



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

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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 through 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/1.73m²).

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/1.73m ²
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/1.73m²). 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 Study¹¹), 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 Group¹⁴ recommends calculation eGFR using CG¹⁵ 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.

$$\text{MDRDeGFR} = 186 \times \left[\frac{\text{Serum Creatinine}}{88.4} \right]^{-1.154} \times \text{Age}^{-0.203} \times (0.742 \text{ if female})$$
$$\text{CG eGFR} = \frac{(140 - \text{Age}) \times \text{Weight} \times 1.04 \times 1.73}{\text{Serum Creatinine}} \div (0.007184 \times \text{Weight}^{0.425} \times \text{Height}^{0.725}) \text{ if female}$$
$$= \frac{(140 - \text{Age}) \times \text{Weight} \times 1.23 \times 1.73}{\text{Serum Creatinine}} \div (0.007184 \times \text{Weight}^{0.425} \times \text{Height}^{0.725}) \text{ if male}$$

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/1.73m²) 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/1.73m² 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/m², $p<0.0001$), 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/1.73m² 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/1.73m² 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/1.73m² 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/1.73m²) among European diabetes patients, eGFR estimated using MDRD and Cockcroft-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/1.73m² 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/1.73m²) among Māori diabetes patients, eGFR estimated using MDRD and Cockcroft-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/1.73m² 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/1.73m²

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 HbA _{1c} < 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 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/1.73m²) using eGFR was similar to that found in general practice populations in Australia.⁶ (24.3% using CG), but lower than that in the UK⁵ (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/1.73m²) 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 [¹²⁵I] 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 European 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 US²⁶ and the National Service Framework for Renal Services in the UK²⁷ 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.

Competing interests: None known.

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References:

1. Ritz E, Orth SR. Nephropathy in Patients with Type 2 Diabetes Mellitus. *N Engl J Med* 1999;341:1127-1133.
2. Levey AS, Eckardt KU, Tsukamoto Y, et al. Definition and classification of chronic kidney disease: a position statement from Kidney Disease: Improving Global Outcomes (KDIGO). *Kidney Int* 2005;67:2089-100.

3. ANZDATA Registry Report 2005, Adelaide, South Australia, 2005.
4. Joshy G, Simmons D. Epidemiology of diabetes in New Zealand: revisit to a changing landscape. *N Z Med J* 2006;119(1235). <http://www.nzmj.com/journal/119-1235/1999/content.pdf>
5. New JP, Middleton RJ, Klebe B, et al. Assessing the prevalence, monitoring and management of chronic kidney disease in patients with diabetes compared with those without diabetes in general practice. *Diabet Med* 2007;24:364-9.
6. Thomas MC, Weekes AJ, Broadley OJ, Cooper ME. The assessment of kidney function by general practitioners in Australian patients with type 2 diabetes (NEFRON-2). *Med J Aust* 2006;185:259-62.
7. Endre Z, Beaven D, Buttimore A. Preventable kidney failure: the cost of diabetes neglect? *NZ Med J* 2006;119(1246). <http://www.nzmj.com/journal/119-1246/2338/content.pdf>
8. Peterson JC, Adler S, Burkart JM, et al. Modification of Diet in Renal Disease Study, Blood Pressure Control, Proteinuria, and the Progression of Renal Disease: The Modification of Diet in Renal Disease Study. *Ann Intern Med* 1995;123:754-762.
9. Ismail N, Becker B, Strzelczyk P, Ritz E. Renal disease and hypertension in non-insulin-dependent diabetes mellitus. *Kidney Int* 1999;55:1-28.
10. Intensive blood-glucose control with sulphonylureas or insulin compared with conventional treatment and risk of complications in patients with type 2 diabetes (UKPDS 33). UK Prospective Diabetes Study (UKPDS) Group. *Lancet* 1998;352:837-53.
11. Levey AS, Bosch JP, Lewis JB, et al. A more accurate method to estimate glomerular filtration rate from serum creatinine: a new prediction equation. Modification of Diet in Renal Disease Study Group.[see comment]. *Annals of Internal Medicine* 1999;130:461-70.
12. Mathew TH; Australasian Creatinine Consensus Working Group. Chronic kidney disease and automatic reporting of estimated glomerular filtration rate: a position statement. *Medical Journal of Australia* 2005;183:138-41.
13. Saleem M, Florkowski C. Australasian Creatinine Consensus Working, Reporting of estimated glomerular filtration rate (eGFR) in New Zealand--what are the clinical laboratories doing? *New Zealand Medical Journal* 2006;119:U2337.
14. Management of Type 2 Diabetes. New Zealand Guidelines Group, Wellington; 2003.
15. Cockcroft DW, Gault MH. Prediction of creatinine clearance from serum creatinine. *Nephron* 1976;16:31-41.
16. Renal Update. *Best Practice Journal* June 2007:18-25.
17. UK Consensus Conference on Early Chronic Kidney Disease. *Nephrol. Dial. Transplant.* 2007;22:ix4-5.
18. Assessment and Management of Cardiovascular Risk. New Zealand Guidelines Group, Wellington; 2003.
19. Simmons D, Kenealy T, Shaw LM, et al. Diabetic Nephropathy and Microalbuminuria in the Community. *Diabetes Care* 1994;17:1404-10.
20. Simmons D. Diabetic nephropathy in New Zealand Maori and Pacific Islands people. *Nephrology* 1998;4:S72-S75.
21. Thomson CF, Simmons D, Collins JF, Cecil A. Predisposition to nephropathy in Polynesians is associated with family history of renal disease, not diabetes mellitus. *Diabetic Medicine* 2001;18:40-6.
22. Lin J, Knight EL, Hogan ML, Singh AK. A Comparison of Prediction Equations for Estimating Glomerular Filtration Rate in Adults without Kidney Disease. *J Am Soc Nephrol* 2003;14:2573-2580.
23. Saleem M, Florkowski CM, George PM, Woltersdorf WWW. Comparison of two prediction equations with radionuclide glomerular filtration rate: validation in routine use. *Ann Clin Biochem* 2006;43:309-313.

24. Kuan Y, Hossain M, Surman J, et al. GFR prediction using the MDRD and Cockcroft and Gault equations in patients with end-stage renal disease. *Nephrol. Dial. Transplant.* 20 (2005) 2394-2401.
25. Rigalleau V, Lasseur C, Perlemoine C, et al. Estimation of glomerular filtration rate in diabetic subjects: Cockcroft formula or modification of Diet in Renal Disease study equation? *Diabetes Care* 2005;28:838-43.
26. The American Society of Nephrology (online February 2008) ASN's renal express: February 2008 [http://www.asn-online.org/newsletter/renal_express/2008/08-2-Rxpress.aspx] (accessed 5 May 2009), 2008.
27. National Service Framework for Renal Services. Part two: chronic kidney disease, acute renal failure and end of life care., Department of Health, London, 2005.
28. Mathew TH, Johnson DW, Jones GR. Chronic kidney disease and automatic reporting of estimated glomerular filtration rate: revised recommendations. *Med J Aust* 2007;187:459-63.
29. Komenda P, Beaulieu M, Seccombe D, Levin A. Regional implementation of creatinine measurement standardization. *J Am Soc Nephrol* 2008;19:164-9.
30. Zhang Q-L, Rothenbacher D. Prevalence of chronic kidney disease in population-based studies: Systematic review. *BMC Public Health* 2008;8:117.
31. Glasscock RJ, Winearls CG. Routine reporting of estimated glomerular filtration rate: not ready for prime time. *Nat Clin Pract Nephrol* 2008;4:422-3.
32. Giles PD, Rylance PB, Crothers DC. New results from the Modification of Diet in Renal Disease study: the importance of clinical outcomes in test strategies for early chronic kidney disease. *QJM* 2008;101:155-8.
33. Clase CM. Glomerular filtration rate: screening cannot be recommended on the basis of current knowledge. *BMJ* 2006;333:1030-1.