac pain and the message "New chest pain lasting 15 minutes or more call 111 for the ambulance" with standard advice (diet, exercise, don't smoke, check blood pressure and cholesterol etc) on primary prevention. The message should also be repeated to all patients with known acute or chronic coronary disease before discharge from hospital or by their general practitioner.

Any reduction in delay would likely be a long time coming, but this of course applies also to other health initiatives, notably advice on smoking!

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References:

1. Swanson N, Nunn C, Holmes S, Devlin G. Door to balloon times: streamlining admission for primary percutaneous coronary intervention. N Z Med J 2010;123;1309(3986). http://www.nzma.org.nz/journal/123-1309/3986/content.pdf
2. Norris RM The pre-hospital phase of acute myocardial infarction: a national audit is needed in New Zealand. N Z Med J 2007;120:120(1255). http://www.nzma.org.nz/journal/120-1255/2560/content.pdf

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Peter Nigel Black

10 April 1957–10 January 2010

Professor Peter Nigel Black was born in Auckland, and was the first of two sons born to Dr Harry Black and Dr Ruth Black who practised there for many years.

He grew up in a family who travelled widely in New Zealand and overseas. Early on he learnt to love art and history and New Zealand nature.

When doing his postgraduate training in England he met Bernadette Salmon, who was doing research in paediatrics, and persuaded her to come back to New Zealand with him. There she became his much loved wife and they were proud and loving parents of Claire.

Peter left Auckland Grammar School when he was aged 16 as he had won a scholarship, and enrolled at the University of Auckland, where he completed his MBChB in 1980. At the time of his sudden death he was Professor of Clinical Pharmacology at the University of Auckland.

The following obituary, published in the University of Auckland News, was written by his colleagues, Professor Iain Martin (Dean, Faculty of Medical and Health Sciences), Associate Professor Phillippa Poole (Department of Medicine), Professor Ian Reid (Deputy Dean, Faculty of Medical and Health Sciences), and Associate Professor James Paxton (Head of Department, Pharmacology and Clinical Pharmacology).

He continued with his postgraduate medical training in New Zealand and 5 years later obtained his Fellowship of the Royal Australasian College of Physicians. That year, his academic talents were recognised through the awarding of a Medical Research Council Overseas Research Fellowship. This saw Peter move to Britain where he spent the next 3 years in the Department of Clinical Pharmacology, at Hammersmith Hospital, in London, under the mentorship of Professor Sir Colin Dollery, one of the founding fathers of the discipline of clinical pharmacology.

This period in London was seminal for Peter and sparked his long-standing interest in clinical pharmacology in general and in respiratory pharmacology specifically. His subsequent academic career was built upon these two strands, a path that saw him appointed to the Chair of Clinical Pharmacology in 2008.

Peter's research career was unusual in its breadth and its depth, covering the complete spectrum from basic research through to translational and clinical research in its many forms, including Cochrane database analysis and investigator-initiated clinical trials.

Furthermore, the research questions that he addressed were pivotal, especially in the field of chronic obstructive pulmonary disease (COPD).

In addition, the epidemiological studies that he was involved in aimed to answer the critical questions on the role of diet, bacteria and vitamin D in the incidence of asthma and will provide important clinical information on this disease, which is particularly prevalent in New Zealand.

Peter published widely and, even with a career cut cruelly short, he had more than 90 peer-reviewed publications and book chapters, with papers in the highest-ranking general medical journals, such as The Lancet and The New England Journal of Medicine.

Similarly, Peter was very successful in obtaining grant funding from the major funding bodies such as the Health Research Council, Auckland Medical Research Foundation and others, including the National Heart Foundation and Child Health Research Foundation.

One of Peter's most significant contributions was to the "Quality and safety of medicines" initiatives where he made major efforts to improve prescribing nationwide. This is particularly critical at this time in medical practice when new and more complex medicines are becoming available, and where the patient population is ageing and often presenting with co-morbidities requiring treatment with multiple drugs.

His wide involvement in all aspects of medical research and clinical practice impacted very positively on Peter's teaching, both at undergraduate and postgraduate levels. He made enormous contributions to undergraduate medical teaching and received the best lecturer award on three occasions (in 2003, 2005 and 2007) from the Medical Students' Association. His stature as a researcher was also demonstrated by numerous invited lectures at both national and international conferences.

Clinically, Peter was passionate not only about clinical pharmacology, but perhaps most significantly about the importance of medical generalists and general medicine. He undoubtedly championed general medicine at a time when the pressures for increasing subspecialisation made this a difficult furrow to plough. Peter was a consummate diagnostician and loved nothing better than to debate diagnoses and patient management, and was increasingly interested in clinical reasoning.

As an individual, Peter, while a serious thinker and a dedicated clinician, had a keen sense of humour. He was a fountain of knowledge, both medical and general, and his opinion was sought by a large variety of individuals and government bodies. For instance, he sat on a wide range of national committees, such as the Ministry of Health Medicines Assessment Advisory Committee and HRC's Standing Committee on Therapeutic Trials, to name just two. He brought energy, integrity and frankness to every activity with which he engaged.

Professor Peter Black was a true champion of the disciplines of clinical pharmacology and academic general medicine, and his sudden loss will be widely felt. He will be mourned by his colleagues, both academic and clinical, his students and trainees. Our thoughts and sympathies are with Bernadette, Claire and Peter's wider family.

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Erratum

Glover M, Cowie N. *Increasing delivery of smoking cessation treatments to Māori and Pacific smokers*. N Z Med J. 2010;123(1210):6–8. http://www.nzmj.com/journal/123-1308/3942 and http://www.nzmj.com/journal/123-1308/3942/content.pdf

The authors of this editorial advise that a phrase could mislead people as to the actual cost now of subsidised NRT product.

The cost per type of NRT product was reduced to $3 from September 2009 for 4–8 weeks supply—e.g. 8 weeks Patches and 4 weeks Lozenges will cost $6.

It previously was listed as $3 per product/4 weeks—but 8 weeks of patch can be accessed for $3.

Please refer to the links above to view the corrected copy.

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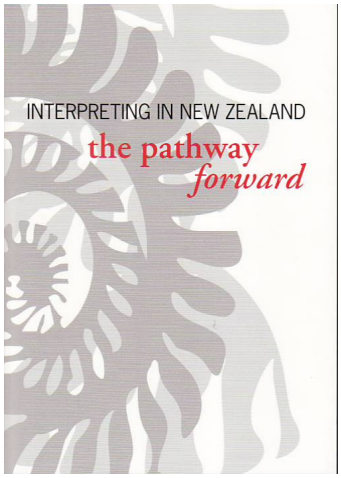
Interpreting in New Zealand, the pathway forward

Diana Clark and Caroline McGrath (eds). Published by Language Line: The Office of Ethnic Affairs (NZ Govt, Wellington), 2009. ISBN 9780478294613. Contains 207 pages. Available from language.line@dia.govt.nz

What health professional in New Zealand has no agreed training, no agreed qualification and no registration authority? The answer is interpreters for people with limited English proficiency.



I suspect many doctors have not thought this was needed for interpreting, it is common practice to use family members, but a moment's thought should suggest otherwise.

Where important health decisions are being made it is essential that the interpreter can be relied upon to interpret accurately, to respect confidentiality and to convey where necessary any relevant cultural practice or belief.

Without a professional structure the doctor has no idea whether this task is being done to a satisfactory standard.

Language Line, the Government unit that supplies telephone interpreting services, has produced a book which supplies useful information for all those who take part in the interpreting communication chain.

Whether it is the health professional, government official, professional interpreter, the interpreting educator or those patients and clients who require and use interpreting, *Interpreting in New Zealand, the pathway forward*, is packed with information from overseas as well as local stakeholders. It is particularly welcome as it fills a niche where there is very little literature available.

For the health professional, while the chapters on health interpreting education and sign language are of obvious interest, the experience and analysis around codes of conduct, ethics and standard setting will also shed light. Ann Corsellis and Jan Cambridge, two gurus of interpreting in the UK look at ways of formalising change both strategically and through formal academic paths. From the Dutch public service comes a comparison of the contents of the codes of conduct of several EU countries. In New Zealand, there's analysis from Duncan Webb now the first Legal Complaints Officer but at the time of writing an ethics professor at the University of Canterbury law school. Daniel Hanks writes incorporating many years of experience in the Deaf community/New Zealand Sign language/mental health arena.

There is also an understanding that this change is not coming about in a vacuum. The book has chapters from those whose experience in a parallel field may help. Among

those included are: the New Zealand Translation Centre or te Taura Whiri i Te Reo Māori, the Māori Language Commission and the NZ Society of Translators and Interpreters.

All will help those of us who realise the days are gone when we could say 'Please bring one of the children, Mrs X' without niggling concerns about risk, informed consent, and confidentiality. A professional interpreting structure with accredited trainers, agreed standards, a code of ethics and a registration body has been developed in other countries. This book sets out the arguments as to why New Zealand needs to set such a structure up to enable health practitioners to ensure all New Zealanders have equity of access to the information and services provided to all by our healthcare system.

One contribution of this book will be that it raises awareness of both the need for neutrality, confidentiality and for clarity in order to address issues of cultural difference. The secondary contribution will be to provide fodder for debate and discussion which it is hoped will lead to improved standards of all interpreting in this country.

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